Robert Sapolsky's Edutaining "Human Behavioral Biology"

Posted on 10 April 2019 by **cjf**

Is human behavior "a magnificent, fascinating, nuanced interaction between nature and nurture"? To what extent is our behavior controlled by our biology and to what extent does our behavior control our biology? What is the nature of a biolgically-based human being? What is unique about the behavior of humans in the animal kingdom?

These questions are the broad concerns of the 25 videos totalling 36 hours and 40 minutes constituting the free on-line edition of Stanford University's Bio 150 course "Human Behavioral Biology" recorded in the spring of 2010 with Robert Sapolsky. The course is an accessible yet scientifically detailed introduction to modern biology's understanding of human behavior. It is a broad biology-centered approach where animal comparisons and a broad systems perspective engage a way of understanding our psychology without too much solipsism. Sapolsky's gift for story-telling and his style of boldly confronting the moral implications of his poignant topics make the lectures engaging edutainment. Topics include sexuality, violence, language, mental illness, religiosity, and individuality which are explained using the main biological subjects of evolutionary theory, genetics, ethology, endocrinology, and neuroscience.

Although I finished studying "Human Behavioral Biology" in July 2016, its continued influence on my thinking and the diffuse locations of my notes has led me to want to document its highlights, my notes on the videos, and the events I organized to explore aspects of the course in small groups. Hence this summary.

Perhaps, Sapolsky's most profound contribution to my thinking came in the first lecture in his course where he composes a list of **the dangers of categorial thinking**:

1. We can miss the big picture by focusing on boundaries.
2. We tend to underestimate differences when two cases happen to fall in the same category.
3. We tend to overestimate differences when cases happen to fall on opposite sides of a boundary.

Of course, as Sapolsky himself acknowledges, categories are essential scaffolding for our thinking. In fact, I cannot imagine how we could think at all without some distinctions, some categories. But these profound dangers affect every distinction, every assumption, and every taxonomy that we might entertain. For me, this realization was stunning, profound, and transformative. Do you see the significance? Can you imagine the comport of its implications?

Another important aspect of the course was to better understand the profound interrelationship between genetics and environment. For me this really hit home when Sapolsky explained how the cure for one of the most devastating single-gene mutation diseases known, PKU (phenylketonuria), is treated with a simple modification to one's diet; a genetic disease "cured" by a simple change to the environment! Nature and nurture are both categories that belie the far more subtle and interfused *gene-environment interaction*. That and the treatment of epigenetics, transcription factors, life history including perinatal development, and the limitations imposed by the requirement of controlled experiments in science gave me a much clearer understanding of how the gene-environment interaction belies many widespread but erroneous assumptions and studies in genetics.

Another significant feature of the course is the way it engages complex systems thinking by looking at the determinants of human behavior from most proximate to most distal: the behavior itself and its releasing stimuli (ethology), neurobiology, acute and chronic hormonal environment, perinatal biology & environment, culture, genetics of the individual, ecological and environmental context, and evolutionary history. Should we think of all causality as this kind of multi-layered, intricate confluences of many overlapping and interconnected systems? I think so! And Sapolsky gives a feeling for this way of thinking that is remarkably effective, if you put in the effort to think it through carefully.

Saplosky's lectures boldly face many of the challenges posed by our modern understanding of biology as applied to human behavior. His engaging lecture style never shys away from controversy so he can address, with accessible but scientifically nuanced detail, broad questions about biological determinism and the biology of morality.

Below I present my curated edition of the course. It will, I hope, help you get even more out of the videos. It might even help you organize your own events to explore on ideas in the course with others. At the end I invite your feedback on this curation, and the materials it includes, and on the prospect that this kind of curatorial approach to organizing group explorations might prototype a new educational service to help our civilization better address its crisis of ignorance.

Before I watched the course, I enjoyed Sapolsky's 37 minute, 13 June 2009 lecture "The Uniqueness of Humans" and organized a 15 April 2012 Thinking Society group exploration "The Uniqueness and Evolution of Humans". Between 14 July 2013 and 2 July 2016, I organized 10 topics and 19 small group conversations to explore the ideas in Sapolsky's course. By finding Jon Dakins's Robert Sapolsky Rocks site, additional web searching, and my own cultivated naïveté, I enhanced these topics with facts, ideas, and questions that go beyond a first-cut listening to the course videos.

In the listing of resources below, roman numbers (e.g., 4) indicate the index of the video in the Stanford YouTube playlist. I affix a letter for supplementary videos. The digbat circled numbers (e.g., ③) refer to the order of topics in the series of events I organized on the course. I generally place my topic before the videos that inspired it. One exception is the first video in the course which I covered in the ⑩th topic organized. My G+ and Facebook notes are essentially identical (for G+, I link to my local archive of my posts, but some links may fail now that G+ has expired), but they are formatted slightly differently and different people commented on one platform or the other.

I wrote digital notes on each video and posted them to social media (G+ and FaceBook). They are very rough. Sometimes I quote Sapolsky without attribution and sometimes I interject my own thoughts. So they are a scholar's nightmare, but I didn't want to spend time rewatching all the videos many additional times to clean up the notes. Their purpose was to highlight many of the key ideas so that others could find them, to spur thinking about the videos, to supplement the group exploration events I organized, and to chase down some of the cross-references. Be very skeptical of the notes as I have already found and fixed numerous embarrassments in them. However, overall, they are, I hope, helpful. And their errors may spur further correspondence and learning. If you notice any errors, please let me know.

If you follow the links to the Meetup events, which are indicated with dingbat circled numbers, you will find a summary of the topic, a list of resources, and, in some cases, comments that highlight

some of the key ideas. They are a condensed re-presentation of my notes which are a condensed re-presentation of each Sapolsky video.

1. Introduction to Human Behavioral Biology (March 29, 2010) 57m (YouTube)

- Course overview. Is human behavior “a magnificent, fascinating, nuanced interaction between nature and nurture”? Includes Sapolsky’s exposition on the dangers of categorial thinking.
- My 2013 notes (G+)
- My 2013 notes (Facebook)
- My 2016 notes (G+)
- My 2016 notes (Facebook)
- Jon Dakins’ notes
- Alex Vermeer’s notes
- John McManamy’s notes
- Open Culture’s overview of the whole course (this is probably how I learned of the course)
- This video is featured in the ⑩th topic below

① The Evolutionary and Genetic Bases of Human Behavior

- What can evolutionary biology and genetics tell us about human behavior?
- Inspired by course videos 2, 3, 4, 5, and 6.
- 14 July 2013 (Meetup) (G+ Announcement)
- 27 July 2013 (Meetup)

2. Behavioral Evolution (March 31, 2010) 96m (YouTube)

- A basic introduction to the theory of evolution; individual selection, kin selection, and reciprocal altruism; prisoner’s dilemma.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins’ notes
- Alex Vermeer’s notes

3. Behavioral Evolution II (April 2, 2010) 96m (YouTube)

- Introduction and critique of sociobiology; intersexual competition; imprinted genes; sperm competition; exogamy and endogamy; spandrels.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins’ notes
- Alex Vermeer’s notes

4. Molecular Genetics I (April 5, 2010) 93m (YouTube)

- DNA, RNA, proteins, mutations, the central dogma of molecular biology and its refutation, epigenetics, transcription factors, DNA “promoter” or “repressor” sequences, splicing enzymes; important examples: phenylketonuria (PKU), testicular-feminizing syndrome, FOXP2 (a language gene), RNA retroviruses, “junk” DNA.
- My notes (G+)
- My notes (Facebook)

- Jon Dakins' notes

5. Molecular Genetics II (April 7, 2010) 74m (YouTube)

- Transposons ("jumping genes") and copy number variants provide more evidence against the central dogma; the story of Nobel laureate Barbara McClintock; microevolution and macroevolution.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

6. Behavioral Genetics I (April 12, 2010) 98m (YouTube)

- Flaws in the methods used to find a genetic basis for behavior; non-mendelian inheritance of traits; twin studies, adoption studies (cross fostering in animal studies), identical twins separated at birth, pre-natal environmental effects, Dutch hunger winter, indirect genetic effects.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

7. Behavioral Genetics II (April 14, 2010) 92m (YouTube)

- Chance, heritability, and the gene-environment interaction; genetic markers; genetic diseases: phenylketonuria (PKU), Huntington's disease, and cystic fibrosis; how controlled studies necessarily overestimate the genetic component of heritability.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

② The Biology of Learning

- What can the field of ethology and neuroethology tell us about human behavior which behaviorists like B. F. Skinner could not? What is the scope of learning in the natural world? This biological context reveals an interesting and possibly unfamilar perspective on human behavior and learning. How does the wonderful world of animal behavior and learning work? What are the roles of instinct, imprinting, prepared learning, and other qualities of how humans and other animals know their relatives, how to nurse, how to deal with heights, snakes, and social dominance?
- Inspired by course videos 8 and 9.
- 10 November 2013 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)
- 30 November 2013 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)

8. Recognizing Relatives (April 16, 2010) 79m (YouTube)

- How to question a scientific study to put its results in perspective. How do organisms recognize kin (relatives)? The major histocompatibility complex, pheromones, autism, Martha McClintock, imprinting, pseudo-kinship.
- My notes (G+)

- My notes (Facebook)
- Jon Dakins' notes

9. Ethology (April 19, 2010) 101m (YouTube)

- How ethology (luminaries: Nikolaas Tinbergen, Karl von Frisch, and Konrad Lorenz) refuted the extremist views of behaviorism (John B. Watson, B. F. Skinner, etc); fixed action patterns, releasing stimulus ("releasers"), innate releasing mechanisms, learning behavior, prepared learning, self-awareness, theory of mind, numerosity.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

③ Brain Science and Human Behavior

- If a human brain consists of about 100 billion neurons each with about 10,000 connections (known as synapses) which communicate to each other by pumping neurotransmitters stored in vesicles into the gap between neurons, why are our brains not completely awash in noise and misfirings and confusion? How can individual differences and diverse responses to experience manifest in the working of the brain? Where and how are concepts & categories & memories stored in the brain? What are the difficulties and challenges in studying the brain? Is there a rational part of the brain or are we emotional creatures?
- Inspired by course videos 10, 11, 12, 13, and 14.
- 12 January 2014 (Meetup) (G+ announcement) (Facebook announcment) (Twitter announcement)
- 18 January 2014 (Meetup)
- 2 February 2014 (facilitated by Joe N. and Joyti Marwah) (Meetup)

10. Introduction to Neuroscience I (April 21, 2010) 60m Nathan Woodling and Anthony Chung-Ming Ng (YouTube)

- Brain anatomy, the neuron and synapse, neurotransmitters.
- My notes (G+)
- My notes (Facebook)

11. Introduction to Neuroscience II (April 23, 2010) 73m Patrick House and Dana Turker (YouTube)

- Memory (hippocampus), neurogensis and plasticity, inhibition; autonomic nervous system (ANS): parasympathetic nervous system (PNS) and sympathetic nervous system (SNS).
- My notes (G+)
- My notes (Facebook)

12. Endocrinology (April 26, 2010) 49m William Peterson and Tom McFadden (YouTube)

- There are four ways for cells to communicate: direct contact, paracrine, neuronal, and endocrine; The endocrine system: chemical messages by hormones in the blood; peptide and steroid hormones; primary brain endocrine glands: the hypothalamus, pituitary, and pineal gland; peripheral endocrine glands include the thyroid gland, adrenal glands, pancreas, ovary, testis; regulation.
- My notes (G+)

- My notes (Facebook)

13. Advanced Neurology and Endocrinology (April 28, 2010) 73m (YouTube)

- Neuroendocrinology, the limbic system, complexities in the neurological and endocrine systems, neuroendocrine axes, hypothalamus, hypothalmic-pituitary-adrenal (HPA) axis, possible regulation mechanisms, negative feedback, autoregulation, steroid hormones, glucocorticoids, GABA, neuromodulation.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

14. Limbic System (April 30, 2010) 88m (YouTube)

- The emotional decision-making system of the brain, unifying theory of the limbic system (central role of hypothalamus), olfaction is just one synapse from the hypothalamus, the model of the triune brain, anatomy of the limbic system, the nature and limitations of brain research, the James-Lang theory of emotion.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

④ The Biology of Human Sexual Behavior

- Inspired by course videos 15, 16, and 17.
- 9 March 2014 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)
- 15 March 2014 (Meetup)

15. Human Sexual Behavior I (May 5, 2010) 101m (YouTube)

- New strategy for the second part of the course: Sapolsky will attempt to understand human behavior by integrating the complex of biological determinants from most proximate to most distal: the behavior itself and its releasing stimuli (ethology), neurobiology, acute and chronic hormonal environment, perinatal biology & environment, culture, genetics of the individual, ecological and environmental context, and evolutionary history.
- Topics: neurobiology of sex, releasing stimuli, libido, attractivity, proceptivity, receptivity, female orgasm, non-reproductive sex, foreplay, homosexuality, masturbation, fantasy, marriage, romance, serial monogamy, hermaphrodite, parthenogenesis, transsexuality.
- My notes (G+)
- My notes (Facebook)

16. Human Sexual Behavior II (May 7, 2010) 100m (YouTube)

- Pheromones, releasing stimuli, hormones, perinatal factors, homosexuality, sexual identity, intersexuality, genetic effects, evolutionary history, cuckoldry, female choice, intersexual friendships, rape.
- My notes (G+)
- My notes (Facebook)

17. Human Sexual Behavior III & Aggression I (May 10, 2010) 96m (YouTube)

- Attractiveness, female-female competition, homosexuality.
- Beginning a broad biological survey of morality, empathy and aggression. What is violence? Violence and aggression, aggressive play and dominance hierarchies, uniqueness of human aggression, the uniqueness of human empathy, the amygdala and aggression, reconciliation, sense of justice, empathy, Patas monkey male-male aggression.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

⑤ The Biology of Morality: the roots of human aggression & empathy

- What can biology tell us about human aggression, empathy, and our moral behavior? "You cannot understand the neurobiology of being violent without understanding the neurobiology of being afraid and being anxious" (Sapolsky quote from video 14 on The Limbic System).
- Inspired by course videos 17, 18, 19, and 20.
- 4 May 2014 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)
- 10 May 2014 (Meetup)

18. Aggression II (May 12, 2010) 105m (YouTube)

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Amygdala and fear, Williams syndrome, the frontal cortex and the modulation of violence, aggression, competition, & cooperation, McNaughton Rule, the functions of the septum, lateral hypothalamus, & anterior cingulate, the connection between thought and emotion, metaphor and affective decision-making in the brain, psychopathological confusion of love and hate, testosterone and aggression.

- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

19. Aggression III (May 14, 2010) 101m (YouTube)

- Moral reasoning and emotion, serotonin & alcohol effects on morality, releasing stimuli for aggression, testosterone and other androgens, perimenstral hormones, environmental triggers of aggression, developmental influences on aggression, theory of mind and empathy, Kohlberg's stages of moral development, effects of parents, peer groups and community on aggression, role of unwanted children in violence, lack of evidence for an effect of moral reasoning.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

20. Aggression IV (May 17, 2010) 102m (YouTube)

- Effects of perinatal development on aggression and empathy, the hypermale hypothesis of autism, genetic effects on aggression, ecological and cultural effects on aggression, cultural

factors for terrorism, evolutionary influences of aggressive behavior, pseudokinship, pseudospeciation, the importance of symbols in peacemaking, evolution of cooperation.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

⑥ Beyond Reductionism: The Biology of Complexity, Chaos & Emergence

- Reductionism is the idea that we can understand systems by decomposing them into more elementary parts which combine under definite rules to produce the whole. Although it is one of the most successful ideas in science, it appears to be inadequate for biology and many other complex and chaotic systems. How does reductionism fail in biological systems? How and why is reductionsim still useful in biology? What is the nature of the successes and failures of reductionism in science? How can we understand the strengths and limitations of reductionism? What is the nature of the new ideas of chaoticism and complex systems which attempt to describe how complex properties emerge from components whose combinations are too unpredictable to encompass precisely?
- Inspired by course videos 21 and 22.
- Sapolsky recommends students read James Glick's Pulitzer Prize winning book "Chaos: Making a New Science" for this part of the course.
- 10 May 2015 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)

21. Chaos and Reductionism (May 19, 2010) 97m (YouTube)

- The weaknesses and strengths of reductionism in science, the butterfly effect, the Lorenzian waterwheel and other models of scale-free and fractal or otherwise complex systems in nature, how reductionism breaks down in biological science, chaoticism, one weakness of science: when you control for variables in your experiments, you may miss the real complexity in the system by designing experiments that only look at the predictable parts of the system, strange attractors, the philosophical possibility is that variability, noise, and unpredictability may be fundamental and inherent.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

22. Emergence and Complexity (May 21, 2010) 102m (YouTube)

- Complex adaptive systems, cellular automata, emergent complexity, neural networks, fractals, power law distributions, "the optima is just an emergent imaginary thing", multimodal neurons in the associational cortex, the cantor set, the Koch snowflake, the Menger sponge, and the packing problem, the "wisdom of the crowds" phenomenon, agent based systems, emergent swarm intelligence, attraction and repulsion rules in agent-based systems.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

⑦ The Biology of Language

- Biology can offer unique clues to understand one of humanity's most profound and mysterious faculties, the ability to communicate through language. What properties are common to all human languages? How does the brain process language? What cognitive role is there in language? How do we learn language? Is sign language different in any fundamental way from spoken language? Is our view of the world influenced by our language? Is a language lost to the mists of history a loss to humanity's ability to understand the world? What are the implications that roughly half of the approximately 7,000 languages on the planet are not being taught to children?
- Inspired by course video 23 and the first 23 minutes of video 24
- 12 July 2015 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)
- 25 October 2015 (facilitated by Sandy Catz) (Meetup)

23. Language (May 21, 2010) 102m (YouTube)

- The behavior and biology of language, meta-communication, neurobiology of language, the acquisition and development of language, animal communication, sign language, prosody, Williams syndrome, Broca's area, Wernicke's area, the arcuate fasciculus, neurology of language acquisition, Sapir-Whorf hypothesis, the relationship between thought and language.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

⑧ The Biology of Mental Illness: Schizophrenia and Depression

- What is the meaning and what are the implications of our modern biological understanding of schizophrenia and depression? What we can learn about our own minds from the clues provided when our biology goes seriously wrong as in these two serious yet poorly understood forms of mental illness? What are the benefits and challenges of trying to understand the biology of depression and schizophrenia?
- Inspired by course videos 24 and 25 and supplementary videos 24-B and 24-C
- 12 September 2015 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)
- 31 October 2015 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)

24. Schizophrenia (May 26, 2010) 100m (YouTube)

- Conclude behavioral biology of language from lecture 23: pidgin languages, linguistic diversity.
- The behavior and biology of Schizophrenia, toxoplasma gondii.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins' notes

24-B. Elyn Saks: Seeing Mental Illness (June 2012)

- Poignant autobiographical story about overcoming the shattered mind of schizophrenia.
- My notes (G+)

- My notes (Facebook)

24-C. On Depression in U.S. (uploaded 10 November 2009) 52m (YouTube)

- The biology of depression, “learned helplessness”, as with schizophrenia covers the full range of behavior: chemical, anatomical, hormonal, genetic, and evolutionary effects.
- My notes (G+)
- My notes (Facebook)
- Jon Dakins’ notes

⑨ The Biology of Religiosity

- What are the biological roots of our religious sense? What does it mean to have religious qualities deeply embedded in our human nature and imposed by our biology? Do we need them? What can we make of the fact that each of our decision’s to be religious or irreligious is one of the most defining decisions of our lives yet may be due to a neurotransmitter hiccup or a genetic influence? What does it mean that some people’s biology causes them to lose faith yet for others their biology builds their faith? Is our religiosity an adaptive trait from an evolutionary perspective?
- Inspired by course video 24 and supplementary videos 24-D, 24-E, and 24-F
- 9 January 2016 (Meetup) (G+ announcement) (Facebook announcement) (Twitter announcement)

24-D. Biological Underpinnings of Religiosity (from 2002, uploaded 30 December 2011) 82m (YouTube)

- Intro: consider several genetic disorders that are terrible except for some situation in which a mild form of the bad gene offers selective advantages; Considers evidence for the hypothesis that schizoptypal personality, a mild form of schizophrenia, may foster shamanism and other forms of spiritual leadership; the biology of ritual, obsessive-compulsive disorder (OCD), Martin Luther and OCD, the healthfulness of religiosity, temporal lobe personality
- Note: The video description on YouTube includes links to 33 other Sapolsky videos which is more than I have seen.
- My notes (G+)
- My notes (Facebook)
- Josh Jones’ review of the video at OpenCulture
- Read Bret Wright’s article in the Colorado Springs Independent on Sapolsky’s take on brains and religion

24-E. Karen Song interviews Robert Sapolsky about his views on religiosity (2008) 4m (YouTube)

24-F. Karen Song interview of Robert Sapolsky (2008) 8m (YouTube)

⑩ The Biology of Human Behavior & Individuality: Is there Free Will?

- What are we to make of our predicament as biological creatures? Biologically, our behavior is directly caused by our neurology responding to environmental stimuli in the context of our acute and chronic hormonal situation which was significantly shaped by our perinatal environment and its biology, not to mention the random factors inherent in our individual genetics which are also governed by the population genetics of our direct lineage which was shaped by the ecological factors that govern the complex of gene-environment interactions

in that population plus the effects of millions of years of evolutionary history. Given this biological causality, what does it mean to be an individual human being? What does it mean that our individuality is the result of both random and deterministic biological processes? Is our individuality not “us”, but the imposition of our biology upon us? If our biology is responsible for who we are and how we behave, is free will illusory?

- Inspired by course videos 1 & 25, 25-A & 25-B
- 12 March 2016 (Meetup) (G+ announcement) (Facebook announcement)
- 2 July 2016 (Meetup) (G+ announcement) (Facebook announcement)

25. Individual Differences (June 2, 2010) 53m (YouTube)

- The biology of individuality, free will and determinism in biology, the biological basis of being human, the biology of our individual differences, quirks, and idiosyncrasies; what is the cause of abnormal human behavior? Insanity Defense Reform Act of 1984, What are the neuropsychiatric “conditions” which lie in the netherland between normal and abnormal human behavior? Temporal lobe personality, Jerusalem syndrome, Stendhal’s disease, Trichophagia or Rapunzel syndrome, apotemnophilia, acrotemophilia (or body integrity identity disorder, BIID). What is the predicament of being human and biological?
- My notes (G+)
- My notes (Facebook)
- Jon Dakins’ notes

25-A Edge TV Interview of Robert Sapolsky on Taxoplasmosis (TOXO: Edge Conversation with Robert Sapolsky)

Here is the full Stanford playlist for “Human Behavioral Biology”: http://www.youtube.com/playlist?list=EC848F2368C90DDC3D.

Please let me know if you find any issues or errors in my notes on any of the videos.

Please post comments or thoughts on any of the videos or events. What did you think of them? What seems important that should be highlighted? What, if anything, seems mistaken?

What do you think of Sapolsky’s course or my curation (re-presentation) of it?

What do you think of re-packaging this free on-line video course to inform explorations of the subject for others as a focus for group dialogue (and as an edutainment guide)? What worked for you and what didn’t? How would you re-package these materials to share them with your friends and associates?

How should ordinary citizens learn about how our worlds work? Is it important to have peers when learning or is watching a video course on your own adequate?

Should sharable free on-line video courses be a part of everyone’s on-going lifelong learning?

Does our civilization need a new institution, outside formal education (with no grades, no degrees, and no credentials), that engages ordinary citizens of diverse backgrounds in explorations of important subjects guided by free on-line courses? Could this help civilization overcome the on-going crisis of ignorance that our world faces?

How would you improve upon my effort with Human Behavioral Biology to prototype this kind of educational service to help ordinary citizens grapple with better understanding how our world works?

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Tags: Brain Science, categorial thinking, Education, ethology, evolution, gene-environment interaction, Genetics, Human Behavioral Biology, Language, morality, Neuroscience, OER, on-line video, Online Courses, Open Educational Resources, Reductionism, religion, Robert Sapolsky, Science, Video Courses

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One Response to “Robert Sapolsky’s Edutaining “Human Behavioral Biology””

1. **WhiplashTM** on 19 May 2021 at 9:23 pm

   Thanks a bunch for putting this together! I’m about 1/3rd into the videos and I decided I want to do the rest of the work for the class (or at least the supplemental reading). I really appreciate you putting this together.

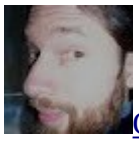
   And thanks for linking to the playlist, too! I couldn’t find a source playlist for this from within YouTube. Found it in Podcasts and iTunes, but I needed the YouTube playlist to watch on my living room TV – uses an Xbox360 for streaming.

   I think what you’ve put together is great. There are aspects of participating in a class that you don’t get from just watching the videos. The ability to ask questions of the professor is one, but the format of the class is mostly just a one way lecture

so there’s very little back and forth. Being able to talk with others about the class, or the days lecture is huge. I would be talking with other classmates as I walked out of class. Asking questions about if they interpreted things the same way I did. Or to clarify what they thought about something. I like the concept, making online classwork more collaborative would recapture a lot of the experience that’s lost. It’d be neat if there were a service or app where you could connect with others interested in similar topics. (I’m thinking along the lines of the 100 I/O, but for academics.)

Thanks again for your efforts!
-M

BELOW ARE COPIES OF cj’S FACEBOOK AND GOOGLE POSTS MENTIONED ABOVE. GENERALLY THE TWO POSTS ARE DUPLICATES BUT I HAVE INCLUDED BOTH IN CASE YOU FIND SMALL DIFFERENCES***

 CJ Fearnley - 2013-03-14 18:26:07-0400 - Updated: 2019-02-11 13:29:48-0500
Introduction to Human Behavioral Biology. 57 minute video lecture.

Interesting beginning to what I expect will be a fascinating course. Sapolsky begins with a real stumper: What is the surprising common connection among these four human conditions: 1) having your period, 2) having a brain tumor, 3) eating a lot of junk food, and 4) taking anabolic steroids (the ones to build muscle mass)? The answer is that each of these conditions has been successfully used in a court of law as a murder defense!

Saposky argues both sides of the coin: sometimes the stuff going on in your body can dramatically influence your behavior; and sometimes what's going on in your head will affect every single outpost in your body. He suggests that there is "a magnificent, fascinating, nuanced interaction between nature and nurture".

The course will examine human behavior and its complex of relationships with environmental stimuli, our anatomy, neurology, endocrinology (hormones), genetics, and evolutionary biology. There are no prerequisites because Sapolsky feels that this subject is so important that everyone in our society needs to understand the basics. The course will present the basics. Cool! I'm ready!!!

However, human social behavior is complicated. One of the most effective ways humans have for dealing with complexity is with categories. But there are three subtle yet profound difficulties with categories: 1) we overestimate differences across the boundary of a category, 2) we underestimate how different two facts are if they fall in the same category, and 3) when you pay too much attention to categories you don't see the big picture. As examples of how we have trouble telling how similar or different two things are that lie across a categorical boundary, Sapolsky mentions how different languages subdivide the color spectrum differently; different sounds are more or less important in different languages: bear vs. pear in Finnish; and 64 vs. 65 are close but 64 is fail and 65 is pass.

Sapolsky cites three biologists who were so blinded by their categories that they uttered ignorant, incorrect, and sometimes horrific ideas: John B. Watson (1878-1958), a pioneer of behaviorism,

Egas Moniz (1874-1955), developer of frontal lobotomy, and Konrad Lorentz, (1903-1989), Nobel Prize winner and Nazis propagandist.

Sapolsky identifies three types of human behaviors: 1) those where humans are just "off the shelf" animals (e.g., the Wellesley effect or Menstrual synchrony which is common in all mammals including humans), 2) those where humans take ordinary animal physiology and use it in ways that no animal could (e.g., the stress response exhibited by chess grandmasters, our ability to sympathize with fictional characters or people in distress on the other side of the planet), and 3) where human behavior is off the charts different from that of most other animals (e.g., our penchant for unreproductive sex). Each of these categories will challenge us especially as Sapolsky argues that we need to think without an excessive hardening of the categories.

The main point of the course he suggests is how our behavior is affected by our biology while resisting the pull of categories to bias our thinking. Let's go! Anyone else want to take the course with me? I plan to watch one (1) video every 4 or 5 days. Since many of the lecture run to an hour and a half, I may need to slow that down. We'll see how it goes (especially since I have some trips coming up).

http://www.youtube.com/watch?v=NNnIGh9g6fA

CJ Fearnley

etpndsrooS1aaa18h0f 38hi3,ca92f514u8r 6204Mh3a346ha80214c030 ·

Introduction to Human Behavioral Biology. 57 minute video lecture.
Interesting beginning to what I expect will be a fascinating course. Sapolsky begins with a real stumper: What is the surprising common connection among these four human conditions: 1) having your period, 2) having a brain tumor, 3) eating a lot of junk food, and 4) taking anabolic steroids (the ones to build muscle mass)? The answer is that each of these conditions has been successfully used in a court of law as a murder defense!
Saposky argues both sides of the coin: sometimes the stuff going on in your body can dramatically influence your behavior; and sometimes what's going on in your head will affect every single outpost in your body. He suggests that there is "a magnificent, fascinating, nuanced interaction between nature and nurture".
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youtube.com
1. Introduction to Human Behavioral Biology
(March 29, 2010) Stanford professor Robert Sapolsky gave the opening lecture of the course entitled Human Behavioral Biology and explains the basic premise o...
CJ Fearnley

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- 12y
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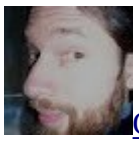
- Edited

Mici Juni
totally rad, this guy. Got through almost the whole 1st lecture. I'm hooked. thanks for finding this and posting it so others can experience. His teaching of the 'big ideas' is my model. He zeros in on the problems of thinking in categories - wonderfully articulated. But I'll have to mull it over - if he's not overlooking other, just as important cognitive biases/errors that we usually run with

- 12y
-

CJ Fearnley
Mici, You and I will especially enjoy lectures 21 & 22. That will probably be one meetup. I'm not sure how to break the course up into meetups yet. I'll try to write up lecture 2 this weekend.

-

 CJ Fearnley - 2016-03-04 22:59:43-0500 - Updated: 2016-07-01 14:12:57-0400
In this video Robert Sapolsky introduces his free on-line course "Human Behavioral Biology" at Stanford University.

One of the main themes in the video is the existence of free will. If our biology causes us to do things both famous and infamous, are we responsible for those behaviors? The course will explore how biology causes human behavior. Sapolsky suggests that free will is supplanted by the biological causes of human behavior. Do you agree? Does biology prove that we are just neurotransmitter hiccups hung on a skeleton clothed with protoplasm?

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What is the thing in common between the following four situations? 1) Having your period, 2) Having a brain tumor, 3) Eating a lot of junk food, 4) Taking a lot of anabolic steroids (the ones that build muscles including testosterone derivatives).

Punchline: "All of these have been used successfully in courts of law to explain the behavior of a murderer."

A major point of the course: "Sometimes the stuff going on in your body can dramatically influence what goes on in your brain."

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"This course is about the intertwining, the interconnections, between your physiology and your behavior, the underlying emotions, thoughts, memories, all of that, and the capacity of each to deeply influence the other under all sorts of circumstances."

It is complicated trying to make sense of human social behavior especially when it comes to grossly abnormal human behavior.

Breaking continua into categories is extremely useful. For example, the continua of colors is broken down by each language into color terms which are different in different languages. Colors that are in the center of each term's range of meaning are easier to remember than colors at the boundaries between the word-categories. "Thinking in categories makes it easier for us to remember stuff. And makes it easier for us to evaluate stuff."

Similarly different languages categorize different sounds (phonemes) differently. So that some people cannot distinguish certain sounds whereas other sounds may be crisply rememberable.

"When you are paying too much attention to categories, you can't differentiate two facts that fall within the same category."

Even though there is very little difference between a test result of a 64 versus a 66, because schools often differentiate those differences between "failing" and "passing", we see a world of difference

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"When you pay too much attention to boundaries, you don't see the big picture. All you see are categories."

"When you think in categories, you underestimate how different two facts are when they fall in the same category. When you think in categories, you overestimate how different they are when there happens to be a boundary in between them. And when you pay attention to categorical boundaries, you don't see big pictures."

In the course, we will use categories to explain behavior, but at the same time resist falling into the problems of thinking in categories. The approach of the course will be to start with an understanding of the behavior. Then trying to understand the proximal causes of the behavior ("what happened a half second before": the "bucket" of neurology and brain science). Then we will look at what environmental factors caused that behavior. Then looking at the more distal cause of hormones which may have made the organism more or less sensitive to the environmental factors. Then looking at the early development of the organism, its genetics, population genetics, and most distally of all its species' evolutionary history.

So the categories (the "buckets" and "outposts" of explanations) are just convenient tools ("temporary platform") to describe what is really very complicated behavior. "There's no buckets. All there are are temporary platforms and each platform is simply the easiest most convenient way of describing the outcome of everything that came beforehand."

Many professional biologists have gotten it wrong:

"Give me a child at birth from any background and let me control the total environment in which he is raised and I will turn him into anything I wish him to be whether doctor, lawyer, beggar, or thief." --- John Watson in 1912, one of the founding fathers of the behaviorist school of psychology

Sapolsky says of him: "Here's a guy living pathologically in this bucket that behavior can be explained solely by understanding reward and punishment."

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"The selection for social utility must be accomplished by some social institution if mankind is not to be ruined by domestication-induced degeneracy. The racial idea as a basis of our state has already accomplished much in this respect. We may, and we must rely on the healthy instincts of the best of our people for the extermination of ailments of the population loaded with dregs." --- Konrad Lorenz, Nobel laureate

Many of the most influential scientists of the last century have been responsible for living pathologically in buckets about how to explain the whole world. We need to "resist the temptation to think inside a bucket and find the explanation."

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1. In some circumstances, there is nothing fancy about humans, "we are just like every other animal out there". "The challenge is to accept that." "Some of the time, we are just a plan old off the rack animal." For example, in all animals, including humans, ovulatory cycles are synchronized by olfaction.

2. Some of the time, we appear to be just like other animals, but we can use standard animal physiology in an unrecognizable way that no other animal could. For example, stress in human grandmaster chess or thinking about our mortality or reading about something awful that has happened to a child on the other side of the planet.

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"For 500 years we all have been using a very simple model for thinking about living systems which is if you want to understand something that is complicated you break it apart into its little pieces. And once you understand the little pieces and put it back together, you will understand the complex thing."

He recommends James Gleich's book "Chaos" which suggests that this reductionist approach doesn't work for complex biological systems. "I found this to be the most influential book in my thinking about science since college."

Much of the end of the lecture is admistrivia. You might want to watch it at a faster playback speed.

CJ Fearnley

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Details

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Behavioral Evolution I (96 minute video) (http://www.youtube.com/watch?v=Y0Oa4Lp5fLE): This video explores how to deduce various qualities of (human) social behavior from the principles of evolutionary theory. That is, it's a basic introduction to sociobiology and evolutionary psychology. Topics discussed include the foundations of evolutionary theory as initiated by Alfred Russel Wallace and Charles Darwin in the 1800s, the principles of natural selection, game theory of cooperation including the Prisoner's Dilemma, problems with group selction, the biology of reciprocal altruism, and the distinction between tournament and pair-bonded species and where humans fit on that spectrum. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/c4k79s7fCx5) Alex Vermeer's Notes (http://alexvermeer.com/human-behavioral-biology-02-behavioral-evolution-i/) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/behavioral-evolution.html) Behavioral Evolution II (96 minute video) (http://www.youtube.com/watch?v=oKNAzl-XN4I) This video builds on the previous one by applying the principles of sociobiology to "understand" infanticide, kidnapping, polyandry, the battle of the sexes, rape, murder, intra-group conflict and inter-group warfaring. The stories explain kin selection and reciprocal altruism. Then Sapolsky discusses multi-level selection and how group selection has some limited validity after all. He concludes with a big warning about sociobiology and critiques three assumptions to this approach to understanding human behavior. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/1ocr9HHzX21) Alex Vermeer's Notes (http://alexvermeer.com/human-behavioral-biology-03-behavioral-evolution-ii/) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/behavioral-evolution-ii.html) Molecular Genetics I (96 minute video) (http://www.youtube.com/watch?v=_dRXA1_e30o) This video starts exploring molecular biology from a conceptual point of view. We learn that the central dogma of biology (DNA->RNA->Protein->biological tools) is wrong! Epigenetics is the regulatory system which controls gene expression and we learn about its mechanisms and its impact. Several fascinating examples are discussed: phenylketonuria (PKU), testicular-feminizing syndrome, the

FOXP2 gene and language. Most of the lecture explains the mechanisms of molecular genetics which are critical to an understanding of behavior from a genetic perspective. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/KbfGBETCeP3) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/molecular-genetics.html) Molecular Genetics II (74 minute video) (http://www.youtube.com/watch?v=dFILgg9_hrU) More mechanisms of molecular biology are discussed including the microevolution / macroevolution distinction, jumping genes, and copy number variants. Examples include vasopressin and the social affiliative behavior of males and some effects of steroid hormones like estrogen, testosterone, and glucocorticoids. Sapolsky weighs the evidence between evolutionary gradualism and punctuated equilibrium and discovers evidence supporting both views. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/BK4xp8PFxav) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/molecular-genetics-ii.html) Behavioral Genetics I (98 minute video) (http://www.youtube.com/watch?v=e0WZx7lUOrY) As Sapolsky explains each of the ever improving approaches to behavioral genetics he exposes serious doubts about their validity. Twin studies and even studies of identical twins separated at birth are criticized strongly. We learn of influential studies whose methodologies and conclusions have been significantly questioned such as Benbow & Stanley's 1980 report "Sex differences in mathematical ability: Fact or artifact?" and Seymour Kety's adoption study of schizophrenia. We learn about behaviors that require no learning, those influenced by pre-natal effects, indirect genetic effects, and "non-Mendelian inheritance of traits" or Lamarckian inheritance (whose ideas have been vindicated in part!). The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/1vyQNjwemgX) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/behavioral-genetics.html) Behavioral Genetics II (92 minute video) (http://www.youtube.com/watch?v=RG5fN6KrDJE) Here Sapolsky describes the modern approach to behavioral genetics which he characterizes with the mantra "go and find the genes". He introduces the third leg in the dichotomy between nature vs. nurture: chance. He overviews the techniques used to find the genes and emphasizes their complexity and statistical nature. With genetic diseases success is achieved. He explains why more complex behaviors have been less amenable to finding the genes. He explains what heritability means (not what you think!) and the importance of the gene-environment interaction and why this means that the science is so often "wrong". The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/QDRe7itaLsk) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/behavioral-genetics-ii.html) If you manage to watch all six videos, you may want to watch the first 30 minutes of the next lecture in Sapolsky's course (http://www.youtube.com/watch?v=P388gUPSq_I) which provides an succinct review of heritability and provides a wonderful exercise in how to interpret scientific studies. Finally, although none of the content in the introductory lecture for Sapolsky's course (http://www.youtube.com/watch?v=NNnIGh9g6fA) will be important for our discussion, you may find the overview interesting. Sapolsky is brilliant, I hope you'll enjoy the lectures and maybe you'll join me and watch the whole course.

Subjects include sociobiology, behavioral ecology, evolutionary psychology, molecular genetics including the prominent role of epigenetics, and behavioral genetics.

Due to strong interest in this topic, it will be repeated on Saturday 27 July (https://www.meetup.com/thinkingsociety/events/128381212/). Please RSVP to the meeting that works best for you.

Comments

700

Patty .

4,375 days ago

Thanks for all your hard work and preparation, CJ. As always, it was a wonderful discussion. I'm glad you shared the videos. Even though I wasn't able to view them before the meet-up, I can view them in the future. It was good to see you and Jeannie.

Tee C.

4,375 days ago

CJ, thank you so much for the great job you did in preparation for this meetup. This was a topic unfamiliar to me (at least the details), but between the videos and the discussion, I feel better familiar with the topic and agree with the author that evolution and genetics and environment are not the total basis of human behavior. Thanks! Tee

Will B.

4,375 days ago

C.J., you do more work on your event hostings than some of my professors on many levels. You should make copies of your notes available. Everyone contributed wonderfully and you do show a fine ability to moderate and encourage the groups. I look forward to follow up on the 27th! For now, it's back to catching up with a few(?) ideas with which I left today. Thanks!

CJ F.

4,375 days ago

Will, if you look carefully at the description, I provided a link for each video to my Google Plus notes (I also post to Facebook, but since Google lets me edit my posts to fix typos that is the best

linkable version). So if you want my notes read through the description and click on the links to "My Notes".

CJ F.

4,370 days ago

Will, you can learn more about the recent Australopithicus discoveries in Lecture 18 of this course http://courses.bilkent.edu.tr/videolib/course_videos.php?courseid=27

Ostensibly that lecture is about Lucy, but after covering that background material Julian Bennett discusses the latest findings.

Greater Philadelphia Thinking Society
See more events

The Evolutionary and Genetic Bases of Human Behavior

Details

Are we more influenced by nature or nurture? Or is human behavior "a magnificent, fascinating, nuanced interaction between nature and nurture"? What can evolutionary biology and genetics tell us about human behavior? What can biology tell us about how we behave the way we do? These are the kinds of questions we will explore in this discussion.

This topic is inspired by six engaging, easy to follow, mostly jargon-free, entertaining, introductory lectures by Robert Sapolsky from his free, on-line video course "Human Behavioral Biology" (http://www.youtube.com/playlist?list=EC848F2368C90DDC3D):

Behavioral Evolution I (96 minute video) (http://www.youtube.com/watch?v=Y0Oa4Lp5fLE): This video explores how to deduce various qualities of (human) social behavior from the principles of evolutionary theory. That is, it's a basic introduction to sociobiology and evolutionary psychology. Topics discussed include the foundations of evolutionary theory as initiated by Alfred Russel Wallace and Charles Darwin in the 1800s, the principles of natural selection, game theory of cooperation including the Prisoner's Dilemma, problems with group selction, the biology of reciprocal altruism, and the distinction between tournament and pair-bonded species and where humans fit on that spectrum. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/c4k79s7fCx5) Alex Vermeer's Notes (http://alexvermeer.com/human-behavioral-biology-02-behavioral-evolution-i/) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/behavioral-evolution.html) Behavioral Evolution II (96 minute video) (http://www.youtube.com/watch?v=oKNAzl-XN4I) This video builds on the previous one by applying the principles of sociobiology to "understand" infanticide,

kidnapping, polyandry, the battle of the sexes, rape, murder, intra-group conflict and inter-group warfaring. The stories explain kin selection and reciprocal altruism. Then Sapolsky discusses multi-level selection and how group selection has some limited validity after all. He concludes with a big warning about sociobiology and critiques three assumptions to this approach to understanding human behavior. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/1ocr9HHzX21) Alex Vermeer's Notes (http://alexvermeer.com/human-behavioral-biology-03-behavioral-evolution-ii/) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/behavioral-evolution-ii.html) Molecular Genetics I (96 minute video) (http://www.youtube.com/watch?v=_dRXA1_e30o) This video starts exploring molecular biology from a conceptual point of view. We learn that the central dogma of biology (DNA->RNA->Protein->biological tools) is wrong! Epigenetics is the regulatory system which controls gene expression and we learn about its mechanisms and its impact. Several fascinating examples are discussed: phenylketonuria (PKU), testicular-feminizing syndrome, the FOXP2 gene and language. Most of the lecture explains the mechanisms of molecular genetics which are critical to an understanding of behavior from a genetic perspective. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/KbfGBETCeP3) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/molecular-genetics.html) Molecular Genetics II (74 minute video) (http://www.youtube.com/watch?v=dFILgg9_hrU) More mechanisms of molecular biology are discussed including the microevolution / macroevolution distinction, jumping genes, and copy number variants. Examples include vasopressin and the social affiliative behavior of males and some effects of steroid hormones like estrogen, testosterone, and glucocorticoids. Sapolsky weighs the evidence between evolutionary gradualism and punctuated equilibrium and discovers evidence supporting both views. The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/BK4xp8PFxav) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/molecular-genetics-ii.html) Behavioral Genetics I (98 minute video) (http://www.youtube.com/watch?v=e0WZx7lUOrY) As Sapolsky explains each of the ever improving approaches to behavioral genetics he exposes serious doubts about their validity. Twin studies and even studies of identical twins separated at birth are criticized strongly. We learn of influential studies whose methodologies and conclusions have been significantly questioned such as Benbow & Stanley's 1980 report "Sex differences in mathematical ability: Fact or artifact?" and Seymour Kety's adoption study of schizophrenia. We learn about behaviors that require no learning, those influenced by pre-natal effects, indirect genetic effects, and "non-Mendelian inheritance of traits" or Lamarckian inheritance (whose ideas have been vindicated in part!). The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/1vyQNjwemgX) Notes from a Sapolsky fan (http://robertsapolskyrocks.weebly.com/behavioral-genetics.html) Behavioral Genetics II (92 minute video) (http://www.youtube.com/watch?v=RG5fN6KrDJE) Here Sapolsky describes the modern approach to behavioral genetics which he characterizes with the mantra "go and find the genes". He introduces the third leg in the dichotomy between nature vs. nurture: chance. He overviews the techniques used to find the genes and emphasizes their complexity and statistical nature. With genetic diseases success is achieved. He explains why more complex behaviors have been less amenable to finding the genes. He explains what heritability means (not what you think!) and the importance of the gene-environment interaction and why that means the science is so often "wrong". The following summaries of the lecture may also be perused: My Notes (https://plus.google.com/u/0/104222466367230914966/posts/QDRe7itaLsk) Notes from a Sapolsky

fan (http://robertsapolskyrocks.weebly.com/behavioral-genetics-ii.html) If you manage to watch all six videos, you may want to watch the first 30 minutes of the next lecture in Sapolsky's course (http://www.youtube.com/watch?v=P388gUPSq_I) which provides an succinct review of heritability and provides a wonderful exercise in how to interpret scientific studies. Finally, although none of the content in the introductory lecture for Sapolsky's course (http://www.youtube.com/watch?v=NNnIGh9g6fA) will be important for our discussion, you may find the overview interesting. Sapolsky is brilliant, I hope you'll enjoy the lectures and maybe you'll join me and watch the whole course.

Subjects include sociobiology, behavioral ecology, evolutionary psychology, molecular genetics including the prominent role of epigenetics, and behavioral genetics.

Note: This topic is a repeat of the July 14th discussion (https://www.meetup.com/thinkingsociety/events/116303742/) which filled to capacity a week early.

Comments

700
Marie

4,355 days ago

CJ you did an excellent job. This was my first meeting and was glad to be there. Everyone was respected in their opinion, you kept everyone on track, and I'm looking forward to more discussions on sapolskys videos. thanks

Sidney .

4,362 days ago

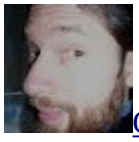
Every time I attend a meetup of the GPTS, without fail I come away feeling a mix of very intellectually stimulated and socially pleased. This one was no exception. Thanks CJ, and great job!

4,362 days ago

I found the discussion very fascinating ! As a retired school psychologist who has spent three decades evaluating students with learning / mental disorders and of superior intelligence , the question of Nature vs Nurture always remained unanswered. However,todays discussion served an

excellent intellectual stimulant for my inquisitive mind!
Thanks CJ for facilitating the group smoothly so that all opinions could be voiced and heard effectively. I found the group very refreshing!

4.8

 CJ Fearnley - 2013-03-18 20:02:57-0400 - Updated: 2013-07-27 08:15:47-0400
Behavioral Evolution I. 1h 36m Robert Sapolsky video.

This lecture starts exploring how we can deduce all sorts of principles of (human) social behavior from the principles of evolutionary theory. The fields of sociobiology (aka behavioral ecology) and later evolutionary psychology have developed this kind of thinking.

Sapolsky gives a quick review of the theory of evolution by natural selection that was developed by Alfred Russel Wallace and Charles Darwin in the 1800s. This is the theory that heritable traits in populations change over time whenever certain traits (there is an assumption of variability in traits including the possibility of mutations leading to new traits) prove to be more adaptive than others. Adaptability is not "survival of the fittest", but rather reproduction of the fittest. Behaviors are also heritable traits that can have selective or adaptive qualities. More adaptive behaviors will over time tend to become more commonplace. Sapolsky riles against Wynne-Edwards' espousal of group selection (the idea that animals behave for the good of the species). Sapolsky asserts that that is not how it works.

Sapolsky identifies three building blocks of natural selection (at least as far as this lecture goes). They are: 1) individual selection which Sapolsky represents with the expression "sometimes a chicken is an egg's way of making another egg". Another aspect of this is sexual selection (choosing mates) which can conflict with the basic "selfish gene" selection proclivity (witness the peacock's tail). 2) kin selection or "inclusive fitness": since each individual shares 1/2 their genes with their siblings and 1/8 of their genes with their cousins, it can happen that traits supporting an increase in the reproductive success of relatives might increase in the population. This is captured by the expression "I will gladly give my life for 2 brothers or 8 cousins" (attributed to Haldane). So evolution selects for organisms who help their relatives. 3) Reciprocal altruism. In addition to truces or stalemates like rock-paper-scissors, organisms (including bacteria, vampire bats, stickelback fish, and naked mole rats) have actual cooperation with non-kin others. This tends to require memory and cheating detection cognition skills. We, like most social organisms, are much better at detecting cheating than "random acts of kindness".

Sapolsky concludes with a long discussion on the mathematical modelling (game theory) of cooperation (prisoner's dilemna and related games), exceptions to cooperation (and the messiness of the real world), and an exquisite exercise to show how even this most basic of introductory accounts on behavioral evolution can detail an extensive understanding of the social behavior of two species given only a basic knowledge of the extent of sexual dimorphism (different traits between males and females of the same species) based on specimen skulls.

Sapolsky goes into depth on the prisoner's dilemna (http://en.wikipedia.org/wiki/Prisoner%27s_dilemma). I read Robert Axelrod's book in the 1980s

and so I'll let you read the details in Wikipedia (or watch the video!!!). The 10,000 foot view is that in computer simulations, the tit-for-tat (TFT) strategy wins because it is nice (always cooperates at first), it is punitive (it retaliates against non-cooperation), it is forgiving, and it is straightforward (not random or probabalistic behavior). But it has a vulnerability if signal noise intervenes. When a mistake or noise turns a C (cooperation) into a D (defection), half of the cooperation can be wiped out. Forgiving TFT (FTFT) does better in these cases because it is more forgiving and builds a sense of trust with partners who have cooperated for a long enough period of time. The strategy named pavlov can exploit FTFT with the rule of doing again whatever won points in the last round, but switching if losing points.

Then biologists searched for strategies used by animals in nature and found many good examples (bacteria, vampire bats, stickelback fish, and naked mole rats). But they also found exceptions: unpunished defectors. With division of labor social organisms can have multiple dimensions of behavior. For example, with the naked mole rats, some large individuals appeared to be cheating. However, it was eventually discovered that they block access to their burrows in the rainy season which "compensates" for doing no work during the rest of the year. Sapolsky warns us that as we delve into behavior more deeply we will find extraordinarily complex behaviors as reputations and complex social roles enter the picture.

Sapolsky asks "how does reciprocal altruism play out in the world of natural selection? In addition to cooperative hunting, he suggests that another good example is given if you and your non-relatives spend an insane amount of time making you both look really good before going to the prom.

Then he does a wonderful exercise wherein he applies the principles of behavioral evolution to deduce the behavioral traits in comparison between a "tournament species" (where there is a large difference in sexual dimorphism, or the physical differences between males and females) and a "pair-bond species". We find that the species with bigger males has more aggression, only a few males father most of the children, the males provide relatively little parental work, the males have shorter lifespans, the females are attracted to bigger males (rather than selecting for males who will help more in childrearing: asking them to bring a worm as part of courtship for some birds), but there is less cuckoldry (females abandoning their children to find a better mate). It is remarkable how much we can accurately deduce about a species from analysis of differences in the skulls of males and females.

Humans are a hybrid species that has some tournament qualities and some pair-bond qualities. So our behavior is particularly complex. Welcome to the wonderful world of Robert Sapolsky's Human Behavioral Biology!

2. Behavioral Evolution

CJ Fearnley

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See more

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2. Behavioral Evolution
(March 31, 2010) Stanford professor Robert Sapolsky lectures on the biology of behavioral evolution and thoroughly discusses examples such as The Prisoner's ...
CJ Fearnley
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Prisoner's dilemma - Wikipedia, the free encyclopedia

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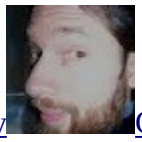
- 12y  CJ Fearnley - 2013-03-23 19:25:54-0400 - Updated: 2013-07-13 15:18:02-0400

Behavioral Evolution II. 1h 37m Robert Sapolsky video.

Sapolsky starts with a nice review of the previous lecture which makes it easy to learn this subtle material. He keeps digging deeper with more examples and theory. Individual selection views behavior as an epiphenomena to get genes propagated. Kin selection and reciprocal altruism round out the basic theoretic foundation for behavioral evolution. These principles of sociobiology / evolutionary psychology "explain" human war, genocide, infanticide, etc. It seems that we can predict most of a species' behavioral characteristics just from knowing a few things about their skeletons. This lecture cites several more successful examples and then builds to some deep criticisms of sociobiology which presage the next lectures on molecular genetics.

There is a pattern to infanticide: it is almost always the males that kill babies of their own species and it tends to happen only in those species where the average interbirth interval in females is greater than the average tenure of a high-ranking male (e.g., langur & vervet monkeys, lions, and mountain gorillas). In species with infanticidal males, some females will abort their pregnancy or enter pseudo-estrus (physiological signs that she is ovulating and ready to mate) if a new high-ranking male enters the picture (so that she doesn't waste time in a pregnancy that will result in the death of her newborn). More variations are presented all corroborating the sociobiological perspective.

Low-ranking males will sometimes kidnap the children of high-ranking males that are attacking them (the message being: lay off or your kid will "get it"). We know that they understand the parental relationships involved because Sapolsky himself documented a Baboon that made a mistake by kidnapping the "wrong" kid and threw the child aside before running away.

Female fetuses tend to be less calorically demanding and are preferred in times of drought or if the mother is low-ranking (her boys would likely be low-ranking and may never father children). 53% of human fertilizations are for males. At birth it is 51% male. Not until adolescence does the male-female ratio switch. After a boy birth, the body weight of the next child tends to be less. Robert Trivers figured out the sociobiology of sex ratios as a function of social context. Whichever sex is more common is less desirable and this creates a dynamic tension in the sex ratio that hovers around equilibrium.

Polyandry is one female with several male mates. Adelphic polyandry means that the males in the "marriage" are all brothers. It happens with lions and there is a group of humans in Tibet where marginal farming land has led to this form of polyandry to prevent the subdivisioning of land which is already too poor for subsistence.

Imprinted genes are a class of genetic traits that work (controlled by methylation) differently depending on the sex of the parent they come from. David Haig developed the theory in the early 1990s. The theory builds on kin selection in the face of genomic conflict between mother and father. All of the imprinted genes from males promote fetal growth: faster metabolism, thicker uterus,

production of more growth hormone. The genes from the female tends to scale back on these fetal growth effects for example by decreasing the receptiveness of the growth hormone receptor. A few imprinted genes affect the child after birth, for example the male gene might increase the suckling response. A fascinating evolutionary arms race over the genomic conflict between males and females. Another such evolutionary arms race was documented by William Rice at Santa Cruz. Some flies have multiple male partners so the female has sperm from several males. Some of these sperm produce a toxin that kills the other sperm.

Baboons have male exogamy: upon sexual maturity males leave their troop to join a group of all related females. In Chimps there is female exogamy: the males in a troup are all related and females leave their birth troop upon sexual maturity to find new group. This has evolutionary implications for behavior. With male exogamy, we find more intra-group fighting between males (and that plays out in the Baboon). With female exogamy we find more inter-group fighting (Chimps are notorious for thier "border patrols" and their inter-group fighting which can reach the point where it meets the UN defintion for genocide). Our closest animal relative!!!

Nice reminder of Marlin Perkins and that great program of my youth: "Wild Kingdom". Though Sapolsky is critical of aspects of the program. Remarkably the program is still running on Cable (though Perkins died in 1986).

After Sapolsky strongly criticized group selection ideas in the previous lectures, he now finds that in a more refined approach it is salient. David Sloan Wilson (distinguished Binghamton University biologist and long-time group selection advocate) and E.O. Wilson (founder of Sociobiology and long-time individual selection advocate) wrote some papers in 2007-8 where they burried the hatchet of their long-time disagreement and agreed to agree that group selection in at least a few particular circumstances actually makes sense. If a small group of individuals becomes isolated from the large population of a species, the smaller isolated group will due to kin selection influences develop more cooperation yielding a larger payoff (in the game theory sense) and this will fix these cooperative traits in the isolated population. Then when these "founders" are reintroduced to the larger population, their cooperative traits will outcompete the others until their group becomes prevalent. This scenario shows how kin selection can lead to reciprocal altruism and it provides a model for fixing cooperation vis-a-vis group selection to "crystalize" cooperation.

This leads to David Sloan Wilson's idea of multi-level selection: depending on the circumstances the most important thing in evolution might be a single gene, a single organism, or a single group.

Two warnings about applying all this to humans: 1) we need to beware of the temptation to take traits and behaviors in the animal world and apply them as inflated models of human behavior. 2) Then you might see rape, infanticide, kidnapping, and genocide as "natural" and inevitable. We can fall into major distortions!!! Humans have a unique evolutionary history and our quirks and complex personalities mean that such modeling can be shaky ... very shaky.

Then he starts to criticize sociobiology. E.O. Wilson said that eventually all of the social sciences will be under the wing of evolutionary biology. But sociobiology has three assuptions and each has been criticized. Sapolsky examines these.

First, there is the assumption of heritability of behavioral traits. The sociobiological accounts of the evolution of biology start sounding like "just so" stories. There is no DNA sequence specified: is it all just imaginary? When will behavioral traits have a genetic influence? In which environments will certain behaviors have a genetic influence. Sociobiology does not solve these problems.

Secondly, there is the assumption that a selected trait is adaptive. Does evolution apply the scalpel

of optimization to behavior selection? Are these "just so" stories disprovable? Stephen Jay Gould and molecular geneticist Richard Lewontin introduced the idea of a spandrel: traits that have evolved not due to their adaptiveness but because they are the accidental byproduct of adaptive traits. The word spandrel is borrowed from the architecture of arches: between two arches, geometrically you will inevitably have a space that architects might as well decorate. For example, the shape of the human chin is a spandrel it results inevitably from a foreshortened muzzle and the angle of our jaw. "Evolution is not an inventor, it is a tinkerer" (Sapolsky cites Andre Lwoff but my google search did not corroborate the attribution). Evolution works with preexisting structures and does not optimize every trait.

Thirdly, there is the assuption of gradualism. Stephen Jay Gould argues that evolution is not gradual, but operates as punctuated equilibrium alternating between periods of rapid change and stasis (equilibrium). Sapolsky suggests that the molecular biology supports this view and hints that we will go in more depth in the next lectures.

In addition to these scientific criticisms of sociobiology, there are social, cultural and political criticisms. It tends to be the Western evolutionary biologists who focus on hyper competition as a scalpel for optimizing selection. The Soviet school of evolutionary biologists tend to emphasize abiotic selection with less competition. But the discussion can also get heatedly political. E.O. Wilson, Robert Trivers, and Irven DeVore, as leading sociobiologists all tended to be white males from the South. Stephen Jay Gould and other critics tended to be Marxists from the East. Once a Marxist pushed Wilson from the stage. Is it scientifically suspicious that a theory that seems to justify the dominance of men happened to be promoted by men from the South? And the theory that would be most amenable to Marxists with their interest in revolution might promote a theory that sees evolution highlighted by periods of rapid change?

Is rape a valid competitive strategy or a psychopathology? Is the fact that children are more likely to be killed by stepfathers rather than their natural parents the result of the natural biology of gene competition or is it due to socioeconomic factors? The impression that sociobiology justifies a world in which male domination in a stratified and aggressive world where competition pays off has plagued the field since its founding.

2. Behavioral Evolution

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CJ Fearnley

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To learn about spandrel, punctuated equilibrium, David Sloan Wilson and the partial revival of group selection, read my detailed notes in the comment below.

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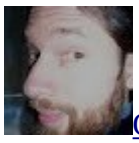
Then he starts to criticize sociobiology. E.O. Wilson said that eventually all of the social sciences will be under the wing of evolutionary biology. But sociobiology has three assumptions and each has been criticized. Sapolsky examines these.
First, there is the assumption of heritability of behavioral traits. The sociobiological accounts of the evolution of biology start sounding like "just so" stories. There is no DNA sequence specified: is it all just imaginary? When will behavioral traits have a genetic influence? In which environments will certain behaviors have a genetic influence. Sociobiology does not solve these problems.

Secondly, there is the assumption that a selected trait is adaptive. Does evolution apply the scalpel of optimization to behavior selection? Are these "just so" stories disprovable? Stephen Jay Gould and molecular geneticist Richard Lewontin introduced the idea of a spandrel: traits that have evolved not due to their adaptiveness but because they are the accidental byproduct of adaptive traits. The word spandrel is borrowed from the architecture of arches: between two arches, geometrically you will inevitably have a space that architects might as well decorate. For example, the shape of the human chin is a spandrel it results inevitably from a foreshortened muzzle and the angle of our jaw. "Evolution is not an inventor, it is a tinkerer" (Sapolsky cites Andre Lwoff but google search cannot corroborate that attribution). Evolution works with preexisting structures and does not optimize every trait.
Thirdly, there is the assumption of gradualism. Stephen Jay Gould argues that evolution is not gradual, but operates as punctuated equilibrium alternating between periods of rapid change and stasis (equilibrium). Sapolsky suggests that the molecular biology supports this view and hints that we will go in more depth in the next lectures.
In addition to these scientific criticisms of sociobiology, there are social, cultural and political criticisms. It tends to be the Western evolutionary biologists who focus on hyper competition as a scalpel for optimizing selection. The Soviet school of evolutionary biologists tend to emphasize abiotic selection with less competition. But the discussion can also get heatedly political. E.O. Wilson, Robert Trivers, and Irven DeVore, as leading sociobiologists all tended to be white males from the South. Stephen Jay Gould and other critics tended to be Marxists from the East. Once a Marxist pushed Wilson from the stage. Is it scientifically suspicious that a theory that seems to justify the dominance of men happened to be promoted by men from the South? And the theory that would be most amenable to Marxists with their interest in revolution might promote a theory that sees evolution highlighted by periods of rapid change?
Is rape a valid competitive strategy or a psychopathology? Is the fact that children are more likely to be killed by stepfathers rather than their natural parents the result of the natural biology of gene competition or is it due to socioeconomic factors? The impression that sociobiology justifies a world in which male domination in a stratified and aggressive world where competition pays off has plagued the field since its founding.

-  CJ Fearnley - 2013-04-02 18:17:39-0400 - Updated: 2013-07-10 17:09:12-0400
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In order to assess sociobiology and study human behavior from another perspective, this lecture starts a deep exploration of molecular genetics. Sapolsky's approach requires no chemistry, but there is a lot of high-level detail provided.

First Sapolsky explains the central dogma of molecular biology (first espoused by Francis Crick): how genes encode RNA which encodes protein sequences which then fold into functional biological structures. Along the way we learn about a series of genes, genetic diseases, and how genes affect behavior. Then we learn that the central dogma is not true!!! Epigenetics is the regulatory system which controls gene expression often by controlling access to genes via methylation or the chromatin structure into which DNA is ensconced. A slogan might be "fertilization is all about genetics; development is all about epigenetics". Sapolsky covers many ways in which epigenetics affects behavior and disease. The new evidence about epigenetics supports the model of punctuated

equilibrium first proposed by Stephen Jay Gould and Niles Eldridge.

Environment can affect DNA expression: it can regulate genetic effects. Chemical messengers can affect gene expression (e.g., hormones: blood-born chemical messengers). Pheromones regulate genes from outside chemicals. Events in the rest of the cell, organism or universe (environment) can affect gene expression. The epigenetic control system is crucial.

Details:

Protein is the structural building block of the body. Our hormones, enzymes, cell structures, etc. are mostly comprised of protein. Proteins work on the lock and key model (exception: prions): when they fit together the action happens (which may make some chemical reaction possible). So protein shape is crucial. There are 20 amino acids which are in turn the building blocks of protein. Each amino acid is more or less hydrophillic or hydrophobic (life is bathed in a water solution). The forces that these water-loving or water-hating amino acids apply cause the protein to fold into the shape that determines its function.

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4. Molecular Genetics I

CJ Fearnley

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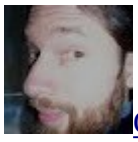
-  CJ Fearnley - 2013-04-17 19:17:39-0400 - Updated: 2013-07-13 15:44:11-0400

Molecular Genetics II. 1h 14m Robert Sapolsky video.

Mutations in a splicing factor can lead to completely new proteins. Mutations in a promoter could change the transcription factor and lead to whole networks of new proteins. That is, mutations in the epigenetic infrastructure can lead to BIG evolutionary changes.

When comparing different species, the more genes the greater is the percentage that are transcription factors. Given a set of n genes, you need 2^n - 1 transcription factors to control every combination of them. A disproportionate share of the differences between the human and chimp genomes are genes that code for transcription factors. Microevolution is about the function of proteins; macroevolution is about which proteins and hence is far more consequential. Macroevolution is about new proteins, new networks, and new if-then clauses. E.g., compared to the chimp, the human immune system is better against tuberculosis but worse against malaria.

Barbara McClintock (1902-1992) was a distinguished plant geneticist developing pathbreaking new techniques in her early work. In 1944, she became the third woman elected to the National Academy of Sciences. But then she made a career ruining discovery: her data on maize implied that there are transposable genes or transposons (so-called "jumping genes"). After decades of reproach, the evidence that she was right finally led in 1983 to her becomming the only woman to receive an unshared Nobel prize in Physiology or Medicine. Plants have an inducible transposable genetic capability as part of their stress response. Animals and humans have a similar capability in their immune response with antibodies. In primates, neural progenitor cells, which make new neurons, have an inducible genetic shuffling. "The cells in your body that have the greatest to do with making you who you are are the least constrained by genetic determinism."

Copy number variants are where there are multiple copies of a gene. The copies could be backups or the effect may be proportional to the number of copies. This gives evolution the opportunity to "experiment" with copies of a gene to try out new things while having the original functionality persist.

Some examples. Vasopressin is a hormone that affects the social affiliative behavior of male voles (and possibly humans!). By controlling a promoter (gene therapy), a polygamous male can be made monogamous. Human males with one promoter are more likely to get divorced than other forms. What does that say about free will?

Dynorphin is a hormone neurotransmitter involved in pain perception. The number of copies of a promoter predicts how readily subjects became addicted to drugs.

Steroid hormones like estrogen, testosterone, and glucocorticoids have two parts which are coded on different exons: a hormone binding domain and a DNA binding domain. Hydrocortisone (aka cortisol), one of the glucocorticoids, is a stress hormone that suppresses the immune system. Progesterone could have evolved by replacing the hormone binding domain to suppress the immune system during pregnancy. Joe Thornton has shown that a lot of the steroid receptors were once duplicated genes.

But it is a long shot for any of these mutations to "get something interesting". So we get long periods of equilibrium with not much change happening. Stabilizing selection will weed out most changes. There can also be selective bottlenecks where only those with a particular trait make it. This happened with Cheetahs about 200 kya: all Cheetahs are so genetically similar that they can take tissue transplants with little risk of complications.

He closes with some evidence for both macro and micro changes in genes that have been noticed. The fossil record shows some evidence for both macro- and micro-evolution. In Chicago over the past 100 years, we know the gene distributions are changing (someone preserved some 100 year old rats that have been compared to modern Chicago rats). The Pima Indians are split about half and half between Mexico and the US. There is a 96% rate of diabetes on the US side. Siberian silver foxes were domesticated for tameness in 35 generations. As a side effect, we get floppy ears, wagging tails, and piebald coloration. Both macro and micro mutation is happening.

5. Molecular Genetics II

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CJ Fearnley's post

CJ Fearnley

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More notes in the first comment.

youtube.com
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CJ Fearnley
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CJ Fearnley
Here is one of the vasopressin that Sapolsky appears to be referencing: http://www.pnas.org/content/105/37/14153.full

PNAS.ORG
Genetic variation in the vasopressin receptor 1a gene (AVPR1A) associates with pair-bonding behavior

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CJ Fearnley
Here is a more readable rendition: http://www.telegraph.co.uk/.../Divorce-gene-linked-to...

TELEGRAPH.CO.UK
'Divorce gene' linked to relationship troubles - Telegraph

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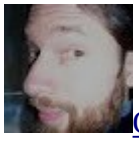 CJ Fearnley - 2013-05-02 19:06:48-0400 - Updated: 2013-05-02 19:06:48-0400
Behavioral Genetics I. 1h 38m Robert Sapolsky video.

This video explores the ways in which we can determine the genetic bases of behavior. Sapolsky defines behavioral genetics: "look for patterns of shared traits among individuals who have differing degrees of shared genes and infer relatedness and infer genetic influences from that." The lecture then documents and exposes deep flaws in several of the approaches which have been attempted (some of which, despite their flaws, have significantly influenced our culture). The lecture not only warns us that many behavioral genetics studies do not stand up to reasoned biological scrutiny, it helps us to deeply understand the challenges that behavioral genetics studies need to address to succeed. The fundamental concern in all these studies is how effectively does the study control for the influence of environment. And the short answer is that many have failed to be convincing upon close examination.

Topics discussed include twin studies, the 1980 study on "Sex differences in mathematical ability", adoption studies (with animals this is known as "cross fostering"), and that weird and wonderful world of identical twins separated at birth (yes, Sapolsky finds deep, deep flaws in these studies too!!!). One of the most fascinating discussions is on the amazing discoveries about how important the pre-natal environment is in fixing phenotypes that we might otherwise naively assume to be genetic! Another is on "non-mendelian inheritance of traits". What? Has Lamarck been vindicated? Partially: modern biology now has evidence for some limited Lamarckian inheritance!!! Blow me

away! Finally, he examines the effects of the cellular material in an egg cell and indirect genetic effects.

Detailed Notes:

The first naive approach to behavioral genetics discussed is "if you see a trait that is universal in a species, obviously its genetic." But environment runs in families and in species too: shared genes and shared environment tend to covary. How can we control for environment?

Twin studies. There are monozygotic (identical) and dizygotic (fraternal) twins. We know that dizygotics are treated differently by parents, differential traits may be caused by parental behavior and not genetics! Moreover, if monozygotic twins split within 5 days, they each get a separate placenta (dichorionic). That happens 1/3 of the time. The other 2/3 of the time, the split happens after 5 days and they share a placenta (monochorionic). Does your twin study account for this environmental difference? Evidently many of them do not! We know that IQ is more highly correlated in monochorionic twins. So environment can certainly be a factor even in twins.

At 1 hour of birth, the average rate of movement of limbs is greater among boys than girls. Sex differences in behavior manifests as early as one hour of birth. Sex differences in the behavior of parents has been seen within moments of birth. Hmm, it is going to be very hard to control for environment even with twin studies.

Benbow & Stanley wrote an enormously influential study in 1980 in Science "Sex differences in mathematical ability: Fact or artifact?". Reader's Digest called it the "math gene" though the study makes much more modest conclusions. The study thought they had controlled for environment by testing "gifted" children before specialization into separate math tracks. Sapolsky observes that as early as first grade, boys are called on and praised more than girls and guidance councellors tend to recommend more math to boys. There are huge environmental differences well before grade 7!

Another approach is with adoption studies (in animal studies this is called cross fostering). Seymour Kety of Harvard studied adoptees in Denmark and determined that in the general population (no family history, and no history in one's adopted family) schizophrenia occurs at a 1% rate. If one's adoptive family has schizophrenia but your biological family has none, then the incidence rate is 3%. If the adoptive family has no schizophrenia but your biological family does, the rate is 9%. If both the adoptive family and your biological family have schizophrenia, the rate is 17%. Sapolsky tears it apart: since adoption doesn't typically happen within nanoseconds of birth, both the prenatal effects and early effects after birth are not controlled. In addition, adoption is not random: adoptees tend to be placed in families that are like the child's biological family.

Tom Bouchard at U Minnesota has studied identical twins separated at birth. 40 pairs were included in the initial studies although 200 have now been identified. Due to the penchant of humans to find pattern even where it doesn't exist, the first studies were bizarre (that's Sapolsky's "technical" term). For example, it was reported that some twins both flush the toilet before and after use. The literature has also provided some more noble results: IQ, introversion/extroversion, and degree of aggression are all about 50-50 genetic-environmental. Since adoption is not random and the sample size is so small, it is not clear how much capital to invest in such studies.

Some behavioral traits occur in the absence of any learning or experience. For example, all babies and fetuses smile. It is a basic motoric pattern. Congenitally blind babies start smiling at the same time of life. Congenitally deaf kids start babbling at the same age as all kids. That suggests it could be genetic.

Then he discusses the fascinating and important world of pre-natal environmental effects. Blood in the prenatal environment is shared. Fred vom Saal showed that there is a different hormonal environment depending on the sex of neighboring fetuses (rats can have 12 pups at once). The more male siblings around a female yields a later onset of puberty. These endocrine (blood-based hormone system) effects can have long-lasting impact.

If mom is stressed during pregnancy, she will have more glucocorticoids in her blood which is shared with her fetus. Her kids will have smaller brain size, a thinner cortex, less learning ability, will be more prone to anxiety, fewer benzodiazepine receptors, more cognitive decline in old age, and girl children will have elevated glucocorticoids in their pregnancies and so the effect is passed on to the grandkids. What?! Inheriting a non-genetic effect!! That's not supposed to happen, Lamarckian inheritance was thoroughly rejected in my high school biology course. The biologists are sensitive to this history and so they call the phenomenon "non-Mendelian inheritance of traits".

Nutrients are also shared with the fetus. In the Winter of 1944 the Nazis diverted food from Holland to Germany and the result is now called the Dutch Hunger Winter. Third trimester fetuses during the Dutch Hunger Winter have a thrifty phenotype: their pancreas more aggressively secretes insulin to store all available calories, their kidneys retain more salt. Adults from these third trimester fetuses of the Dutch Hunger Winter have a 19-fold increase in obesity, hypertension, diabetes, and metabolic syndrome (a complex of several, usually at least three, of the following factors: abnormal blood fat, high blood pressure, fatty liver, insulin resistance, and new fat deposition). This phenomenon has launched a new field "fetal origins of adult disease". Biologists have traced the effect of the thrifty phenotype to the epigenetic coding for insulin. Interestingly, the effect only occurs if the starvation happens during the third trimester, in the Lennigrad starvation which was more gradual there is no thrifty phenotype effect.

Another example of pre-natal environmental effects is when fetuses are exposed to more phytoestrogens (plant-based "dietary estrogens") there is a higher risk of estrogen dependent breast cancer (Wikipedia says there are conflicting studies). If flavored sucrose is added to the amniotic fluid (evidently they drink it!), after birth the baby will prefer that flavor (tested by measuring the "sucky motions" of newborns which is believed to represent an indication of preference).

Mothers reading to their fetuses results in their newborns responding with "sucky motions" when the same story is read after birth. Father's voices don't carry through the thoracic diaphram so well and their stories have no effect.

The age of the mother at the birth of her child correlates to her estrogen levels (lower when she is young and tapering off to be lower again in old age). The age of a child reaching puberty is closely correlated to this effect (higher age of puberty when the estrogen levels are low). I wonder how this correlates to the historical averages for age of onset of puberty and what this tells us about how Mom's age has changed through history?

Rats have been bred for different types of anxiety traits: apparently these are genetic traits. Darlene Francis of Berkeley developed a surgical technique to transfer fetuses to foster mothers. It turns out that the anxiety traits were not genetic, something about the fetal-mother relationship spreads the anxiety trait. Another instance of "non-Mendelian inheritance of traits" or Lamarckian inheritance!

You get more genetic material from your mother than your father! Your mitochondria come exclusively from Mom. Your father's genes may not come from your father (according to Bob Wyman about 10-15% of children don't have the father they're supposed to have!). The egg that became you was a fully functioning cell which in addition to mitochondria has a full set of proteins including transcription factors (proteins that promote or block genetic expression). If an

environmental toxin knocks out a transcription factor or other vital protein in the egg that becomes you (or at any vital step during development), genetic expression may be unalterably affected for generations to come (Lamarckian inheritance).

The idea of indirect genetic effects comes from psychologist Judith Rich Harris' book "The Nurture Assumption" which has a section on behavioral genetics. She argues that some genetic effects can in turn cause other behavioral effects. For example, one's score on the introversion-extroversion scale may be mediated by the genetic effect of height: people who are treated better become more extroverted (studies have shown that height is correlated in this way). In birds, heritability of pecking order has been shown to be mediated by the melanism or color and iridescence of the feathers. The heritability of chicks to eat grubs is mediated by their interest in their feet which are genetically programmed to be "interesting". There is a 70% heritability in Americans of political party affiliation which is apparently mediated by one's tolerance for ambiguity (conservatives are less tolerant of ambiguity than liberals --- what does this have to do with Kohlberg's scale of moral development?). Rat strains that show heritability of aggression appear to be mediated by tolerance to pain (more aggressive rats have a lower threshold for pain sensitivity). Many behavioral and personality traits are now known to be caused by indirect genetic effects.

Michael Meaney at McGill University found that the effect of petting rats for 3 minutes (vs. a deleterious effect from long petting/removal sessions) causing greater resistance to several neurological insults, lower glucocorticoids, bigger brains, and better learning ability is mediated by the mother's response to the absence of the pup (she gives prolonged licking and attention to the ones that have been temporarily removed and neglects the ones that were absent for long periods). The effect is multi-generational: Mom's attention affects transcription factors for receptors to stress hormones and estrogen. The effect is reversible: if half of one's childhood is spent with a negligent mom and the other half with an attentive mom, the epigentic pattern changes back. Behavior & personality traits is often caused by indirect genetic effects.

6. Behavioral Genetics I

CJ Fearnley's post

CJ Fearnley

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Behavioral Genetics I. 1h 38m Robert Sapolsky video.
This video explores the ways in which we can determine the genetic bases of behavior. Sapolsky defines behavioral genetics: "look for patterns of shared traits among individuals who have differing degrees of shared genes and infer relatedness and infer genetic influences from that." The lecture then documents and exposes deep flaws in several of the approaches which have been attempted (some of which, despite their flaws, have significantly influenced our culture). The lecture not only warns us that many behavioral genetics studies do not stand up to reasoned biological scrutiny, it helps us to deeply understand the challenges that behavioral genetics studies need to address to

succeed. The fundamental concern in all these studies is how effectively does the study control for the influence of environment. And the short answer is that many have failed to be convincing upon close examination.
Topics discussed include twin studies, the 1980 study on "Sex differences in mathematical ability", adoption studies (with animals this is known as "cross fostering"), and that weird and wonderful world of identical twins separated at birth (yes, Sapolsky finds deep, deep flaws in these studies too!!!). One of the most fascinating discussions is on the amazing discoveries about how important the pre-natal environment is in fixing phenotypes that we might otherwise naively assume to be genetic! Another is on "non-mendelian inheritance of traits". What? Has Lamarck been vindicated? Partially: modern biology now has evidence for some limited Lamarckian inheritance!!! Blow me away! Finally, he examines the effects of the cellular material in an egg cell and indirect genetic effects.

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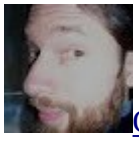 CJ Fearnley - 2013-05-04 08:40:13-0400 - Updated: 2013-05-04 08:40:13-0400
Behavioral Genetics II. 1h 32m Robert Sapolsky video.

After building our critical thinking skills in evaluating behavioral genetics studies in the previous lecture, Sapolsky now describes the modern approach to the field. Since the 1980s, the emphasis for any attribution of behavior to genetics has taken the mantra of "go and find the genes". This can be done by looking at two populations of traits or proteins and finding genetic markers. Or by starting with a gene and looking for traits that it expresses (this is problematic because the object of our study will tend to be the preferred explanation for any implicated phenotypic behavior rather than objectively searching for the factors contributing to that trait). He describes several genes whose behavioral effects are known, but admits they only explain a small part of the variation in behavior that exists.

Then he explains how the Nature / Nurture dichotomy is missing its third leg: chance! Chance plays a big role which he explains in terms of the mechanism of cell division itself. He spends most of the rest of the lecture trying to explain that "heritability" means the part of behavior that is NOT explained by genes (which he claims is not what most people think it means!). This discussion exposes the importance of the gene-environment interaction: all genes encode for if-then clauses about environmental effects! Fasten your seat belts, this discussion gets very strange and surprising at times! The bottom line is that the nature / nurture dichotomy is nonsense: genes act on the environment and vice versa: it is a both-neither situation!

Detailed Notes:

At first, finding the genes affecting behavior was done by statistically comparing the genomes of two populations (one with the trait and the other without it). Generally this means sifting through the genes to find a genetic marker (which is not the gene itself; and note that many diseases probably involve several genes). Approaches to examine two distinct proteins (by chemical or physical properties) have also been developed. Pat Brown at Stanford helped perfect the DNA microarray technique which can now coax a cell to transcribe all of its genes to RNA which can be used to make a biochip to assess genomic differences. QTL (quantitative trait loci) can be used to look at more complex genetic patterns.

The first successes came from diseases where the traits are clearly distinguished by "you've got it" or "you don't". Phenylketonuria (PKU), Huntington's disease, and cystic fibrosis were found to be genetic. Because these approaches are inherently statistical, there is always a possibility of false positives. This has spurred the development of extra care in the science (are you sure you got the statistics right?) and new work in bioethics: should we terminate this pregnancy if the child has a 94.2% chance of contracting Huntington's disease sometime between their teen years (unlikely) and old age (they may have lived a full healthy life before onset)? If someone just cared for their parent dying of a horrible genetic disease should we offer to test them and then have to tell them that there is a high (but not 100%) probability that they have the bad gene. What if they then jump from a tall building?

Some actual behavior affecting genes have been found. As discussed in the lecture on Molecular Genetics II, the vasopressin gene marker in voles distinguishes between monogamous and polygamous males. The same genes in humans affect how males handle social connectedness. The BDNF (brain-derived neurotrophic factor) gene prompts neural growth in the Amygdala (the part of the brain dealing with fear and anxiety). Variants of the BDNF gene studied in rats also explains human anxiety and depression. Dopamine receptor D4 also shows variations in rats and humans with similar effects in both species which explains human risk-taking, sensation-seeking, and novelty-craving behaviors. Neuropeptide Y (NPY) is a neurotransmitter with variants in its promoter which also has effects in the Amygdala. Sapolsky emphasizes that all of these genetic variations explain only tiny differences in the behavior.

The vital role of chance. Not all of the mitochondria in a cell are genetically identical. When the egg that becomes you splits in meiosis (sexual reproduction), roughly half of the mitochondria go into each daughter cell. Statistically, there is some chance that you will get a different mix of types of mitochondria from your mom. Moreover, at every mitotic cell division the mix of mitochondria may change. Not only does each daughter cell get a mix of mitochondria, but chance affects the mix of protein and transcription factors that make it into each daughter cell. Even if you know all the genes, you can't know where each molecule will be when the cell divides!

In behavioral genetic studies, the number that makes it into headlines is called "heritability": Y% of the variability in trait X is heritable. What does that mean? Sapolsky warns: not what you think!!! What heritability really means is now non-heritable the trait is! Heritability tells what genes have to do with the degree of variability observed in the trait. But then Sapolsky explains how science works: best practice dictates that you must control for extraneous environmental factors. That is, you don't run the experiment in the Gobi Desert, in a temperate climate, in the tropics, and at the poles. You just run it once and determine the heritability of the traits that are expressed in that environment. But this practically guarantees that the genetic component will be overestimated!!! So science is always giving us exaggerated values for heritability! Hmmph, even after getting down to the level of actual genes, behavioral genetics still falls quite far from "hitting the mark".

Then he discusses gene-environment interaction: genes behave differently depending on the environment. So it is impossible to say what a gene does, you can only say what the effect of a gene

is in the environments studied so far! He gives an interesting example: the number of fingers you have is 5, 4, 3, 2, or 1 depending on how many accidents you have had. So the heritability of number of fingers is 0% (all variability is explained by industrial accidents). In 1952 Eisenhower America, there was a 0% change that a human male would have an earring and a 100% chance that, at that time, a human female would have an earring. So earrings were a 100% heritable trait! Heritability is a strange concept, no? Sapolsky concludes: it is meaningless to ask what a gene does as it can only be answered vis-a-vis the environment in which you test it. The genes code for environmental interactions, that's what they do!

Then he gives some more relevant examples of gene-environment interaction. In phenylketonuria (PKU) the body's ability to metabolize the amino acid phenylalanine is genetically missing. The phenylalanine builds up and destroys your brain. This is a 100% heritable trait. By eating a diet that severely restricts or eliminates foods with phenylalanine in them, patients can lead healthy lives! A heritable trait which environment can control? Doesn't that mean that heritability should be 0%? This is truly weird!

From animal studies on serotonin (a neurotransmitter) a genetic variant leads to more depression (via an interaction with glucocorticoid stress hormones). But in humans it was discovered that the effect is mediated by a gene-environment interaction: the number of environmental stress events (childhood traumas such as losing a parent). Surprisingly, the "bad" gene results in fewer(!) depressions if you had no childhood traumas. In animal studies a variant of the enzyme monoamine oxidase (MAO) predisposes subjects to more aggression than the other variant. In humans a study suggests that it is correlated with antisocial (what used to be called sociopathic) behavior. A similar effect is seen with another serotonin factor, but that one is mediated by child abuse. Another study has a gene mediating social attachment if and only if the mother is cold and withdrawn. Another gene gives some predictability for IQ if and only if the subject was breast fed.

Sapolsky cites Paul Ehrlich for the quote "asking whether genes or environment have more to do with some trait is akin to asking whether length or width have more to do with the area of a rectangle" (Wikipedia credits Donald Hebb with a similar quote; I modified Sapolsky's version to be mathematically accurate). There is no such thing as a gene influence outside the context of an environmental interaction.

Many, many studies have shown that having a Y chromosome (that is, being male) predicts better math skills. Paola Sapienza and colleagues analyzed data from a test designed by the OECD and administered in 40 countries to 276,165 15-year olds. She discovered that the gender gap is mediated according to a country's score on the The World Economic Forum's Gender Gap Index (GGI). Iceland even has females outperforming males in math and two Scandinavian countries were at statistical parity. It turns out that males tend to outperform females at math in those societies with a gender gap! Here is the math study if you are interested:
http://www.kellogg.northwestern.edu/faculty/sapienza/htm/science.pdf

My conclusion: Nature / Nurture is another one of those "both-neither" phenomena!

In lecture 5 on Molecular Genetics II, Sapolsky discussed the work of Dmitri Belyaev's team in "domesticating" the silver fox in some 35 generations. He starts this lecture by describing the street dogs of Moscow studied by Andrei Poyarkov who has observed that over time these dogs have reacquired wolf-like characteristics such as a longer muzzle, more erect ears, loss of a piebald coloration.

7. Behavioral Genetics II

CJ Fearnley

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Behavioral Genetics II. 1h 32m Robert Sapolsky video.
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Then he explains how the Nature / Nurture dichotomy is missing its third leg: chance! Chance plays a big role which he explains in terms of the mechanism of cell division itself. He spends most of the rest of the lecture trying to explain that "heritability" means the part of behavior that is NOT explained by genes (which he claims is not what most people think it means!). This discussion exposes the importance of the gene-environment interaction: all genes encode for if-then clauses about environmental effects! Fasten your seat belts, this discussion gets very strange and surprising at times! The bottom line is that the nature / nurture dichotomy is nonsense: genes act on the environment and vice versa: it is a both-neither situation!

youtube.com
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CJ Fearnley
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CJ Fearnley
In behavioral genetic studies, the number that makes it into headlines is called "heritability": X% of the variability in trait X is heritable. What does that mean? Sapolsky warns: not what you think!!! What heritability really means is now non-heritable the trait is! Heritability tells what genes have to do with the degree of variability observed in the trait. But then Sapolsky explains how science works: best practice dictate that you must control for extraneous environmental factors. That is, you don't run the experiment in the Gobi Desert, in a temperate climate, and at the poles. You just run it once and determine the heritability of the traits that are expressed in that environment. But this practically guarantees that the genetic component will be overestimated!!! So science is always giving us exaggerated values for heritability! Hmmph, even after getting down to the level of actual genes, behavioral genetics still falls quite far from "hitting the mark".
Then he discusses gene-environment interaction: genes behave differently depending on the environment. So it is impossible to say what a gene does, you can only say what the effect of a gene is in the environments studied to date! He gives an interesting example: the number of fingers you have is 5, 4, 3, 2, or 1 depending on how many accidents you have had. So the heritability of number of fingers is 0% (all variability is explained by industrial accidents). In 1952 Eisenhower America, there was a 0% change that a human male would have an earring and a 100% chance that, at that time, a human female would have an earring. So earrings are a 100% heritable trait! Heritability is a strange concept, no? Sapolsky concludes: it is meaningless to ask what a gene does as it can only be answered vis-a-vis the environment in which you test it. The genes code for environmental interactions, that's what they do!
Then he gives some more relevant examples of gene-environment interaction. In phenylketonuria (PKU) the body's ability to metabolize the amino acid phenylalanine is genetically missing. The phenylalanine builds up and destroys your brain. This is a 100% heritable trait. By eating a diet that severely restricts or eliminates foods with phenylalanine in them (careful it is found in breast milk), patients can lead healthy lives! A heritable trait which environment can control? Doesn't that mean that heritability should be 0%? This is truly weird!

From animal studies on serotonin (a neurotransmitter) a genetic variant leads to more depression (via an interaction with glucocorticoid stress hormones). But in humans it was discovered that the effect is mediated by a gene-environment interaction: the number of environmental stress events (childhood traumas such as losing a parent). Surprisingly, the "bad" gene results in fewer(!) depressions if you had no childhood traumas. In animal studies a variant of the enzyme monoamine oxidase (MAO) predisposes subjects to more aggression than the other variant. In humans a study suggests that it is correlated with antisocial (what used to be called sociopathic) behavior. A similar effect is seen with another serotonin factor, but that one is mediated by child abuse. Another study has a gene mediating social attachment if and only if the mother is cold and withdrawn. Another gene gives some predictability for IQ if and only if the subject was breast fed.
Sapolsky cites Paul Ehrlich for the quote "asking whether genes or environment have more to do with some trait is akin to asking whether height or width have more to do with the area of a rectangle" (Wikipedia credits Donald Hebb with a similar quote). There is no such thing as a gene influence outside the context of an environmental interaction.
Many, many studies have shown that having a Y chromosome (males) predicts better math skills. Paola Sapienza analyzed data from a test designed by the OECD and administered in 40 countries to 276,165 15-year olds. She discovered that the gender gap is mediated according to a country's score on the The World Economic Forum's Gender Gap Index (GGI). Iceland even has females outperforming males in math and two Scandinavian countries were at statistical parity. Males tend to outperform females at math in those societies with a gender gap!
My conclusion: Nature / Nurture is another one of those "both-neither" phenomena!
In lecture 5 on Molecular Genetics II, Sapolsky discussed the work of Dmitri Belyaev's team in "domesticating" the silver fox in some 35 generations. He starts this lecture by describing the street dogs of Moscow studied by Andrei Poyarkov who has observed that over time these dogs have reacquired wolf-like characteristics such as a longer muzzle, more erect ears, loss of a piebald coloration.
Here is the math study if you are interested:
http://www.kellogg.northwestern.edu/.../htm/science.pdf

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The Biology of Learning

Details

What can behavioral biology tell us about learning? Are there hard-wired learning behaviors in animals and humans? What is the scope of learning in the natural world? This biological context reveals an interesting and possibly unfamilar perspective on human behavior and learning. It will also give us a chance to discuss the wonderful world of animal behavior and learning.

This discussion is based on two lectures from Robert Sapolsky's free on-line course (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D) "Human Behavioral Biology (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D)". Both videos are long (roughly an hour and a half each). Both are exquisite, fairly introductory, yet richly detailed and

complex (like their subject). The two videos stand fairly well on their own, but they do assume some background from previous videos in the course.

Due to strong interest in this topic, "The Biology of Learning" will be repeated on Sat Nov 30th (https://www.meetup.com/thinkingsociety/events/148844402/).

• Recognizing Relatives (http://www.youtube.com/watch?v=P388gUPSq_I). We will focus on the last 50 minutes of this video (the first 30 minutes is a review of the first 7 lectures in the course and is excellent, but slightly out of scope for this discussion). The technical word "spandrel" (meaning traits that have evolved as the incidental byproduct of adaptive traits) is the only technical word that you might not know. The video does assume some familiarity with evolution by natural selection (https://en.wikipedia.org/wiki/Natural_selection) and the "lock and key model" for protein function. Here are two summaries of the lecture: My notes on Sapolsky's lecture on Recognizing Relatives (https://plus.google.com/104222466367230914966/posts/ELj4gs7WARi) and A Sapolsky fan's notes on Recognizing Relatives (http://robertsapolskyrocks.weebly.com/recognizing-relatives.html).

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Questions that we might discuss from this video:

How does the major histocompatibility complex affect innate understanding of self, other, and relative in organisms and humans? How can pheromones affect human (and animal behavior)? How can social anosmias (inability to smell) affect humans? How do humans, baboons, and other animals recognize their relatives (so that evolutionary theories about kin selection and cooperation can apply to the world of real organisms)? How is human mating affected by living with someone during their youth? What is pseudo-kinship (http://en.wikipedia.org/wiki/Fictive_kinship#Use_in_sociobiology)? What is pseudospeciation (https://en.wikipedia.org/wiki/Pseudospeciation)? How does this context provide important background for understanding learning?

• Ethology (http://www.youtube.com/watch?v=ISVaoLlW104). This lecture introduces ethology (the study of animal behavior) framed by the field of psychology (with a particular emphasis on behaviorism which was founded by John B. Watson and led by B. F. Skinner). It provides deep insights into the unique approach of ethology to the study of behavior including human behavior. It is also a fascinating introduction to the subfields of neuroethology and cognitive ethology. Although we will focus on learning, we will discuss some of the broader issues the video raises about ethology and behavioral psychology. Here are two summaries of this lecture: My notes on Sapolsky's lecture on ethology (https://plus.google.com/104222466367230914966/posts/YUnRBsoio66) and A Sapolsky fan's notes on ethology (http://robertsapolskyrocks.weebly.com/ethology.html).

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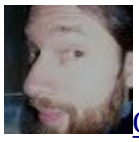
Is maternal competence a learned behavior or innate? What animal behaviors did Sapolsky describe that surprised you? Can some animals learn with just one attempt? Are some animals (and humans!) innately prepared for certain types of learning? What is "theory of mind"? Do some animals have theory of mind? Can animals distinguish intentional from accidental behavior? Can ethology determine if animals have awareness? Do some animals have numerosity (a sense of number) and sophisticated logic skills? What take home message can we draw about the nature of the tools of ethology for revealing the breadth and depth of animal behavior especially with respect to the role of learning in animals and humans.

What major advance did behavioral psychology contribute to our understanding of human and animal behavior? What problems does Sapolsky find with behaviorism and its flagship reinforcement learning as it was influentially pushed by B. F. Skinner and others? Since Sapolsky didn't highlight many benefits of behaviorism, should we infer that it is now disreputable? Can behaviorism make a comeback? How ought we value the contributions of both ethology and behavioral psychology as approaches to understanding human behavior? Is thinking about subdisciplines in this way distorting? How ought we view the various paradigm changes that run through sciences like biology?

I have led three prior discussions on Robert Sapolsky whose notes and references you might enjoy: The Uniqueness and Evolution of Humans (15 Apr 2012) (https://www.meetup.com/thinkingsociety/events/50061542/), The Evolutionary and Genetic Bases of Human Behavior (14 Jul 2013) (https://www.meetup.com/thinkingsociety/events/116303742), and The Evolutionary and Genetic Bases of Human Behavior (27 Jul 2013) (https://www.meetup.com/thinkingsociety/events/128381212). The latter two discussions covered videos 2-7 of Sapolsky's course BIO 250, HUMBIO 160: Human Behavioral Biology (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D).

 CJ Fearnley - 2013-10-27 09:51:53-0400 - Updated: 2013-10-27 09:51:53-0400
Join me in two weeks to discuss two Robert Sapolsky videos on the basics of the behavioral biology of learning: "The Biology of Learning". We will discuss instinct, imprinting, prepared learning, and other qualities of how humans and other animals know their relatives, how to nurse, how to deal with heights, snakes, and social dominance. What can the field of ethology and neuroethology tell us about human behavior which behaviorists like B. F. Skinner could not?

http://www.meetup.com/thinkingsociety/events/144382142/

The Biology of Learning

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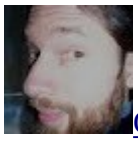
Events in Philadelphia, PA

- 
- CJ Fearnley - 2013-11-22 10:43:29-0500 - Updated: 2013-11-22 10:43:29-0500

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The Biology of Learning

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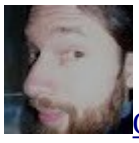
CJ Fearnley
Andrew Moberly: As I'm preparing for this discussion tomorrow and reflecting on how much the prior discussion on Nov 10th focused on smell, I'm wondering if you interest in smell neurology is profoundly interesting. The first Robert Sapolsky lecture cited in the event description discussed Martha McClintock's research that we are most attracted by the scent of our 3rd cousins, the possible role of social anosmias in autism, the way prolactin stimulates neurogenesis in the olfactory bulb during pregnancy, the research that we are affected by the fear in the scent of skydivers. It all makes me wonder if smell doesn't drive human behavior in much more profound ways than most of us imagine. Should we develop, practice and strengthen our smell awareness? How deeply tied into human behavior is our smell? How much of our learning is smell-based?

 CJ Fearnley - 2013-05-13 19:41:06-0400 - Updated: 2013-10-07 18:21:08-0400
Recognizing Relatives. 1h 20m Robert Sapolsky video.

The first half-hour is an effective review of the course to date. He summarizes behavioral genetics (the preceding two lectures) by these two statements: "The fact that humans usually have 5 fingers reflects the fact that this trait is inherited. The fact that when there are some circumstances of humans having other than 5 fingers, it is overwhelmingly due to environment is an indication that nevertheless the variability (heritability) is essentially 0%." Does that clear it up for you?

Then he reviews the definition of epigenetics at three levels: 1) the way environment affects biology (genetics), 2) the way environment turns genes on and off, and 3) the regulation of chromatin remodeling, methylation of genes, etc.

Sapolsky uses a review of two studies to help us understand how to be critical of the scientific literature and learn what questions to ask to put such studies into perspective. He is brilliant. The effect is masterful. One of the studies proved that first borns have a higher IQ than later borns. Among the many questions that we as critically thinking reviewers of a scientific study ought to ask are Did the study effectively control for other factors? How good is the science / statistics? And critically, how big is the effect? Is the 2.3 IQ point advantage of first borns meaningful?

Then the last 50 minutes of the video discusses the problem of how do organisms recognize kin (relatives)? There are three basic means of recognizing relatedness: 1) innate which is effectively olfactory (of the sense of smell) 2) learning or imprinting (learning that occurs at a particular phase such as right after birth), and 3) cognitively (thinking it out).

The Details:

The major histocompatibility complex is a protein that provides a unique signature of "self" and is presented on the cell membrane of every cell in the body. It is essential for immune functioning. Sapolsky suggests that pheromones (chemical triggers of social behavior) may signal both uniqueness ("self") and relatedness. The "self" protein may be built from an arguably unique juggle of the genes (randomness) plus some way of capturing the pattern that is your genome which is shared more closely with closer relatives. It is probably similar (or identical) with the mechanism used for the immune function. Receptors could sense relatedness by how good a lock-and-key protein fit is made.

In the olfactory bulb, the hormones oxytocin and vasopressin are active in mediating pheromone detection by increasing the number of receptors thereby increasing sensitivity. There is an emerging literature suggesting that mutations in the genes related to oxytocin and vasopressin affect autistic families. Autism is characterized by enormous deficits in normal socialization, interaction, social bonding, and social affiliation. Anosmia is the inability to smell. A social anosmia refers to being unable to distinguish between individuals.

Adult neurogenesis (generating new nerve cells) only happens in two areas of the brain: 1) the hippocampus (associated with learning & memory) an enriching environment stimulates neurogenesis whereas stress generates less and 2) behind the olfactory bulb in females around the time of pregnancy prolactin stimulates neurogenesis to build a revamped olfactory system to be ready at birth (this could be the reason that smell and taste are weird during pregnancy: it might be a

spandrel!).

Martha McClintock (the one who published on the Wellesley effect) tested swabs of armpits to determine that humans have an innate ability to distinguish degree of relatedness: she determined that our 3rd cousins have the most "appeal" (a 3rd cousin is the optimal level of relatedness to minimize genetic effects of matings with close relations but maximize kin selection).

Some species do not have this innate mechanism. Imprinting is how many organisms learn who Mom is and how Mom learns who is their kid. The learning is innate, but it takes experience to set the learning upon a specific individual. Different species use different degrees of smell, sound, and sight.

Some animals (including humans) recognize relatives cognitively (through thinking). Sapolsky reports that baboons do it based on a statistical kind of thinking. David Sloan Wilson has detected cognitive effects in sun fish: if a male sees another male with his mate, he won't take care of the kids. In humans, sheep, pigeons, and primates, the fusiform cortex (use "fusiform face area" in Wikipedia) supports facial recognition (it is inactive in autistic children). At birth mothers and their babies have olfactory recognition: babies tend to turn their heads towards Mom's smell. There is also some imprinting from hearing Mom's voice in utero and making more sucky motions indicating interest when she repeats those sounds after birth.

Anthropologist Joseph Shepher found that humans who played extensively together before the age of 6 (in a large study of Israeli kibbutzim), never married or had relations with those peers. Arthur Wolf, an anthropologist at Stanford, found that arranged marriages in Taiwan between couples who were raised together from a young age were disastrous. Wikipedia's article "Incest taboo" suggests that the effect is not supported by all studies.

Pseudo-kinship is the phenomenon in humans wherein non-relatives feel like a band of brothers (e.g., military units). Pseudospeciation is where we are manipulated to perceive some humans as hardly counting as being human at all. Are these unique traits of humans? Could we represent them in a positive, non-pathological framing or are these troubling effects?

8. Recognizing Relatives

CJ Fearnley

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Details in the comments.

youtube.com
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- 12yCJ Fearnley's post

CJ Fearnley

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Another way to understand the biology of behavior is through ethology whose approach is characterized by 1) study behavior in lots of different environments 2) study behavior in the natural environment (get out of the lab), and 3) assume you are going to have to translate a language. Nobel laureate Nikolaas Tinbergen says "ethology is the process of interviewing an animal but in its own language."
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Then Sapolsky gives several examples showing that animals do not need to learn many behaviors, but they do need to learn how to do the behavior better and they need to learn the right context for their innate behaviors. Visual cliff response behavior: in the lab put babies of various species on a plate of glass that is "very high" to give them the visual sensation that they just fell off a cliff. As you might expect the animals experience terror (a panic response behavior). Excepting baby sloths who show no fear of the situation: aha, a species-specific behavior refuting the behaviorists claim that you can experiment on any animal with the same results. Once the babies learn that the glass is safe, the panic behavior gradually wanes.

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John Wingfield is doing neuroethology studies in the field. Sapolsky does research on baboons and anxiety in the field in Africa. He observes that we need the ethological context in order to make sense of the brain and what sensory systems it pays attention to so that we can study neuroethology. In this context, the fixed action patterns are the behavior, the releasing stimulus is the trigger, and the innate releasing mechanism are the neuro-hormonal circuitries.

What is the role of learning?

Maternal competence in monkeys is not instinctual, not a fixed action pattern: it must be learned. Rhesus monkey offspring survive better if their mothers have an older sister. More aunting behavior and chimp/monkey children survive better. Similarly, if mom was a Big Sister. Mothering is not instinctual? Does it work that way in humans? Should parenting school be a requirement for pregnant women and their prospective fathers?

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Learning is enhanced in those who pay attention better (since female chimps learn tool-making better, were females the first tool makers?). Those who live in larger social groups tend to learn more younger. Learning by experience is of course very important. Male chimps have been observed fashioning weapons from large pieces of wood.
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Humans and other primates have prepared learning for fear of snakes and spiders, but there are many cultures with no fear of spiders or snakes so it is not innate. We also have prepared learning to notice animate objects over inanimate ones (also snake shaped objects). Martin Seligman provided the interesting example of Sauce-Béarnaise Syndrome: he developed a taste aversion to Béarnaise sauce months after getting an upset stomach even though a performance was closer in time than his eating of the sauce. We have prepared learning to associate upset stomach with food violating a key principle in reinforcement theory.
Cognitive ethology attempts to discover the mechanisms in the head of the animal, the actual mechanics of the innate releasing mechanisms. In the book "Question of Animal Awareness" (1976) Donald Griffin (who discovered echolocation in bats) outlined an initially controversial research program to discover the cognitive and emotional awareness of animals. We now know that animals have strategic awareness and all sorts of other unexpected cognitive capabilities.
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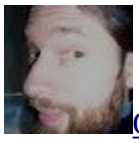 CJ Fearnley - 2013-10-31 14:33:13-0400 - Updated: 2013-11-30 00:13:02-0500
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Brain Science and Human Behavior

Details

Due to strong interest in this topic, "Brain Science and Human Behavior" will be repeated on Saturday 18 Jan (https://www.meetup.com/thinkingsociety/events/158107662/). Please RSVP whichever date works best for you.

How does the emotional brain (the so-called limbic system) work? Is there a rational part of the brain or are we all emotional creatures? Why does every excitatory center in the limbic system seem to have an inhibitory center to counterbalance it? What is the evidence in support of the James-Lang theory of emotions, namely, that the state of your body tells the brain what to feel? If a human brain consists of about 100 billion neurons each with about 10,000 connections (known as synapses) which communicate to each other by pumping neurotransmitters stored in vesicles into the gap between neurons, why are our brains not completely awash in noise and misfirings and confusion? How can individual differences and diverse responses to experience manifest in the working of the brain? Where are concepts & categories stored in the brain? What are the difficulties and challenges in studying the brain?

This discussion is based on a 1½ hour video lecture called "Limbic System" (http://www.youtube.com/watch?v=CAOnSbDSaOw) from Robert Sapolsky's free on-line course "Human Behavioral Biology" (http://www.youtube.com/playlist?list=EC848F2368C90DDC3D) (although the course is introductory in nature and concerns a vitally important subject, this lecture gets a little bit technical at times. Understanding the details is not important, instead I recommend listening to get a sense of the big picture). The video depends on four lectures that introduce some high-level but technical details of Brain Science including the basics of neuroscience and endocrinology.

• The Limbic System (http://www.youtube.com/watch?v=CAOnSbDSaOw). You may be able to follow this video without first watching the four supplemental videos below, but it assumes some familiarity with neuroendocrinology. During the discussion, I will try to explain anything you could not understand during the discussion (although I am not a neuroscientist I have watched each video twice and I've taken extensive notes). Here are my notes on Sapolsky's limbic system video (https://plus.google.com/104222466367230914966/posts/MW1pq8XzT33). Here are the notes of "a Sapolsky fan" on the limbic system (http://robertsapolskyrocks.weebly.com/limbic-system.html).

http://www.youtube.com/watch?v=CAOnSbDSaOw

Background Videos: These videos explain basic ideas about the neuroendocrine system that constitutes modern brain science from the perspective of Human Behavior. Understanding Sapolsky's video on the Limbic System will be significantly improved if you watch these videos first:

• Introduction to Neuroscience I (http://www.youtube.com/watch?v=5031rWXgdYo). My notes on the Neuroscience I video (https://plus.google.com/104222466367230914966/posts/NEv2sBTELNd).

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• Introduction to Neuroscience II (http://www.youtube.com/watch?v=uqU9lmFztOU). My notes on the Neuroscience II video (https://plus.google.com/104222466367230914966/posts/EKwuB88TvAf).

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• Endocrinology (http://www.youtube.com/watch?v=yETVsV4zfFw). My notes on the Endocrinology video (https://plus.google.com/104222466367230914966/posts/7RzgcWMoRBb).

http://www.youtube.com/watch?v=yETVsV4zfFw

• Advanced Neurology and Endocrinology (http://www.youtube.com/watch?v=kAfz0yAcOyQ). My notes on Sapolsky's advanced neuroendocrinology video (https://plus.google.com/104222466367230914966/posts/SR4RiH4CNbJ). The notes of "a Sapolsky fan" on advanced neuroendocrinology" (http://robertsapolskyrocks.weebly.com/advanced-neurology-and-endocrinology.html).

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I have led several prior discussions on Robert Sapolsky whose descriptions (and videos) you may enjoy. The Uniqueness and Evolution of Humans (15 Apr 2012) (https://www.meetup.com/thinkingsociety/events/50061542/) which is based on a Sapolsky lecture. The other discussions have been based on Sapolsky's course BIO 250, HUMBIO 160: Human Behavioral Biology (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D). There were two discussions on "The Evolutionary and Genetic Bases of Human Behavior (https://www.meetup.com/thinkingsociety/events/116303742)" which covered videos 2-7 of the course on 14 Jul 2013 (https://www.meetup.com/thinkingsociety/events/116303742) and 27 Jul 2013 (https://www.meetup.com/thinkingsociety/events/128381212), two discussions on "The Biology of Learning" which covered videos 8 & 9 of t (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D)he course on 10 Nov 2013 (https://www.meetup.com/thinkingsociety/events/144382142/) and 30 Nov 2013 (https://www.meetup.com/thinkingsociety/events/148844402/).

Events in Philadelphia, PA

CJ Fearnley

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Join me on Sunday Jan 12th to discuss "Brain Science and Human Behavior" based on several Robert Sapolsky videos.

meetup.com
Greater Philadelphia Thinking Society
How does the emotional brain (the so-called limbic system) work? Is there a rational part of the brain or are we all emotional creatures? Why does every excitatory center in the limbic system seem to

Brain Science and Human Behavior (repeat)

Details

Due to strong interest in this topic, "Brain Science and Human Behavior" will be repeated on Sunday 2 Feb. (https://www.meetup.com/thinkingsociety/events/161011212/) Since the waitlist is so long, new signups should RSVP to the Feb 2nd edition of the topic.

How does the emotional brain (the so-called limbic system) work? Is there a rational part of the brain or are we all emotional creatures? Why does every excitatory center in the limbic system seem to have an inhibitory center to counterbalance it? What is the evidence in support of the James-Lang theory of emotions, namely, that the state of your body tells the brain what to feel? If a human brain consists of about 100 billion neurons each with about 10,000 connections (known as synapses) which communicate to each other by pumping neurotransmitters stored in vesicles into the gap between neurons, why are our brains not completely awash in noise and misfirings and confusion? How can individual differences and diverse responses to experience manifest in the working of the brain? Where are concepts & categories stored in the brain? What are the difficulties and challenges in studying the brain?

This discussion is based on a 1½ hour video lecture called "Limbic System" (http://www.youtube.com/watch?v=CAOnSbDSaOw) from Robert Sapolsky's free on-line course "Human Behavioral Biology" (http://www.youtube.com/playlist?list=EC848F2368C90DDC3D) (although the course is introductory in nature and concerns a vitally important subject, this lecture gets a little bit technical at times. Understanding the details is not important, instead I recommend listening to get a sense of the big picture). The video depends on four lectures that introduce some high-level but technical details of Brain Science including the basics of neuroscience and endocrinology.

• The Limbic System (http://www.youtube.com/watch?v=CAOnSbDSaOw). You may be able to follow this video without first watching the four supplemental videos below, but it assumes some familiarity with neuroendocrinology. During the discussion, I will try to explain anything you could not understand during the discussion (although I am not a neuroscientist I have watched each video twice and I've taken extensive notes). Read my notes on Sapolsky's limbic system video (https://plus.google.com/104222466367230914966/posts/MW1pq8XzT33). Here are the notes of "a Sapolsky fan" on the limbic system (http://robertsapolskyrocks.weebly.com/limbic-system.html).

http://www.youtube.com/watch?v=CAOnSbDSaOw

Background Videos: These videos explain basic ideas about the neuroendocrine system that constitutes modern brain science from the perspective of Human Behavior. Understanding Sapolsky's video on the Limbic System will be significantly improved if you watch these videos first:

• Introduction to Neuroscience I (http://www.youtube.com/watch?v=5031rWXgdYo). My notes on the Neuroscience I video (https://plus.google.com/104222466367230914966/posts/NEv2sBTELNd).

http://www.youtube.com/watch?v=5031rWXgdYo

• Introduction to Neuroscience II (http://www.youtube.com/watch?v=uqU9lmFztOU). My notes on the Neuroscience II video (https://plus.google.com/104222466367230914966/posts/EKwuB88TvAf).

http://www.youtube.com/watch?v=uqU9lmFztOU

• Endocrinology (http://www.youtube.com/watch?v=yETVsV4zfFw). My notes on the Endocrinology video (https://plus.google.com/104222466367230914966/posts/7RzgcWMoRBb).

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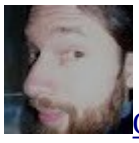
http://www.youtube.com/watch?v=kAfz0yAcOyQ

This topic is a repeat from the one on Sun 12 Jan (https://www.meetup.com/thinkingsociety/events/153389892).

I have led several prior discussions on Robert Sapolsky whose descriptions (and videos) you may enjoy. The Uniqueness and Evolution of Humans (15 Apr 2012) (https://www.meetup.com/thinkingsociety/events/50061542/) which is based on a Sapolsky lecture. The other discussions have been based on Sapolsky's course BIO 250, HUMBIO 160: Human Behavioral Biology (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D). There were two discussions on "The Evolutionary and Genetic Bases of Human Behavior (https://www.meetup.com/thinkingsociety/events/116303742)" which covered videos 2-7 of the course on 14 Jul 2013 (https://www.meetup.com/thinkingsociety/events/116303742) and 27 Jul 2013 (https://www.meetup.com/thinkingsociety/events/128381212), two discussions on "The Biology of Learning" which covered videos 8 & 9 of t (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D)he course on 10 Nov 2013 (https://www.meetup.com/thinkingsociety/events/144382142/) and 30 Nov 2013 (https://www.meetup.com/thinkingsociety/events/148844402/).

 CJ Fearnley - 2013-11-03 21:20:36-0500 - Updated: 2013-11-25 14:25:14-0500
Introduction to Neuroscience I, 1h of lectures by Nathan Woodling and Anthony Chung-Ming Ng for lecture 10 in the free on-line course "Human Behavioral Biology"

Neuroscience is another way to understand animal behavior. This brain-centered science has limitations, but it offers powerful insights.

"The chief function of the body is to carry the brain around." -- Thomas Edison

The nervous system consists of the central nervous system (CNS, brian & spinal cord) and the peripheral nervous system (all motor & sensory nerves outside the spinal cord including the nerves controlling heartbeat, digestion, etc.)

Compartmentalization of the brain: Different parts of the brain are specialized for different functions: different parts, different functions, different sizes.

The last 3m is an excellent video in a video: "The Synaptic Cleft" by The Glue-Tang Clan. You might want to watch that part even if you skip the details.

The rest of this gets detailed, but I typed in the notes so I might as well share them!

10. Introduction to Neuroscience I

Parts of the brain

brain stem: interface/regulation between spinal cord and brain
cerebellum: helps control motor movement and learning of motor skills by trial and failure corrections

4 lobes of the cortex (outer layer):
occipital lobe: in the back or posterior part of the skull; receives visual information
parietal lobe: senses touch information (larger parts of the brain control more sensitive body parts such as the touch-sensitive fingertips)
temporal lobe: behind temples, receives auditory information, and memory formation
frontal lobe: at the front or anterior part of the skull: plans actions and controls movements; the part of the frontal lobe controlling each body part is arranged roughly in accord with the arrangement of the body itself (knee above foot)

limbic system: below the cortex and above brain stem; it controls things associated with emotion, learning, and memory; it includes the hippocampus ("seahorse": important for memory and forming new memories; discovered when surgery to allay H.M.'s siezures caused him to lose the ability to form new memories but he could remember things from childhood) & amygdala (almond-shaped in front of the hippocampus; involved in fear & anxiety such as smelling sweat from sky divers)

Brain systems associated with hormones:
hypothalamus (near center of the brain): 4f's: flight, fight, feeding behavior, reproductive behavior
pituitary gland (underneath hypothalamus)

spinal cord specialized: motor nerves send out signals, sensory nerves receive information

Santiago Ramón y Cajal (1852-1934): father of modern neuroscience & nobel laureate (1906): did the anatomical work showing the brain consisted of branching structures of cells

Heinrich Wilhelm Gottfried von Waldeyer-Hartz (1836-1921) popularized the neuron theory that the mush of the nervous system consists of separate cells called neurons

Cell types in the brain:
Most (∼90%) cells in the nervous system are glia (defined as everything but neurons): they are the glue of the nervous system
astrocytes (star-shaped): provide nutrients to neurons and help regulate how they fire
oligodendrocytes and Schwann cells wrap around the axons to help with neuron firing
microglia: the brain's immune cells
neurons: computational units of the brain; binary signaling: either on or off (resting potential); ∼100 billion neurons in the average brain each with about 10,000 connections (synapses) to other neurons, so ∼1 quadrillon synapses in your brain: ∼400 billion stars in the Milky Way, so ∼2000 times more synapses than stars in the Milky Way

pre-synaptic neuron: neuron sending the signal
post-synaptic neuron: neuron receiving the signal
Parts of a neuron:
* soma: cell body
* dendrite: branched projections receiving information from other neurons
* nucleus: like all other cells has the DNA
* axon hillock: sums up signals received and decides whether to fire or not

* axon: "wire" to send information out to the next neuron
* terminal: the end of the axon that passes the signal to the next neuron; it stores neurotransmitter in spherical vesicles
* In the rest state or highly polarized state, the neuron pumps +ions out of the cell, so - charge inside the neuron

synapse: junction between pre-synaptic & post-synaptic neuron: neurotransmitters emitted from the terminal to receptors on the dendrite which open channels to pump ions (+ or -: excitatory or inhibitory)

Functioning of the synapse:
* if enough +ions enter the neuron locally it enters a depolarized state
* If enough +charge makes it to the axon hillock to exceed a threshold (the action potential), then the axon hillock opens more channels to let more +ions in which feeds forward down the axon resulting in a release of neurotransmitter from the synaptic vesicles in the axon terminal
* In addition to firing, the neurotransmitter or the state of charge could induce a genomic effect by activating transcription factors which may increase or decrease the responsiveness of the neuron

neurotransmitters occur in the axon terminal, they release in event of the action potential, and it induces a change of charge in the dendrite of the post-synaptic neuron. There are many types of neurotransmitters some excitatory some inhibitory. Neuroscience currently thinks there are a few hundred neurotransmitters. Used neurotransmitter must clear out to allow further signaling. One process is called reuptake: recycling of used neurotransmitter back into the pre-synaptic neuron. Another is degrading: break them down which can then be detected in the blood, urine or cerebrospinal fluid.

Some common neurotransmitters:
* dopamine: associated with the reward system (pleasure), but it has many, many other functions (due to compartmentalization of the brain)
* epinephrine = adreniline: fight or flight response
* serotonin: regulation of sleep, appetite & mood
* Acetylcholine
* GABA (gamma-Aminobutyric acid): the chief inhibitory neurotransmitter in the mammalian central nervous system
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neuropharmacology: external manipulation of synaptic events
Mescaline, LSD, Psilocybin are halucinogens that interfere with the serotonin receptors: There are many ways to manipulate events at the synapse

Parkinson's disease: inadequate dopamine in the part of the brain controlling motor movement

CJ Fearnley

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Parkinson's disease: inadequate dopamine in the part of the brain controlling motor movement

youtube.com

10. Introduction to Neuroscience I

(April 21, 2010) Nathan Woodling and Anthony Chung-Ming Ng give a broad overview of the field of neuroscience and how it relates to human biology. They discu...

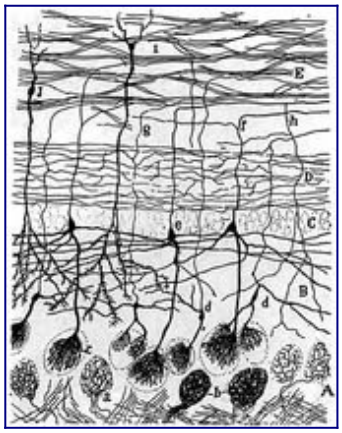
CJ Fearnley

Andrew Moberly: now I know who Santiago Ramón y Cajal was!

- 11y
-
- See translation

Andrew Moberly

His images are pretty amazing. Some of the cell types he described morphologically still have an unknown function in brain circuits. Here's his illustration of the cellular organization of the olfactory bulb.



CJ Fearnley

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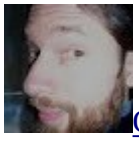
Introduction to Neuroscience II, 1¼h of lectures by Patrick House and Dana Turker for Human Behavioral Biology
Patrick talks about memory and plasticity. Why do some memories last while others are fleeting? How does context (environment) fit into shaping memory? Can neuroscience explain Stephen Wiltshire the autistic who can draw detailed aerial views of whole cities from short helicopter rides.
Dana talks about the autonomic nervous system (ANS) which is part of the peripheral nervous system (that is, the part that deals with systems outside the brain and spinal cord). It manages those functions that happen automatically: the stuff we do not have conscious control over. E.g., heart beating, digestion, goosebumps, orgasm, etc. A highlight of her discussion is an explanation for penile erection and male ejaculation in terms of the two main subsystems of the ANS: the parasympathetic nervous system (PNS) and sympathetic nervous system (SNS). Her slides are fantastic!
Detailed notes on both talks are below.
http://www.youtube.com/watch?v=uqU9lmFztOU
Detailed notes on Patrick's talk on memory and plasticity.
See Stephen Wiltshire's stunning aerial view of London:
http://www.stephenwiltshire.co.uk/show_print.aspx?Id=1565
To understand Patrick I had to look up a word in WordNet. Potentiate is a verb meaning to increase the effect of or act synergistically with. Potentiation is the noun form meaning the synergistic (positive feedback) effect of something.
Memory happens, to the best of our knowledge, in the synapse, the space between two neurons. It involves modulation and changes in the synapse. Memory is synaptic plasticity by strengthening and enhancing signal transmission between two neurons. This is known as LTP (long-term potentiation): "neurons that fire together wire together" is a caricature of Hebbian theory (named after Donald Hebb who proposed the idea in 1949).
Information in the brain is transferred by excitation of neurons and glutamate is the chief excitatory neurotransmitter in the vertebrate nervous system. So "repetition drives memory".
Some mechanisms of synaptic potentiation: 1) add more excitatory neurotransmitter, 2) increase sensitivity of the neurotransmitter receptors in the post-synaptic neurons, 3) increase the number of post-synaptic receptors.
But how do the pre- and post-synaptic neurons know when to potentiate? There are retrograde neurotransmitters (e.g., nitrous oxide N_2O) which feedback from the post-synaptic neuron to the pre-synaptic neuron to modulate release of pre-synaptic neurotransmitters. This is the mechanism of LTP. NB: this counters the normal flow of neural information: fascinating!
Autistic savant memories are not rote, but formed by a spatio-geographical "walk" through of their experiences.
The hippocampus is the site of memory and LTP. First evidence: when HM had his hippocampi removed to stop seizures, he lost the ability to form new memories. Brain scanning during learning shows LTP in the hippocampus. If you block LTP pharmocologically, learning and activity in the hippocampus decrease.
There is adult neurogensis and adult plasticity.
How do motor memories work and how does emotion trigger stronger memories?
LTP happens all over the brain

excitation = plasticity = potentiation
Post-traumatic stress is caused by potentiation of emotionally hyperactive memories that are not relevant to the current situation.
Since memory is a physiological process, like every physico-mechanical process it is subject to failure without prior notice (that's my Dad's expression). Patrick simply observes that things can go wrong: even when we try, sometimes we cannot remember something and other memories just fade away.
Some mechanisms for intentional disruption of LTP. When you are hungry (hypoglycemic states), insulin disrupts LTP. Stress hormones can give us short-term enhanced memory (slow-motion rememory of a car crash). Excess stress hormones can weaken memory formation. Alcohol disrupts LTP.
He concludes by explaining how this myoptic view of neurons and synapses misses the complexity of 100 billion neurons each interacting with 10,000 neurons using a chemico-electrical physical system implies some degree of noise in the individual inter-neuron signal transfers. Random and spontaneous generation of action potentials happen. So the brain needs to try to distinguish between signal and noise. Neurons operate in groups giving another layer of complexity to the system. Inhibition is another mechanism the brain uses to damp out noise. Neurons can inhibit themselves so that their firing contains more signal and less noise. This allows for temporal sharpening of its signal.
Lateral (or spatial) inhibation: a neuron inhibiting its neighbors to enhance its own signal among the noise of those neighbors. For example, in pain sensation, fast sharp pain is carried by one group of neurons while another carries the slow dull pain. The fast, sharp pain can activate the slow dull pain neuron group which then gradually inhibits the fast sharp pain causing it to stop.
Vision gives a more complex example. Nobel laureates David Hubel and Torsten Wiesel found a spatiotopic (orientation and alignment) relationship between neurons in the primary visual cortex (V1) in the occipital lobe and neurons in the retina of the eye. At the next layer up (V2), they discovered that certain visual patterns stimulated neurons in V2 even though stimulation of individual retinal neurons did nothing. Different features are extracted out by the different levels of neuronal analysis in the visual cortex.
Neural networks: when you combine 100 billion neurons each with about 10,000 synaptic connections to other neurons we can imagine how the neural representation of context or environment must be stored somehow in the network itself. So concepts & categories and indeed memories themselves are a kind of emergent property of brains. Individual genetic and cellular and historical differences can affect these neural networks yielding the differences that make each of us unique.
Details from Dana's talk about the autonomic nervous system (ANS) which is part of the peripheral nervous system (which includes the ANS plus the somatic nervous system which delivers sensory inputs to the CNS (central nervous system: brain and spinal cord) and the voluntary nervous system which transmits motor signals between the CNS and muscles, this system is myelinated and therefore fast; it move muscles). The ANS is unmyelinated and therefore slow. The ANS is involuntary and moves organs.
The ANS is subdivided into the parasympathetic nervous system and the sympathetic nervous system. The parasympathetic and sympathetic tend to work in opposition to one another. So usually when one is on, the other is off (but one thing by now in this course you will have noticed is that it tends to be more complicated than that!). The sympathetic deals with arousal, alertness, stress,

emergencies, fight or flight whereas the parasympathetic deals with growth and repair and calm, vegetative functions (e.g., digestion).
For example, the fight or flight response of the sympathetic nervous system gets the body ready for action: it releases adrenaline, raises blood pressure, releases glucose for muscle energy, slows digestion, releases cortisol to suppress the immune system. Then our pupils dilate, mouth gets dry, neck & shoulders tense, heart pumps faster, we sweat, breathe faster, etc.
The sympathetic nervous system releases the excitatory hormone norepinephrine (NE) in the target organs. It releases epinephrine (also called adrenaline) in the adrenal gland. Norepinephrine is oxidized (loss of electron(s)) to epinephrine, so they are separated by one biochemical step. The parasympathetic nervous system releases acetylcholine (ACh) in target organs.
Comparison of parasympathetic and sympathetic effects:
Parasympathetic Sympathetic
- constricts pupils - dilates pupils
- stimulates tear glands - no effect on tear glands
- strongly stimulate salivation - weakly stimulate salivation
- inhibits heart - accelerates heart
- dilates arterioles - constricts arterioles
- constricts bronchi - dilates bronchi
- stimulates stomach, pancreas - inhibits stomach, pancreas
- stimulates intestines - inhibits intestines
- contracts bladder - relaxes bladder
- stimulates erection - stimulates ejaculation
Sympathetic is not always excitatory (it inhibits the GI tract) and the parasympathetic is not always inhibitory (it stimulates the GI tract), it depends on the organ. So we need two different receptors for each system (an excitatory one and an inhibatory one), that is, the sympathetic (parasympathetic) system can have excitatory NE (Ach) receptors in some organs and inhibatory ones in other organs.
Homeostasis is a dynamic balance between the parasympathetic and sympathetic systems.
Stress can impair the parasympathetic system sometimes leading to erectile dysfunction (60% of cases) and immune functions can also weaken.
Regulation of the autonomic nervous system is controlled by the hypothalmus (which also controls the pituitary gland and the endocrine system). The cells of the hypothalmus are just one synapse away from the neurons that project to the target organs. E.g., barorecptors in blood vessels detect low blood pressure and send signals to hypothalmus which then sends a signal via the spinal cord to adjust blood pressure by projection into the heart which then beats faster.
The limbic system (emotions, behavior, memory) surrounds the hypothalmus. E.g., seeing things or people you like can cause a sympathetic (fight or flight) response.
Our cortex is also wired in, so that purely cognitive thoughts can trigger the ANS. A test can trigger the sympathetic system.
Hypothalmus is found even in reptiles. The limbic system is in all mammals. The cortex is in primates.
ANS function is highly plastic (malleable): receptivity can change over time. E.g, sustained stress (lots of NE needed) can stimulate the production of more NE to sustain the stress response.
Habituation or sensitization are both possible changes that the ANS can experience. Biofeedback (think pleasant thoughts) can decrease blood pressure.

youtube.com
11. Introduction to Neuroscience II
(April 23, 2010) Patrick House discusses memories and how they are formed. Dana Turker then lectures about the autonomic nervous system and its functions. St...

 CJ Fearnley - 2013-11-24 20:35:15-0500 - Updated: 2013-12-18 20:42:22-0500
Endocrinology. 50m lectures by William Peterson and Tom McFadden for Human Behavioral Biology

This is a good overview of the endocrine system and the way it works. It starts with communication. Multicellular life requires communication. There are four ways cells communicate:
- cell-cell contact: short range: 1:1
- Paracrine: short range: 1:many
- Neuronal: long-range, fast, specific
- endocrine: long-range, slow, widespread (not specific)

The endocrine system is about chemical messangers (hormones) in the blood. The endocrine system is slow but allows for complex coordination of multiple systems (e.g., metamorphosis of catepillar where every cell transforms in a coordinated way, coordination of response to a given environmental trigger, puberty, pregnancy, sex determination, stress, sexual behavior).

12. Endocrinology

There are two main types of hormones: Peptide & Steroid hormones (there are also other amino acid based hormones).

Peptides are made from amino acids; hydrophilic = water-loving = polar = lipophobic and so water soluble; they travel freely in the blood; receptors are on the cellular membrane which sets off in the cell a secondary messenger cascade (e.g., opening ion channels); quick acting with short effects mainly through protein activity; e.g., insulin, vasopressin, oxytocin, ACTH, CRH, etc.

Steroids are made from cholesterol; hydrophobic = water-hating = nonpolar = lipophillic and so not water soluble; they travel bound to chaperones (protein carriers); as lipids, they can get through the cell membrane and bind to receptors within the cell; slow acting with lasting effects mainly through transcription e.g., glucocorticoids, androgens (testosterone), estrogen, etc.

Note that the evolutionary perspective suggests that receptors being able to distinguish subtle chemical differences is important.

Note that dopamine & epinephrine are neurotransmitters, but can also be released into the blood making them hormones. So some chemicals play different roles at different times and locations in the body.

How the brain controls hormones?

Endocrine glands are specialized structures to release hormones into the bloodstream. The primary brain endocrine glands are the hypothalamus & pituitary gland (also the pineal) which control the peripheral endocrine glands including the thyroid gland, adrenal glands, pancreas, ovary, testis. The

hypothalamus links the nervous system to the endocrine system via the pituitary which is underneath it. Anterior pituitary hormones include ACTH, FSH, LH, TSH, prolactin, endorphins, GH. Posterior pituitary hormones include vasopressin, oxytocin. Hormones are stored in vesicles to be ready for release.

An important example which shows how the brain controls the endocrine system: The hypothalmic-pituitary-adrenal (HPA) axis: hypothalamus releases CRH to anterior pituitary which releases ACTH into the blood which signals the adrenal cortex (on kidneys) to release glucocorticoids (stress hormones) such as cortisol. Some receptors are in the pituitary/hypothalamus so they know how much is in the system (feedback). Negative feedback: more cortisol can trigger a reduction in ACTH release. the HPA axis shows how the brain controls (regulates) hormones throughout the body.

How hormones influence the brain

For hormones to influence the brain they need to cross the blood-brain barrier (which is just special epithelial cells lining the blood vessels in the brain to tightly regulate what can get in). The type, location & density of receptors affects the effects and also the level of hormone + qualities of receptors affect the effects.

down-regulation: reducing the number or sensitivity of receptors in response to high hormone levels
up-regulation: increase in the number or sensitivity of receptors

Hormones can have the following actions on neurons: change membrane potential via ion channels; change transcription of genes (including receptors); change protein activity & transport (could affect neurotransmission, formation of new synapses, etc.)

Note hormones can affect networks of neurons to shape behavior at the system level.

Types of behavior and the hormones that are involved:
- Stress: glucocorticoids
- sexual behavior: testosterone, estrogen, vasopressin, oxytocin
- Aggression: testosterone, glucocorticoids, estrogen, epinephrine
- Depression: glucocorticoids, thryoid hormone, estrogen, progesterone, melatonin

CJ Fearnley

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Endocrinology. 50m lectures by William Peterson and Tom McFadden for Human Behavioral Biology
This is a good overview of the endocrine system and the way it works. It starts with communication. Multicellular life requires communication. There are four ways cells communicate:
- cell-cell contact: short range: 1:1
- Paracrine: short range: 1:many
- Neuronal: long-range, fast, specific
- endocrine: long-range, slow, widespread (not specific)

The endocrine system is about chemical messangers (hormones) in the blood. The endocrine system is slow but allows for complex coordination of multiple systems (e.g., metamorphosis of catepillar where every cells transform in a coordinated way, coordination of response to a given environmental trigger, puberty, pregnancy, sex determination, stress, sexual behavior).
http://www.youtube.com/watch?v=yETVsV4zfFw
There are two main types of hormones: Peptide & Steroid hormones (there are also other amino acid based hormones).
Peptides are made from amino acids; hydrophilic = water-loving = polar = lipophobic and so water soluble; they travel freely in the blood; receptors are on the cellular membrane which sets off in the cell a secondary messenger cascade (e.g., opening ion channels); quick acting with short effects mainly through protein activity; e.g., insulin, vasopressin, oxytocin, ACTH, CRH, etc.
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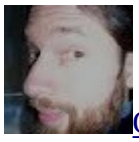
12. Endocrinology
(April 26, 2010) William Peterson and Tom McFadden introduce the field of endocrinology. They explore at the contextual basis of the endocrine system, peptid...

 CJ Fearnley - 2013-12-04 18:27:07-0500 - Updated: 2014-01-17 23:05:40-0500
Advanced Neurology and Endocrinology. 1h 15m Robert Sapolsky video.

Genes code for proteins which in neurology critically means neurotransmitters and their receptors in the dendrites. So sociobiology, evolutionary theory, genetics, ethology (with its "innate releasing mechanisms"), all parts of the course so far, interrelate and feed into the brain's communication systems as neurology & endocrinology.

There are two major themes about neurology & endocrinology Sapolsky emphasizes 1) there are lots of different ways the nervous & endocrine systems can change their functions over time 2) there are lots of realms where individual differences can manifest (genetic & environmental). This lecture shows that the standard first-order understanding of neuroendocrinology hides much of the complexity, information tools used to organize our behavior, and signposts for individuality present in our exquisite biology.

This lecture is a bit involved, but it is still pretty high level with the goal of relaying just enough brain science to understand human behavior. My notes are extensive.

13. Advanced Neurology and Endocrinology

Neuroendocrinology: how hormones affect the neurons and neurons affect hormones
The limbic system: about emotion, affect & behavior (personality, temperment)
exocytosis: dumping neurotransmitters from their vesicles

Dale's law #2 (Henry Hallett Dale): each neuron has 1 characteristic type of neurotransmitter. In 1980's it was found that multiple neurotransmitters are released by some neurons (with a mixture of both/all types of neurotransmitters used by that neuron are stored in its vesicles). Some vesicles contain 3 types of neurotransmitters ("the world record"). This produces a potential for more information! The different neurotransmitters in a neuron's axon terminal tend to have different structures (one simple, one complex; one with a rapid short term effect, one with a slower long term effect which might mean changes to gene transcription). Interestingly it was found that we have receptors for the neurotransmitter on the axon terminal too: the transmitting neuron needs feedback on how much neurotransmitter is in the synapse.

ACTH: Adrenocorticotropic hormone also known as corticotropin
CRH: Corticotropin-releasing hormone also known as corticotropin-releasing factor (CRF)

Similar complexity is found in the endocrine system. In general neuro-endocrine axes work with the hypothalamus releasing a set of hormones to encode a pituitary response which then sends hormones into general circulation to signal receptors all over the body to respond appropriately. For the HPA axis (from the previous lecture: https://plus.google.com/104222466367230914966/posts/7RzgcWMoRBb), the hypothalamus releases NE (norepinephrine), E (epinephrine/adrenaline), OT (oxytocin), CRH, VP (vasopressin), and other hormones to the anterior pituitary (always depicted on the left) whose glands release ACTH to general circulation. The mixture of released hormones from the hypothalamus encode a stress signature enabling us to orchestrate and fine tune different responses to different types of stress. The shape of the secretory curve of ACTH differs with different signatures. In addition, the array of signature hormones can have other effects in the pituitary. Moreover, there is evidence for corticotropin inhibiting factors (best idea is peptide delta sleep-inducing factor (DSIP in Wikipedia): going to sleep may be a reasonable time to turn off the stress response?!

Dale's law #1: an action potential in a neuron will result in the release of neurotransmitter at every axon terminal. This principle is pretty well established. But Jerry Lettvin at MIT showed that you can have blockades that prevent the action potential from reaching some of the branches: neurons can regulate which of their branches get the message: more complexity = more information. There is an unexplored world of possible regulation mechanisms on the dendritic branches as well.

GH=growth hormone
Pro=Prolactin
FSH=Follicle stimulating hormone

Specialized cells in the pituitary produce just one kind of hormone which are distributed throughout the gland. So there are local neighborhoods with distinct groupings of specialized cells. There is all sorts of communication between cells in the pituitary regulating output.

Negative feedback is needed to regulate all biological outputs including the release of hormones once the concentration / effects have reached some measured threshold (otherwise output would continue forever).

Autoreceptors for neurotransmitter occur on the axon terminal to provide measurments needed for negative feedback regulation. Sometimes one of the several neurotransmitters released by a neuron is there to do the bookkeeping by binding to autoreceptors. Similar behavior occurs in the neuroendocrine system to regulate hormones to reach set points (thresholds) with negative feedback to stop the release process by an inhibitory signal. Most endocrine negative feedback works by the brain measuring the amount of end product. Sometimes the brain measures not threshold amounts (which tends to be the focus later in the stress response process) but rates of change (particularly early in the stress response such as when the pituitary regulates ACTH: no one understands this mechanism which was predicted by Mary Dallman of UCSF in a theoretical study).

In biochemistry and pharmacology, a ligand is a substance that binds to a receptor which is a complex of proteins which in turn implies they are encoded by multiple genes implying that genetic variants are likely.

Autoregulation: adjusting receptors based on levels of ligands: so if the ligand signal is strong, we down regulate reception; if the ligand concentration is weak, we up regulate reception. There may even be autoregulatory effects in changing the autoreceptors.

SSRI: Selective serotonin reuptake inhibitor (such as Prozac)

Depression: may be caused by abnormalities in seratonin, dopamine, norepinephrine: SSRIs change blood concentrations, but only later does autoregulation change the number of receptors creating the delayed depression-improving effect.

Insulin resistance in adult onset diabetes: abnormality in insulin down regulation signaling to store away glucose leading to a cascade that eventually wears out the pancreas.

By choosing different protein mixes to build the receptor, changes in receptor regulation can be effected. Some receptor variation can be caused by a protein substitution that triggers high levels of excitation (epilepsy). Changes in the axon hillock's threshold can be caused by changes to the receptors in the dendrites. Similar issues affect hormone receptors.

As explained in the molecular genetics lecture (https://plus.google.com/104222466367230914966/posts/BK4xp8PFxav), steroid hormones have two parts: a hormone binding domain and a DNA binding domain (e.g., glucocorticoids, estrogen, progesterone, etc). Many steroid receptors have cofactors (another set of proteins that modify behavior in different cells). By changing the cofactors, a cell can induce different effects when their steroid ligands bind. Many receptors can bind more than one ligand. E.g., GABA, the main inhibitory neurotransmitter in mammals, has receptors consisting of a whole complex of proteins which include minor binding sites for 1) the major tranquilizers such as barbituates 2) the minor tranquilizers such as the benzodiazepines (such as Valium and Librium), and 3) derivatives of progesterone (seems to play a role in perimenstrual syndrome, PMS, which can effect mood alterations during the reproductive cycle of females). Binding these hormones potentiates GABA which increases its inhibitatory effects. GABA neurons form axoaxonic synapses which connect to the axon terminal of the neurons to which they project (instead of projecting to the dendrites as per standard neuroscience wiring schema). So instead of triggering a neural cascade the inhibitory neurons act to reduce the impact of the signaling of other neurons. So there must be GABA receptors on the axon (most neurotransmitter receptors live on the dendrites not on the axons). So GABA forms a neuromodulatory role by affecting upstream neurons not the neurons themselves.

Similarly some hormones can potentiate the effects of other hormones even though they may have no direct effects themselves.

Conclusion: the brain has lots of mechanisms for modulation of the "normal" first approximation to neural and endocrine signaling systems. These mechanisms give us lots of room for individual variability and lots of ways to respond to experience.

CJ Fearnley

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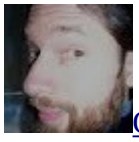 CJ Fearnley - 2013-12-18 21:17:16-0500 - Updated: 2014-01-11 20:00:23-0500
Limbic System. 88m Robert Sapolsky video.

The Limbic system is the emotional decision-making system of the brain. Sapolsky doesn't define "emotional" and it is not fully clear to me what the word means.

Unifying theory of the limbic system: every circuit in the limbic system tries to influence the hypothalamus. Since the hypothalamus is the central hub of the neuroendocrine system, it is the main control system for the body: "Emotions change how your body works". That is, the limbic system tells the hypothalamus what to do. Each part of the limbic system has mechanisms to inform the hypothalamus about what to do and mechanisms to inhibit the signaling of other parts of the brain from sending their signals to the hypothalamus. The brain is a complex drama, a cooperative fight of stimulus & inhibition. Those brain regions with fewer synapses between them and the hypothalamus have a greater "say" in what the hypothalamus should do. So it is surprising that every form of human sensory input has to go through a minimum of 3 or 4 synapses to get to the hypothalamus except olfaction which is 1 synapse from the limbic system! Is smell our most importance sense or is it a spandrel?

Most of the lecture is a survey of the neuroanatomy of the limbic system. But toward the end it gets interesting. First he examines the nature of brain research, how we know what we know, and the limitations of such research. Finally, Sapolsky provides six strong pieces of evidence for the James-Lang theory of emotion which proposes that the physiological changes happen in the body first and the brain senses that and decides what emotion it is feeling. More and more evidence suggests that some of the basis of emotion is indeed physiological (= your body's state). Detailed notes below.

14. Limbic System

The limbic system was known as the rhinencephalon, or the nose brain, due to rat studies which found the olfactory bulb constituted some 40% of the brain of a rodent. The ethological perspective clarifies this connection: rat emotions are associated with smell.

The limbic system is not the nose brain, rather it is the part of the brain that processes the information most pertinent to emotional life.

Paul D. MacLean developed the model of the triune brain with three layers of brain function: reptilian (automatic / regulatory which includes the hypothalamus & pituitary), the paleomammalian or limbic system, and the neocortex or neomammalian (the analytical brain; present in all vertebrates but more developed in some).

Contrary to popular conception, the neocortex is deeply influenced by the limbic system which is why all of us have made terrible emotional decisions under duress as our neocortexes are subject to hormonal influence from the limbic system. Moreover, the neocortex affects the limbic system so that if you think of your own mortality, your limbic system will start pumping out CRH (the

hypothalamic hormone involved in the hypothalamic-pituitary-adrenal (HPA) axis regulating the fight-flight response behavior). The brain is interconnected and the parts of the triune brain are in constant communication with no strict separation of functions.

Ondine's curse (affects about 1/200,000 children): damage to the reptilian brain prevents automatic breathing so you die of sleep deprivation as you keep waking up to breathe.

Antonio Damasio wrote "Descartes' Error": Sapolsky:"it is complete nonsense" that there is a strong separation between thought and emotion.

James Papez sorted out the anatomy of the limbic system in the 1930s. He noted the interconnectedness of the parts (the Papez circuit) that are now identified as the emotional brain.

Cell bodies tend to be grey in color: grey matter are nuclei: cell bodies packed into a region of space
myelination tends to be white in color: white matter is "cables" of axons wrapped in myelin

Anatomy of the limbic system

PFC: Prefrontal cortex: larger in humans than other species: most recently evolved: the last part of the brain to fully mature (isn't fully myelinated until the mid-20s): provides impulse control. Ian Dunbar determined that the size of the PFC is correlated in ∼150 primate species to the size of their average social group. Dunbar's works suggests that the PFC evolved for gossip, social relations, appropriate behavior, and social intelligence. It is involved in learning when to be aggressive and appropriate sexual behaviors.

AC: Anterior cingulate: part of the PFC which is intimately connected to all parts of the limbic system: the cortical component of the limbic system. It is affected in people with clinical depression ("hypersensitivity to pains of life and the world"). It is involved in empathy and feeling the pain of others.

Hipp: hippocampus (Latin for seahorse, but Sapolsky claims "it actually looks like a jelly-roll"). Sapolsky spent 30 years obsessing over it, but now thinks the PFC is the most interesting part of the brain. The hippocampus is involved in learning & memory and turning off the stress response (it measures the levels of glucocorticoids). Which makes sense because survival may depend on remembering how you escaped last time.
hippocampal-amygdala pathway: bidirectional projections between Hipp and Amyg: learning to be afraid
fimbria/fornix: bidirectional projections between Hipp and SEPT (fornix means arch and the pathway is arch-shaped)

Amyg: Amygdala (means almond): center of fear & anxiety & aggression. It also plays a role in male sexual motivation which may explain various pathologies confusing sexual & aggressive behaviors. It receives olfactory data directly: only 1 synapse. Sapolsky: "You cannot understand the neurobiology of being violent without understanding the neurobiology of being afraid and being anxious".
stria terminalis: from Amyg to Hipp by arching the long way around the fimbria/fornix. Such "nutty wiring" tells us that during embryonic development the structures were not close to each other resulting in the long pathway or it might tell us something about the evolutionary history ("evolution is not an inventor, it is a tinkerer").

Thal: Thalamus (above hypothalamus): a bidirectional circuit with the PFC

M: mammillary bodies are tucked behind the hippocampus; it is involved in maternal behavior
mammillothalamic fasciculus: bidirectional connections between M & Thal

SEPT: septum (general term for a midline structure) at the midline of the brain; 2 synapses from Amyg; it inhibits aggression.
Medial forebrain bundle (MFB): major projection from SEPT to Hipp to M and back to SEPT: major limbic pathway

NAcc: Nucleus accumbens: involved in appetitive behavior (anything involving an appetite), releasing dopamine (involved in anticipating pleasure: not about the reward or pleasure but anticipating that a reward is available and empowering the behavior to get the reward, to pursue pleasure). It is affected by depression and drug addictions.

VTA: Ventral tegmental area is important in depression. It sends a lot of information to the NAcc which then connects bidirectionally to each of the other parts of the limbic system

hypothalamus: above pituitary, center of neuroendocrine function
centers in the hypothalamus:
ventral medial hypothalamus & medial preoptic area are involved in sexual behavior. Size differences based on sex (including transgender individuals) and sexual orientation.
SCN: suprachiasmatic nucleus: circadian rhythms
PVN: paraventricular nucleus: makes CRH (corticotropin-releasing factor: the backbone of the stress response)
arcuate nucleus: bottom of hypothalmic funnel: where the hormones come out into the bloodstream
lateral hypothalamus: related to hunger (not aggression as early studies wrongly concluded): measures blood glucose & insulin levels: also involved in broader types of hunger (hunger for information, etc.)

Walle Nauta (1916-1994) was the greatest neuroanatomist of the last half of the 20th century. He suggested that the prefrontal cortex (aka the frontal cortex) should be considered part of the limbic system based on its wiring projections.

There are several methods for studying brain functioning. Missile wounds and other lesions or brain damage allow us to learn by observing the functions that were destroyed by damage to that part of the brain. For example, Subject N.A. had his septum "taken out" by a "miniature fencing foil" at a wedding. Another approach is to stimulate a brain region with electrodes that artificially stimulate action potentials (rarely done on humans except for some rare therapeutic techniques). Instead of a stimulating electrode, you can put in a recording electrode to report on when these neurons are getting excited. Some people record or stimulate just one neuron at a time. "Patch clamping" can record the outputs of a single ion channel on an axon. Measuring the biochemistry such as neurotransmitter concentrations and molecular biology (which genes are being expressed). Finally brain imaging techniques let us study even live human brains in action (CAT or CT scans, MRI, etc.) which can show which areas are active and the metabolic rates and can see the whole brain at once.

Brain imaging studies have shown that some regions of the brain change in size over time. The Amygdala gets bigger over time in people with post-traumatic stress disorder (PTSD). Periods of severe stress cause neurons in the Amygdala to grow more dendritic processes which probably accounts for the size increase. People with long-term depression show shrinking in their hippocampus.

There are some difficulties in interpreting the data from the methods of studying brain function. Centers vs. pathways (neural bodies vs. axons): in lesion studies you may have severed communications and not the center for the given functionality. The notion of a center of neural functionality is a "pretty flimsy concept". The ethological context is important to understanding the wiring in terms of the animal's own language. You have to understand the species and their fixed action patterns as well as the individual and their social situation.

Different areas of the limbic system tend to work in opposition to each other (e.g., Amgy & SEPT stimulating and inhibiting aggression).

We tend to discuss the ways the brain influences outcomes throughout the body. In addition, body functions can affect the brain. The autonomic nervous system seems in particular to report physiology to influence the brain.

Evidence for the James-Lang theory of emotion:
- Stanley Schachter classic experiment (but unethical in modern pharmacology) showing that epinephrine enhances or modulates the emotional state of the social situation (anger & upset or joy & happiness, etc.). Note that epinephrine/adrenaline is associated with arousal of the sympathetic nervous system.
- Benzodiazepines (like Valium) are prescribed to decrease anxiety and muscle relaxation (the exact same dose): anxiety is about monitoring the level of tension in your body ... getting feedback from muscle tone; when your body signals relaxed muscles the brain realizes it doesn't need to feel so anxious: meditation & biofeedback: learning what thoughts allow the brain to regulate blood pressure, for example.
- After a fight, frequently a couple can apologize and everything is all right. But while still in the excited state, memories of prior issues can be brought out leading to another fight: the sympathetic nervous system hasn't realized that the cognitive situation is solved, so it still has hormones pushing you to the agitated state and the brain wonders why it is so upset and goes to find a good reason "from the second Roosevelt administration".
* In females, it takes longer for the sympathetic nervous system to return hormone levels back to baseline
* Similarly, after orgasm males go back to baseline faster than females
- Treat people with clinical depression to mechanically force themselves to smile, after a half hour they feel better
- Test scores received while sitting upright (vs. slouched over), result in happier assessments of the results and more pride. The posture of your spine influences your emotional response!

CJ Fearnley

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Endocrinology. 50m lectures by William Peterson and Tom McFadden for Human Behavioral Biology
This is a good overview of the endocrine system and the way it works. It starts with communication. Multicellular life requires communication. There are four ways cells communicate:
- cell-cell contact: short range: 1:1
- Paracrine: short range: 1:many
- Neuronal: long-range, fast, specific

- endocrine: long-range, slow, widespread (not specific)

The endocrine system is about chemical messangers (hormones) in the blood. The endocrine system is slow but allows for complex coordination of multiple systems (e.g., metamorphosis of catepillar where every cells transform in a coordinated way, coordination of response to a given environmental trigger, puberty, pregnancy, sex determination, stress, sexual behavior).

http://www.youtube.com/watch?v=yETVsV4zfFw

There are two main types of hormones: Peptide & Steroid hormones (there are also other amino acid based hormones).

Peptides are made from amino acids; hydrophilic = water-loving = polar = lipophobic and so water soluble; they travel freely in the blood; receptors are on the cellular membrane which sets off in the cell a secondary messenger cascade (e.g., opening ion channels); quick acting with short effects mainly through protein activity; e.g., insulin, vasopressin, oxytocin, ACTH, CRH, etc.

Steroids are made from cholesterol; hydrophobic = water-hating = nonpolar = lipophillic and so not water soluble; they travel bound to chaperones (protein carriers); as lipids, they can get through the cell membrane and bind to receptors within the cell; slow acting with lasting effects mainly through transcription e.g., glucocorticoids, androgens (testosterone), estrogen, etc.

Note that the evolutionary perspective suggests that receptors being able to distinguish subtle chemical differences is important.

Note that dopamine & epinephrine are neurotransmitters, but can also be released into the blood making them hormones. So some chemicals play different roles at different times and locations in the body.

How the brain controls hormones?

Endocrine glands are specialized structures to release hormones into the bloodstream. The primary brain endocrine glands are the hypothalmus & pituitary gland (also the pineal) which control the peripheral endocrine glands including the thyroid gland, adrenal glands, pancreas, ovary, testis. The hypothalmus links the nervous system to the endocrine system via the pituitary which is underneath it. Anterior pituitary hormones include ACTH, FSH, LH, TSH, prolactin, endorphins, GH. Posterior pituitary hormones include vasopressin, oxytocin. Hormones are stored in vessicles to be ready for release.

An important example which shows how the brain controls the endocrine system: The hypothalmic-pituitary-adrenal (HPA) axis: hypothalmus releases CRH to anterior pituitary which releases ACTH into the blood which signals the adrenal cortex (on kidneys) to release glucocorticoids (stress hormones) such as cortisol. Some receptors are in the pituitary/hypothalmus so they know how much is in the system (feedback). Negative feedback: more cortisol can trigger a reduction in ACTH release. the HPA axis shows how the brain controls (regulates) hormones throughout the body.

How hormones influence the brain

For hormones to influence the brain they need to cross the blood-brain barrier (which is just special epithelial cells lining the blood vessels in the brain to tightly regulate what can get in). The type, location & density of receptors affects the effects and also the level of hormone + qualities of receptors affect the effects.

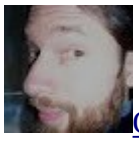
down-regulation: reducing the number of receptors in response to high hormone levels

up-regulation: increase in the number of receptors

Hormone can have the following actions on neurons: change membrane potential via ion channels; change transcription of genes (including receptors); change protein activity & transport (could affect neurotransmission, formation of new synapses, etc.)

Note hormones can affect networks of neurons to shape behavior at the system level.
Types of behavior and the hormones that are involved:
- Stress: glucocorticoids
- sexual behavior: testosterone, estrogen, vasopressin, oxytocin
- Aggression: testosterone, glucocorticoids, estrogen, epinephrine
- Depression: glucocorticoids, thryoid hormone, estrogen, progesterone, melatonin

youtube.com
12. Endocrinology
(April 26, 2010) William Peterson and Tom McFadden introduce the field of endocrinology. They explore at the contextual basis of the endocrine system, peptid...

 CJ Fearnley - 2013-12-04 18:27:07-0500 - Updated: 2014-01-17 23:05:40-0500
Advanced Neurology and Endocrinology. 1h 15m Robert Sapolsky video.

Genes code for proteins which in neurology critically means neurotransmitters and their receptors in the dendrites. So sociobiology, evolutionary theory, genetics, ethology (with its "innate releasing mechanisms"), all parts of the course so far, interrelate and feed into the brain's communication systems as neurology & endocrinology.

There are two major themes about neurology & endocrinology Sapolsky emphasizes 1) there are lots of different ways the nervous & endocrine systems can change their functions over time 2) there are lots of realms where individual differences can manifest (genetic & environmental). This lecture shows that the standard first-order understanding of neuroendocrinology hides much of the complexity, information tools used to organize our behavior, and signposts for individuality present in our exquisite biology.

This lecture is a bit involved, but it is still pretty high level with the goal of relaying just enough brain science to understand human behavior. My notes are extensive.

13. Advanced Neurology and Endocrinology

Neuroendocrinology: how hormones affect the neurons and neurons affect hormones
The limbic system: about emotion, affect & behavior (personality, temperment)
exocytosis: dumping neurotransmitters from their vesicles

Dale's law #2 (Henry Hallett Dale): each neuron has 1 characteristic type of neurotransmitter. In 1980's it was found that multiple neurotransmitters are released by some neurons (with a mixture of both/all types of neurotransmitters used by that neuron are stored in its vesicles). Some vesicles contain 3 types of neurotransmitters ("the world record"). This produces a potential for more information! The different neurotransmitters in a neuron's axon terminal tend to have different structures (one simple, one complex; one with a rapid short term effect, one with a slower long term effect which might mean changes to gene transcription). Interestingly it was found that we have receptors for the neurotransmitter on the axon terminal too: the transmitting neuron needs feedback on how much neurotransmitter is in the synapse.

ACTH: Adrenocorticotropic hormone also known as corticotropin

CRH: Corticotropin-releasing hormone also known as corticotropin-releasing factor (CRF)

Similar complexity is found in the endocrine system. In general neuro-endocrine axes work with the hypothalamus releasing a set of hormones to encode a pituitary response which then sends hormones into general circulation to signal receptors all over the body to respond appropriately. For the HPA axis (from the previous lecture: https://plus.google.com/104222466367230914966/posts/7RzgcWMoRBb), the hypothalamus releases NE (norepinephrine), E (epinephrine/adrenaline), OT (oxytocin), CRH, VP (vasopressin), and other hormones to the anterior pituitary (always depicted on the left) whose glands release ACTH to general circulation. The mixture of released hormones from the hypothalamus encode a stress signature enabling us to orchestrate and fine tune different responses to different types of stress. The shape of the secretory curve of ACTH differs with different signatures. In addition, the array of signature hormones can have other effects in the pituitary. Moreover, there is evidence for corticotropin inhibiting factors (best idea is peptide delta sleep-inducing factor (DSIP in Wikipedia): going to sleep may be a reasonable time to turn off the stress response?!

Dale's law #1: an action potential in a neuron will result in the release of neurotransmitter at every axon terminal. This principle is pretty well established. But Jerry Lettvin at MIT showed that you can have blockades that prevent the action potential from reaching some of the branches: neurons can regulate which of their branches get the message: more complexity = more information. There is an unexplored world of possible regulation mechanisms on the dendritic branches as well.

GH=growth hormone
Pro=Prolactin
FSH=Follicle stimulating hormone

Specialized cells in the pituitary produce just one kind of hormone which are distributed throughout the gland. So there are local neighborhoods with distinct groupings of specialized cells. There is all sorts of communication between cells in the pituitary regulating output.

Negative feedback is needed to regulate all biological outputs including the release of hormones once the concentration / effects have reached some measured threshold (otherwise output would continue forever).

Autoreceptors for neurotransmitter occur on the axon terminal to provide measurments needed for negative feedback regulation. Sometimes one of the several neurotransmitters released by a neuron is there to do the bookkeeping by binding to autoreceptors. Similar behavior occurs in the neuroendocrine system to regulate hormones to reach set points (thresholds) with negative feedback to stop the release process by an inhibitory signal. Most endocrine negative feedback works by the brain measuring the amount of end product. Sometimes the brain measures not threshold amounts (which tends to be the focus later in the stress response process) but rates of change (particularly early in the stress response such as when the pituitary regulates ACTH: no one understands this mechanism which was predicted by Mary Dallman of UCSF in a theoretical study).

In biochemistry and pharmacology, a ligand is a substance that binds to a receptor which is a complex of proteins which in turn implies they are encoded by multiple genes implying that genetic variants are likely.

Autoregulation: adjusting receptors based on levels of ligands: so if the ligand signal is strong, we down regulate reception; if the ligand concentration is weak, we up regulate reception. There may even be autoregulatory effects in changing the autoreceptors.

SSRI: Selective serotonin reuptake inhibitor (such as Prozac)

Depression: may be caused by abnormalities in seratonin, dopamine, norepinephrine: SSRIs change blood concentrations, but only later does autoregulation change the number of receptors creating the delayed depression-improving effect.

Insulin resistance in adult onset diabetes: abnormality in insulin down regulation signaling to store away glucose leading to a cascade that eventually wears out the pancreas.

By choosing different protein mixes to build the receptor, changes in receptor regulation can be effected. Some receptor variation can be caused by a protein substitution that triggers high levels of excitation (epilepsy). Changes in the axon hillock's threshold can be caused by changes to the receptors in the dendrites. Similar issues affect hormone receptors.

As explained in the molecular genetics lecture (https://plus.google.com/104222466367230914966/posts/BK4xp8PFxav), steroid hormones have two parts: a hormone binding domain and a DNA binding domain (e.g., glucocorticoids, estrogen, progesterone, etc). Many steroid receptors have cofactors (another set of proteins that modify behavior in different cells). By changing the cofactors, a cell can induce different effects when their steroid ligands bind. Many receptors can bind more than one ligand. E.g., GABA, the main inhibitory neurotransmitter in mammals, has receptors consisting of a whole complex of proteins which include minor binding sites for 1) the major tranquilizers such as barbituates 2) the minor tranquilizers such as the benzodiazepines (such as Valium and Librium), and 3) derivatives of progesterone (seems to play a role in perimenstrual syndrome, PMS, which can effect mood alterations during the reproductive cycle of females). Binding these hormones potentiates GABA which increases its inhibitatory effects. GABA neurons form axoaxonic synapses which connect to the axon terminal of the neurons to which they project (instead of projecting to the dendrites as per standard neuroscience wiring schema). So instead of triggering a neural cascade the inhibitory neurons act to reduce the impact of the signaling of other neurons. So there must be GABA receptors on the axon (most neurotransmitter receptors live on the dendrites not on the axons). So GABA forms a neuromodulatory role by affecting upstream neurons not the neurons themselves.

Similarly some hormones can potentiate the effects of other hormones even though they may have no direct effects themselves.

Conclusion: the brain has lots of mechanisms for modulation of the "normal" first approximation to neural and endocrine signaling systems. These mechanisms give us lots of room for individual variability and lots of ways to respond to experience.

CJ Fearnley's post

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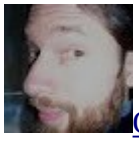 CJ Fearnley - 2013-12-18 21:17:16-0500 - Updated: 2014-01-11 20:00:23-0500
Limbic System. 88m Robert Sapolsky video.

The Limbic system is the emotional decision-making system of the brain. Sapolsky doesn't define "emotional" and it is not fully clear to me what the word means.

Unifying theory of the limbic system: every circuit in the limbic system tries to influence the hypothalamus. Since the hypothalamus is the central hub of the neuroendocrine system, it is the main control system for the body: "Emotions change how your body works". That is, the limbic system tells the hypothalamus what to do. Each part of the limbic system has mechanisms to inform the hypothalamus about what to do and mechanisms to inhibit the signaling of other parts of the brain from sending their signals to the hypothalamus. The brain is a complex drama, a cooperative fight of stimulus & inhibition. Those brain regions with fewer synapses between them and the hypothalamus have a greater "say" in what the hypothalamus should do. So it is surprising that every form of human sensory input has to go through a minimum of 3 or 4 synapses to get to the hypothalamus except olfaction which is 1 synapse from the limbic system! Is smell our most importance sense or is it a spandrel?

Most of the lecture is a survey of the neuroanatomy of the limbic system. But toward the end it gets interesting. First he examines the nature of brain research, how we know what we know, and the limitations of such research. Finally, Sapolsky provides six strong pieces of evidence for the James-Lang theory of emotion which proposes that the physiological changes happen in the body first and the brain senses that and decides what emotion it is feeling. More and more evidence suggests that some of the basis of emotion is indeed physiological (= your body's state). Detailed notes below.

14. Limbic System

The limbic system was known as the rhinencephalon, or the nose brain, due to rat studies which found the olfactory bulb constituted some 40% of the brain of a rodent. The ethological perspective clarifies this connection: rat emotions are associated with smell.

The limbic system is not the nose brain, rather it is the part of the brain that processes the information most pertinent to emotional life.

Paul D. MacLean developed the model of the triune brain with three layers of brain function: reptilian (automatic / regulatory which includes the hypothalamus & pituitary), the paleomammalian or limbic system, and the neocortex or neomammalian (the analytical brain; present in all vertebrates but more developed in some).

Contrary to popular conception, the neocortex is deeply influenced by the limbic system which is why all of us have made terrible emotional decisions under duress as our neocortexes are subject to hormonal influence from the limbic system. Moreover, the neocortex affects the limbic system so

that if you think of your own mortality, your limbic system will start pumping out CRH (the hypothalamic hormone involved in the hypothalamic-pituitary-adrenal (HPA) axis regulating the fight-flight response behavior). The brain is interconnected and the parts of the triune brain are in constant communication with no strict separation of functions.

Ondine's curse (affects about 1/200,000 children): damage to the reptilian brain prevents automatic breathing so you die of sleep deprivation as you keep waking up to breathe.

Antonio Damasio wrote "Descartes' Error": Sapolsky:"it is complete nonsense" that there is a strong separation between thought and emotion.

James Papez sorted out the anatomy of the limbic system in the 1930s. He noted the interconnectedness of the parts (the Papez circuit) that are now identified as the emotional brain.

Cell bodies tend to be grey in color: grey matter are nuclei: cell bodies
packed into a region of space
myelination tends to be white in color: white matter is "cables" of axons
wrapped in myelin

Anatomy of the limbic system

PFC: Prefrontal cortex: larger in humans than other species: most recently evolved: the last part of the brain to fully mature (isn't fully myelinated until the mid-20s): provides impulse control. Ian Dunbar determined that the size of the PFC is correlated in ∼150 primate species to the size of their average social group. Dunbar's works suggests that the PFC evolved for gossip, social relations, appropriate behavior, and social intelligence. It is involved in learning when to be aggressive and appropriate sexual behaviors.

AC: Anterior cingulate: part of the PFC which is intimately connected to all parts of the limbic system: the cortical component of the limbic system. It is affected in people with clinical depression ("hypersensitivity to pains of life and the world"). It is involved in empathy and feeling the pain of others.

Hipp: hippocampus (Latin for seahorse, but Sapolsky claims "it actually looks like a jelly-roll"). Sapolsky spent 30 years obsessing over it, but now thinks the PFC is the most interesting part of the brain. The hippocampus is involved in learning & memory and turning off the stress response (it measures the levels of glucocorticoids). Which makes sense because survival may depend on remembering how you escaped last time.
hippocampal-amygdala pathway: bidirectional projections between Hipp and Amyg: learning to be afraid
fimbria/fornix: bidirectional projections between Hipp and SEPT (fornix means arch and the pathway is arch-shaped)

Amyg: Amygdala (means almond): center of fear & anxiety & aggression. It also plays a role in male sexual motivation which may explain various pathologies confusing sexual & aggressive behaviors. It receives olfactory data directly: only 1 synapse. Sapolsky: "You cannot understand the neurobiology of being violent without understanding the neurobiology of being afraid and being anxious".
stria terminalis: from Amyg to Hipp by arching the long way around the fimbria/fornix. Such "nutty wiring" tells us that during embryonic development the structures were not close to each other resulting in the long pathway or it might tell us something about the evolutionary history ("evolution is not an inventor, it is a tinkerer").

Thal: Thalamus (above hypothalamus): a bidirectional circuit with the PFC

M: mammillary bodies are tucked behind the hippocampus; it is involved in maternal behavior
mammillothalamic fasciculus: bidirectional connections between M & Thal

SEPT: septum (general term for a midline structure) at the midline of the brain; 2 synapses from Amyg; it inhibits aggression.
Medial forebrain bundle (MFB): major projection from SEPT to Hipp to M and back to SEPT: major limbic pathway

NAcc: Nucleus accumbens: involved in appetitive behavior (anything involving an appetite), releasing dopamine (involved in anticipating pleasure: not about the reward or pleasure but anticipating that a reward is available and empowering the behavior to get the reward, to pursue pleasure). It is affected by depression and drug addictions.

VTA: Ventral tegmental area is important in depression. It sends a lot of information to the NAcc which then connects bidirectionally to each of the other parts of the limbic system

hypothalamus: above pituitary, center of neuroendocrine function
centers in the hypothalamus:
ventral medial hypothalamus & medial preoptic area are involved in sexual behavior. Size differences based on sex (including transgender individuals) and sexual orientation.
SCN: suprachiasmatic nucleus: circadian rhythms
PVN: paraventricular nucleus: makes CRH (corticotropin-releasing factor: the backbone of the stress response)
arcuate nucleus: bottom of hypothalmic funnel: where the hormones come out into the bloodstream
lateral hypothalamus: related to hunger (not aggression as early studies wrongly concluded): measures blood glucose & insulin levels: also involved in broader types of hunger (hunger for information, etc.)

Walle Nauta (1916-1994) was the greatest neuroanatomist of the last half of the 20th century. He suggested that the prefrontal cortex (aka the frontal cortex) should be considered part of the limbic system based on its wiring projections.

There are several methods for studying brain functioning. Missile wounds and other lesions or brain damage allow us to learn by observing the functions that were destroyed by damage to that part of the brain. For example, Subject N.A. had his septum "taken out" by a "miniature fencing foil" at a wedding. Another approach is to stimulate a brain region with electrodes that artificially stimulate action potentials (rarely done on humans except for some rare therapeutic techniques). Instead of a stimulating electrode, you can put in a recording electrode to report on when these neurons are getting excited. Some people record or stimulate just one neuron at a time. "Patch clamping" can record the outputs of a single ion channel on an axon. Measuring the biochemistry such as neurotransmitter concentrations and molecular biology (which genes are being expressed). Finally brain imaging techniques let us study even live human brains in action (CAT or CT scans, MRI, etc.) which can show which areas are active and the metabolic rates and can see the whole brain at once.

Brain imaging studies have shown that some regions of the brain change in size over time. The Amygdala gets bigger over time in people with post-traumatic stress disorder (PTSD). Periods of severe stress cause neurons in the Amygdala to grow more dendritic processes which probably accounts for the size increase. People with long-term depression show shrinking in their

hippocampus.

There are some difficulties in interpreting the data from the methods of studying brain function. Centers vs. pathways (neural bodies vs. axons): in lesion studies you may have severed communications and not the center for the given functionality. The notion of a center of neural functionality is a "pretty flimsy concept". The ethological context is important to understanding the wiring in terms of the animal's own language. You have to understand the species and their fixed action patterns as well as the individual and their social situation.

Different areas of the limbic system tend to work in opposition to each other (e.g., Amgy & SEPT stimulating and inhibiting aggression).

We tend to discuss the ways the brain influences outcomes throughout the body. In addition, body functions can affect the brain. The autonomic nervous system seems in particular to report physiology to influence the brain.

Evidence for the James-Lang theory of emotion:
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Limbic System. 88m Robert Sapolsky video.
The Limbic system is the emotional decision-making system of the brain. Sapolsky doesn't define "emotional" and it is not fully clear to me what the word means.
Unifying theory of the limbic system: every circuit in the limbic system tries to influence the hypothalamus. Since the hypothalamus is the central hub of the neuroendocrine system, it is the main control system for the body: "Emotions change how your body works". That is, the limbic system tells the hypothalamus what to do. Each part of the limbic system has mechanisms to inform the hypothalamus about what to do and mechanisms to inhibit the signaling of other parts of the brain from sending their signals to the hypothalamus. The brain is a complex drama, a cooperative fight of stimulus & inhibition. Those brain regions with fewer synapses between them and the hypothalamus have a greater "say" in what the hypothalamus should do. So it is surprising that every form of human sensory input has to go through a minimum of 3 or 4 synapses to get to the hypothalamus except olfaction which is 1 synapse from the limbic system! Is smell our most importance sense or is it a spandrel?
Most of the lecture is a survey of the neuroanatomy of the limbic system. But toward the end it gets interesting. First he examines the nature of brain research, how we know what we know, and the limitations of such research. Finally, Sapolsky provides six strong pieces of evidence for the James-Lang theory of emotion which proposes that the physiological changes happen in the body first and the brain senses that and decides what emotion it is feeling. More and more evidence suggests that some of the basis of emotion is indeed physiological 😊 your body's state). Detailed notes below.
http://www.youtube.com/watch?v=CAOnSbDSaOw
The limbic system was known as the rhinencephalon, or the nose brain, due to rat studies which found the olfactory bulb constituted some 40% of the brain of a rodent. The ethological perspective clarifies this connection: rat emotions are associated with smell.
The limbic system is not the nose brain, rather it is the part of the brain that processes the information most pertinent to emotional life.
Paul D. MacLean developed the model of the triune brain with three layers of brain function: reptilian (automatic / regulatory which includes the hypothalamus & pituitary), the paleomammalian or limbic system, and the neocortex or neomammalian (the analytical brain; present in all vertebrates but more developed in some).
Contrary to popular conception, the neocortex is deeply influenced by the limbic system which is why all of us have made terrible emotional decisions under duress as our neocortexes are subject to hormonal influence from the limbic system. Moreover, the neocortex affects the limbic system so that if you think of your own mortality, your limbic system will start pumping out CRH (the hypothalamic hormone involved in the hypothalamic-pituitary-adrenal (HPA) axis regulating the fight-flight response behavior). The brain is interconnected and the parts of the triune brain are in constant communication with no strict separation of functions.
Ondine's curse (affects about 1/200,000 children): damage to the reptilian brain prevents automatic breathing so you die of sleep deprivation as you keep waking up to breathe.
Antonio Damasio wrote "Descartes' Error": Sapolsky:"it is complete nonsense" that there is a strong separation between thought and emotion.

James Papez sorted out the anatomy of the limbic system in the 1930s. He noted the interconnectedness of the parts (the Papez circuit) that are now identified as the emotional brain.
Cell bodies tend to be grey in color: grey matter are nuclei: cell bodies
packed into a region of space
myelination tends to be white in color: white matter is "cables" of axons
wrapped in myelin
Anatomy of the limbic system
PFC: Prefrontal cortex: larger in humans than other species: most recently evolved: the last part of the brain to fully mature (isn't fully myelinated until the mid-20s): provides impulse control. Ian Dunbar determined that the size of the PFC is correlated in ∼150 primate species to the size of their average social group. Dunbar's works suggests that the PFC evolved for gossip, social relations, appropriate behavior, and social intelligence. It is involved in learning when to be aggressive and appropriate sexual behaviors.
AC: Anterior cingulate: part of the PFC which is intimately connected to all parts of the limbic system: the cortical component of the limbic system. It is affected in people with clinical depression ("hypersensitivity to pains of life and the world"). It is involved in empathy and feeling the pain of others.
Hipp: hippocampus (Latin for seahorse, but Sapolsky claims "it actually looks like a jelly-roll"). Sapolsky spent 30 years obsessing over it, but now thinks the PFC is the most interesting part of the brain. The hippocampus is involved in learning & memory and turning off the stress response (it measures the levels of glucocorticoids). Which makes sense because survival may depend on remembering how you escaped last time.
hippocampal-amygdala pathway: bidirectional projections between Hipp and Amyg: learning to be afraid
fimbria/fornix: bidirectional projections between Hipp and SEPT (fornix means arch and the pathway is arch-shaped)
Amyg: Amygdala (means almond): center of fear & anxiety & aggression. It also plays a role in male sexual motivation which may explain various pathologies confusing sexual & aggressive behaviors. It receives olfactory data directly: only 1 synapse. Sapolsky: "You cannot understand the neurobiology of being violent without understanding the neurobiology of being afraid and being anxious".
stria terminalis: from Amyg to Hipp by arching the long way around the fimbria/fornix. Such "nutty wiring" tells us that during embryonic development the structures were not close to each other resulting in the long pathway or it might tell us something about the evolutionary history ("evolution is not an inventor, it is a tinkerer").
Thal: Thalmus (above hypothalamus): a bidirectional circuit with the PFC
M: mammillary bodies are tucked behind the hippocampus; it is involved in maternal behavior
mammillothalamic fasciculus: bidirectional connections between M & Thal
SEPT: septum (general term for a midline structure) at the midline of the brain; 2 synapses from Amyg; it inhibits aggression.
Medial forebrain bundle (MFB): major projection from SEPT to Hipp to M and back to SEPT: major limbic pathway
NAcc: Nucleus accumbens: involved in appetitive behavior (anything involving an appetite), releasing dopamine (involved in anticipating pleasure: not about the reward or pleasure but anticipating that a reward is available and empowering the behavior to get the reward, to pursue pleasure). It is affected by depression and drug addictions.

VTA: Ventral tegmental area is important in depression. It sends a lot of information to the NAcc which then connects bidirectionally to each of the other parts of the limbic system
hypothalamus: above pituitary, center of neuroendocrine function
centers in the hypothalamus:
ventral medial hypothalamus & medial preoptic area are involved in sexual behavior. Size differences based on sex (including transgender individuals) and sexual orientation.
SCN: suprachiasmatic nucleus: circadian rhythms
PVN: paraventricular nucleus: makes CRH (corticotropin-releasing factor: the backbone of the stress response)
arcuate nucleus: bottom of hypothalmic funnel: where the hormones come out into the bloodstream
lateral hypothalamus: related to hunger (not aggression as early studies wrongly concluded): measures blood glucose & insulin levels: also involved in broader types of hunger (hunger for information, etc.)
Walle Nauta (1916-1994) was the greatest neuroanatomist of the last half of the 20th century. He suggested that the prefrontal cortex (aka the frontal cortex) should be considered part of the limbic system based on its wiring projections.
There are several methods for studying brain functioning. Missile wounds and other lesions or brain damage allow us to learn by observing the functions that were destroyed by damage to that part of the brain. For example, Subject N.A. had his septum "taken out" by a "miniature fencing foil" at a wedding. Another approach is to stimulate a brain region with electrodes that artificially stimulate action potentials (rarely done on humans except for some rare therapeutic techniques). Instead of a stimulating electrode, you can put in a recording electrode to report on when these neurons are getting excited. Some people record or stimulate just one neuron at a time. "Patch clamping" can record the outputs of a single ion channel on an axon. Measuring the biochemistry such as neurotransmitter concentrations and molecular biology (which genes are being expressed). Finally brain imaging techniques let us study even live human brains in action (CAT or CT scans, MRI, etc.) which can show which areas are active and the metabolic rates and can see the whole brain at once.
Brain imaging studies have shown that some regions of the brain change in size over time. The Amygdala gets bigger over time in people with post-traumatic stress disorder (PTSD). Periods of severe stress cause neurons in the Amygdala to grow more dendritic processes which probably accounts for the size increase. People with long-term depression show shrinking in their hippocampus.
There are some difficulties in interpreting the data from the methods of studying brain function. Centers vs. pathways (neural bodies vs. axons): in lesion studies you may have severed communications and not the center for the given functionality. The notion of a center of neural functionality is a "pretty flimsy concept". The ethological context is important to understanding the wiring in terms of the animal's own language. You have to understand the species and their fixed action patterns as well as the individual and their social situation.
Different areas of the limbic system tend to work in opposition to each other (e.g., Amgy & SEPT stimulating and inhibiting aggression).
We tend to discuss the ways the brain influences outcomes throughout the body. In addition, body functions can affect the brain. The autonomic nervous system seems in particular to report physiology to influence the brain.
Evidence for the James-Lang theory of emotion:

- Stanley Schachter classic experiment (but unethical in modern pharmacology) showing that epinephrine enhances or modulates the emotional state that the social circumstances have generated. Note that epinephrine/adrenaline is associated with arousal of the sympathetic nervous system.
- Benzodiazepines (like Valium) are prescribed to decrease anxiety and muscle relaxation (the exact same dose): anxiety is about monitoring the level of tension in your body ... getting feedback from muscle tone; when your body signals relaxed muscles the brain realizes it doesn't need to feel so anxious: meditation & biofeedback: learning what thoughts allow the brain to regulate blood pressure, for example.
- After a fight, frequently a couple can apologize and everything is alright. But while still in the excited state, memories of prior issues can be brought out leading to another fight: the sympathetic nervous system hasn't realized that the cognitive situation is solved, so it still has hormones pushing you to the agitated state and the brain wonders why it is so upset and goes to find a good reason "from the second Roosevelt administration".
* In females, it takes longer for the sympathetic nervous system to return hormone levels back to baseline
* Similarly, after orgasm males go back to baseline faster than females
- Treat people with clinical depression to mechanically force themselves to smile, after a half hour they feel better
- Test scores received while sitting upright (vs. slouched over), result in happier assessments of the results and more pride. The posture of your spine influences your emotional response!

The Biology of Human Sexual Behavior

Details

Due to strong interest in this topic, "The Biology of Human Sexual Behavior" will be repeated on Saturday 15 Mar (follow this link to RSVP) (https://www.meetup.com/thinkingsociety/events/168419202/). Since the Saturday version is easier to get into, please RSVP to it if your schedule permits.

What can biology tell us about human sexual behavior? What kinds of releasing stimuli lead to sexual behavior? What is the effect of pheromones, hormones, genes and our evolutionary history upon our behavior? Robert Sapolsky discusses these issues and more in a broad biological survey of human behavior that starts with an ethologists attention to understanding the behavior itself and then examines the full timeline of biological factors that lead to the behavior from the most proximal (closest) to the most distal (furthest) factors: neurology, releasing stimuli, acute and chronic hormonal situation, cultural factors, perinatal biology and environment, genetics, and the evolutionary and environmental influences.

This discussion is based on 2½ Robert Sapolsky videos totaling 4 hours of lectures. Watching the videos and reading my notes are optional, but the material is so fascinating that I invite you to delve into it and explore it more deeply noting any questions that occur to you.

Human Sexual Behavior I (http://www.youtube.com/watch?v=LOY3QH_jOtE) (1h 40m video). In this video Sapolsky introduces his approach to comprehensively explore the biology of sexual behavior. After the introduction he explores big picture issues including the scientific approach to discussing sexual behavior and the challenges of the science. Then he explores what is unique about human sexual behavior in the animal kingdom. Then he discusses the neurobiology of sex followed by its endocrinology (hormonal system). At the end he begins discussing the releasing stimuli for sexual behavior. The rest of that discussion continues in the next video. Read my extensive notes summarizing Sapolsky's discussion. (https://plus.google.com/104222466367230914966/posts/Z4AHhac1eqi)

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Human Sexual Behavior II (http://www.youtube.com/watch?v=95OP9rSjxzw) (1h 40m video). In this video Sapolsky continues with the discussion about releasing stimuli for sexual behavior. Then he discusses perinatal factors, genetic effects, and the evolutionary history of human sexual behavior. Along the way we learn more about the biology of homosexuality and transexuality. Is each of us intersexual? Read my extensive notes summarizing Sapolsky's discussion. (https://plus.google.com/104222466367230914966/posts/6rTVmAdkvdG)

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I have led several prior discussions on Robert Sapolsky whose descriptions (and videos) you may enjoy. The Uniqueness and Evolution of Humans (15 Apr 2012) (https://www.meetup.com/thinkingsociety/events/50061542/) is based on a commencement speech Sapolsky delivered. The other discussions have been based on Sapolsky's course BIO 250, HUMBIO 160: Human Behavioral Biology (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D). There were two discussions on "The Evolutionary and Genetic Bases of Human Behavior (https://www.meetup.com/thinkingsociety/events/116303742)" which covered videos 2-7 of the course on 14 Jul 2013 (https://www.meetup.com/thinkingsociety/events/116303742) and 27 Jul 2013 (https://www.meetup.com/thinkingsociety/events/128381212), two discussions on "The Biology of Learning" which covered videos 8 & 9 of the course on 10 Nov 2013 (https://www.meetup.com/thinkingsociety/events/144382142/) and 30 Nov 2013 (https://www.meetup.com/thinkingsociety/events/148844402/). There were three discussions on "Brain Science and Human Behavior (https://www.meetup.com/thinkingsociety/events/153389892)" which covered videos 10-14 of the course on 12 Jan 2014 (https://www.meetup.com/thinkingsociety/events/153389892/), 18 Jan 2014 (https://www.meetup.com/thinkingsociety/events/158107662/), and 2 Feb 2014 (https://www.meetup.com/thinkingsociety/events/161011212/).

CJ Fearnley

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Join me on Sat March 15th to discuss the Biology of Human Sexual Behavior. Read the description for full details including links to the four (4) hours of Robert Sapolsky videos on which the discussion will be based.

meetup.com

The Biology of Human Sexual Behavior (repeat)

Details

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This topic is a repeat of the one on Sunday March 9th. (https://www.meetup.com/thinkingsociety/events/153541762/)

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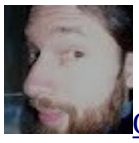 CJ Fearnley - 2014-01-22 23:04:39-0500 - Updated: 2014-03-14 19:13:14-0400
Human Sexual Behavior I. 101m Robert Sapolsky video.

Part 1 of 3 in a broad biological survey of the nature of human sexual behavior from neuroendocrinology to evolutionary history in the context provided by the animal world. If you want to understand some of the differences and similarities of the sexes, read my detailed notes below.

15. Human Sexual Behavior I

Plan for the rest of the course: look at the behavior from an ethological perspective. Then we will look at the timeline of what led to the behavior (from the big picture evolutionary and environmental influences all way to the neurology that triggered the behavior). Here is Sapolsky's

proximity of influence "timeline" for behavior:

evolution of species, peptides including the ecology and environmental situation, genetics of individual, perinatal biology & environment, culture, chronic and acute hormonal situation, the releasing stimuli, and the neurobiology that leads to the behavior.

Everything to the end (the right) depends on everything that comes before it (to the left). Proximal & distal explanations of behavior: to maximize the number of genes to pass on to the next generation (most distal) or for pleasure (proximal). With sex the driving forces are very proximal: close to the behavior on the behavior timeline. This lecture focuses on the neurobiology of sex and just starts discussing the releasing stimuli of sex at the end.

Fixed action patterns (the ethologists answer to the question: What is the behavior?) of sexual behavior across species tend to be strongly conserved within the species but tend to be very different between species.

The conservation of species reproductive specificity is due to the releasing stimulus of each mate providing the fixed action pattern for the other: this reinforcing interplay provides reproductive selectivity for those organisms whose sexual selection is effectively coordinated.

The lingo for discussing sexual behavior:
libido: referring to sexual arousal and motivation; horniness
The following three words are preferred:
attractivity: how attractive an individual is to another
proceptivity: the active behaviors that are done in response to being attracted by another
receptivity: how receptive an individual is to the interest of another

Martha McClintoch quote "studying female rat sexual behavior in a cage is like trying to study the swimming behavior of a dolphin in a bathtub" Female rats will engage in proceptive courting behaviors if they are not confined to a cage (dogma used to be that rat females are very passive, receptive).

Mathematical biologist Joel Cohen proposed in the 1980s (http://www.rockefeller.edu/labheads/cohenje/PDFs/142Cohen.pdf, http://lab.rockefeller.edu/cohenje/PDFs/162StatConceptsAIDSSymposiumStatSciIndustryPublPolicy1989.pdf) using the method of randomized response originally proposed by Fiddler and Kleinknect in 1977 to determine actual patterns of behavior despite the potentially embarrassing nature of some questions.

Fertility does not depend on female orgasm. So why did it evolve? Monkeys and Apes have female orgasm. [Note: these studies do not receive federal grant money.] Theories: Female orgasm may facilitate fertilization (facilitation). Sperm motility is improved with vaginal secretions which are enhanced in female orgasm (not proven). Other theories: orgasm exhausts the female, so she lays down so sperm don't need to swim against gravity. Reinforcement theory: it feels good, so increases the behavior. But most studies have shown no relationship between fertility and propensity to orgasm. Female orgasm appears to play no role in reproductive success. Why are clitoral orgasms more easily brought about than vaginal orgasm? Sapolsky: it may be a spandrel! Male reproductive success is improved by orgasm and females may carry along that physiology as evolutionary baggage. Similarly nipples are spandrels in males.

What isn't unique to humans?
Non-reproductive sex: bonobo chimps, cetaceans like dolphins. In most species, the endocrinology

of ovulation makes sex pleasurable (for both female and male). In humans, females do not need higher estrogen levels to have "tactile responsiveness to sexually arousing stimuli" (however, it enhances the effect).
Foreplay: bonobos have more patience with foreplay than do humans
Homosexuality & Masturbation: not just in zoos, ethological studies have documented it in other species.
Fantasy: difficult to know, but Sapolsky tells of a low-ranking juvenile baboon who masturbated after the hottest baboon female walked by (could be pheromone induced)

What is unique to humans?
Egalitarian sex: in humans, unlike marmoset monkeys, all adults are allowed to reproduce
Sex in private: in no other species is most sexual behavior done out of sight of others
Men sometimes psychopathologically confusing sexual behavior with violence

Marriage: all human cultures have some form of it: more than 90% of people wind up in each culture's equivalent of a permanent stable relationship. Even though the majority of human cultures have been polygamous, nonetheless, the vast majority of individuals have been in monogamous relationship. But there is a lot less monogamy than you might think. Alfred Kinsey questionnaires and other research shows that humans have high degrees of social monogamy but much less sexual monogamy. Between 10 and 40% of children do not have the father they are supposed to have.

Romance is viewed as a relatively new cultural invention. Sapolsky suggests a couple of centuries old. Sapolsky asserts that the notion that romance and passion should persist throughout a lifetime is only about 30-50 years old in most westernized cultures. Do you believe this? Doesn't "The Tale of Genji" and Dante's "Commedia" refute the hypothesis? Is this an arrogance of modern scholarship or does the historical record support the thesis? Wikipedia doesn't shed much light on the subject: https://en.wikipedia.org/wiki/Romance_(love). Plato's Symposium has an interesting speech by Aristophanes which suggests to me that the passion of romance was strong in Greek understanding.

Across cultures the average duration of marriage is 2-4 years (roughly corresponds to the inter-birth interval). Humans tend to be serial monogamists.

Compared to many animals, in humans sex is relatively "normal". Many species are hermaphroditic (individuals with male and female sex organs), opportunistic sex changing species (fish), parthenogenesis (reproduction without fertilization: some snakes reproduce parthenogenetically only after sex with a male whose sperm is not used)

The neurobiology of sexual behavior is heavily centered in the limbic system.

Klüver–Bucy syndrome: brain damage to the limbic system causing sex with inanimate objects, and other behavior changes.

The ventral medial hypothalamus is implicated in female sexual behavior: if stimulated you get the behavior of an ovulating female, damage it and sexual behavior goes away. It is the location of estrogen and progesterone receptors. The midbrain also appears to be involved in female sexual behavior. The lordosis reflex (female back-arching effect in hamsters) indicates special spinal pathways that do not exist in males.

The medial preoptic area is the location of testosterone and androgen receptors and is implicated in male sexual behavior (especially performance). The amygdala is involved in male sexual motivation (in addition to its role in fear, anxiety, and aggression). This may be why it is far more

likely for male humans to confuse sexuality with aggression.

The autonomic nervous system is involved in penile erection (parasympathetic nervous system which is involved in growth, repair, & vegetative functions) and ejaculation (sympathetic nervous system which is involved in arousal and the fight or flight reflex). The same mechanism is behind clitoral erections in females.

Vascular v. muscular erections: vascular erections are caused by increased blood flow to the penis and blocking its escape leading to vascular engorgement. In muscular erections (such as in rodents) there is the erector levi muscle. Vascular erections last longer, muscular ones can happen quicker. Both methods use the same autonomic physiology.

In females it takes longer for the sympathetic nervous system to return to baseline after arousal (whether an orgasm or a fight).

There are many regions of the brain with sexual dimorphism (difference in size depending on gender) both in the size of a nuclei (center of neuron cell bodies where the nucleus is situated) and the number of axons (the part of a neuron that "connects" to other neurons) between centers. The INAH3 (third interstitial nucleus of the anterior hypothalamus) is a small nucleus in the hypothalamus which is about twice the size in men than women.

But much of the neuroanatomy is the same in males and females. Orgasm is the transition from parasympathetic to sympathetic nervous system activity. Premature ejaculation is when the transition from parasympathetic to sympathetic is too rapid. Women tend to have the more difficulty transitioning from parasympathetic to sympathetic (so, difficulty in reaching orgasm).

The neurobiology of anticipation, pleasure, & reward is the same in both sexes. The mesolimbic pathway is a dopaminergic pathway in the brain. The VTA (ventral tegmental area) sends a big dopaminergic projection to the NAcc (Nucleus accumbens) which then passes it all around the brain. If this pathway is depleted of dopamine, there will be a diminution of proceptivity. This frequently happens in clinical depression. Dopamine is about the anticipation of reward and then fueling the behavior needed to get the reward. If the reward is only presented half the time (intermittent reinforcement), then dopamine rises even higher than when it is a sure thing. "Maybe" is highly reinforcing! Brain scans of people presented with pornography activate the dopaminergic pathways (Sapolsky implies that males respond more than females).

The effects are subtle: if a guy sees an attractive to him female, the dopaminergic pathway activates if her eyes are directed towards him (not if she is looking elsewhere). Is this why we avoid eye contact? If the guy rates the woman as unattractive, his dopaminergic pathways activate if she is looking away. What is that about?

D1 & D2 are two dopamine receptor subtypes. In pair-bonded species (rodents), after mating the D2 receptors are down-regulated and the D1 receptors are up-regulated. D2 seems to reward mating (the attachment) and D1 rewards pair-bonding (faithfulness). In humans one study found a very small effect between the D1/D2 ratio and more stable, longer lasting relationships.

In a brain scan study, seeing a picture of one's beloved activates the dopaminergic pathways if they have been together for 2½ years, but if they were together for 5 years there is dopaminergic response instead in the activation of the AC (anterior cingulate) which is associated with empathy and comfort.

Sapolsky's college dormmate said "a relationship is the price you pay for the anticipation of it". Is

dopamine the basis for this?

In general, the neurobiology of homosexuality works the same: just switch the gender of one of the individuals. What does that mean?

The role of the frontal cortex: regulating behavior, impulse control

Gratification postponement plays a large role in sexual behavior. The frontal cortex teaches you appropriate sexual behavior, it keeps you from doing things you will regret afterwards. Cortical damage can cause highly inappropriate sexual behavior (an 80 year old guy with stroke damage to his prefrontal cortex raped an 80 year old woman). Sometimes (e.g., in ungulates = hooved animal) the frontal cortex is involved in getting you to engage in terrifying courtship behaviors. Mostly the frontal cortex is about reigning in sexual behavior that would be inappropriate: it is changing the context in which the fixed action patterns occur.

What are the hormonal responses to sexual behavior?

In females, having sex increases secretion of progesterone-derived hormones (reinforcing the pleasure), testosterone and other androgens (females have only 5% of the levels of males, but levels rise after sex), oxytocin (a hormone, neurotransmitter, and a neuromodulator). In females, androgens (produced in the adrenal gland) are involved in mediating sexual motivation (arousal). In human females, removal of the adrenal glands tends to reduce sexual motivation; give them replacement androgens and their sexual arousal returns. Oxytocin plays a central role in forming attachments. Ocytocin aerosols in the nose makes them more trusting (they are more likely to agree with an argument and play games more cooperatively). Hence the new field of neuromarketing. Woah, is that OK: oxytocin coming out of my TV set before an ad comes on? Oxytocin's main role appears to be for nursing behavior. Hence the theory that monogamy may be a descendant of the neurobiology of mother-offspring attachment.

In males, having sex increases testosterone, vasopressin (also a neuromodulator). The evidence that high testosterone levels make males more sexually active is non-existent. Vasopressin is a neuromodulator: it is to males as oxytocin is to females. Vasopressin is critical for males to form a pair-bond. In monogamous species, the vasopressin receptor gene is expressed on neurons that release dopamine. A gene transfer study in voles showed that polygamous males become monagamous after getting the "right" genes. Those males with more receptors form pair-bonds faster. In monkeys, Marmosets pair-bond (and they have the gene), Rhesus monkeys (tournament species, polygamous, they have the "right" genes for that). Chimps have the polygamous vasopressin receptor gene. Bonobos have the monagamous gene version, but they are polygamous! In humans, the gene is about half-way between the polygamous & monagamous versions. Two studies in humans show that the monagamous version correlates to a high chance of getting married, the marriage is likely to last longer, and both partners are more likely to rate the marriage as stable and happy (but it is a small effect). In families with autism there are mutations on the vasopressin gene where subjects show very little attachment to other humans.

Oxytocin & vasopressin are associated with social attachment in animals & humans.

Neurobiology of sexual orientation

INAH3 is twice the size in males in other species & humans. Simon LeVay showed that the INAH3 in gay men is half the size as in heterosexual men (about the same size as in heterosexual women). The study has been replicated and the effect is distinct. Since the brains were taken from AIDS patients, that may confound the effect. Dick Swaab found the region adjacent to INAH3 is twice the

size in women and gay men than in straight men. Swaab's findings were condemned by the gay community wheareas openly gay LeVay's findings were heralded. Gay pride: "the only thing small about me is the size of my sexually dimorphic nucleus". LeVay's 1991 paper in the journal Science "the most influential science journal in this country", influenced the debate about gays in the military during the 1992 presidential election (the paper was held for a few months to maximize its impact on the election).

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Sapolsky mentions that the otoacoustic reflex is a vibration in the ear that differs by sex and sexual orientation, but Wikipedia's description and Sapolsky's may not agree (cf. http://en.wikipedia.org/wiki/Neuroscience_and_sexual_orientation#Auditory_evoked_potentials).

Sapolsky implies that these effects are due to the prenatal hormonal environment. He also mentions that gay women show similar effects for digit lengths and ear vibrations.

Neurobiology of transsexuality, 1:24:40-1:29:45

Being homosexual was considered a psychopathology until the 1970s. Transsexuality is still considered a psychopathology by the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders.

The central subdivision of the bed nucleus of the stria terminalis is reliably twice as large in men as in women except in a study of transsexuals where they had the size normally associated with the sex that they insisted they were. So do transexuals have the wrong bodies for their brain? Wikipedia suggests the story isn't as clear as Sapolsky presents it (http://en.wikipedia.org/wiki/Stria_terminalis#Bed_nucleus_of_the_stria_terminalis_.28BNST.29). Sapolsky is referring to another Swaab study published in Nature (http://www.nature.com/nature/journal/v378/n6552/abs/378068a0.html), but Wikipedia and the Nature article appear to involve only six subjects. Why does he think it is a strong effect? When the penis is removed due to cancer, there is often phantom penile sensation. But transgendered individuals do not report phantom penile sensation.

Releasing stimuli for sexual behavior

Male turkeys depend on visual stimuli (styrofoam turkey experiments). Rhesus monkeys are visually attracted to females in estrus. Humans are also highly visual in our sexual responsiveness. Male humans are more responsive than females shown by brain-imaging studies showing more dopaminergic response as well as an effect in the amygdala to visually arousing material. Tactile responsiveness is also well established. Some tactile stimuli are more arousing than others (erogenous zones). Tactile responses vary depending on hormone levels. In women, ovulation enhances tactile responsiveness. In men, more testosterone enhances tactile responsiveness (castration reduces responsiveness).

Sex pheromones are generated only when the testosterone (male) or estrogen (female) levels are high enough. Pheromones tend to be the breakdown products of sex hormones (androgens in males; estrogens in females). Olfactory receptors can detect remnants of the sex hormones. Perfumes have typically been made from the sweat of male animals. Chanel #5 is made from "whipped male Abyssinian cats". Synthetic perfumes tend to be made from synthetic versions of androgens.

Attractive to females, but not so much to males. Olfactory communication tells the species, the gender, the relative strength of their sex hormones, it tells about their health, if they are afraid, and how related they are. Men without testosterone and women without estrogen will not be able to detect the sex pheromones that are present. Women can detect male pheromones better when they are ovulating. Gay men can detect the smell of gay men better than straight men or women.

CJ Fearnley

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Human Sexual Behavior I. 101m Robert Sapolsky video.
Part 1 of 3 in a broad biological survey of the nature of human sexual behavior from neuroendocrinology to evolutionary history in the context provided by the animal world. If you want to understand some of the differences and similarities of the sexes, read my detailed notes below.
http://www.youtube.com/watch?v=LOY3QH_jOtE
Plan for the rest of the course: look at the behavior from an ethological perspective. Then we will look at the timeline of what led to the behavior (from the big picture evolutionary and environmental influences all way to the neurology that triggered the behavior). Here is Sapolsky's proximity of influence "timeline" for behavior:
evolution of species, peptides including the ecology and environmental situation, genetics of individual, perinatal biology & environment, culture, chronic and acute hormonal situation, the releasing stimuli, and the neurobiology that leads to the behavior.
Everything to the end (the right) depends on everything that comes before it (to the left). Proximal & distal explanations of behavior: to maximize the number of genes to pass on to the next generation (most distal) or for pleasure (proximal). With sex the driving forces are very proximal: close to the behavior on the behavior timeline. This lecture focuses on the neurobiology of sex and just starts discussing the releasing stimuli of sex at the end.
Fixed action patterns (the ethologists answer to the question: What is the behavior?) of sexual behavior across species tend to be strongly conserved within the species but tend to be very different between species.
The conservation of species reproductive specificity is due to the releasing stimulus of each mate providing the fixed action pattern for the other: this reinforcing interplay provides reproductive selectivity for those organisms whose sexual selection is effectively coordinated.
The lingo for discussing sexual behavior:
libido: referring to sexual arousal and motivation; horniness
The following three words are preferred:
attractivity: how attractive an individual is to another
proceptivity: the active behaviors that are done in response to being attracted by another
receptivity: how receptive an individual is to the interest of another
Martha McClintoch quote "studying female rat sexual behavior in a cage is like trying to study the swimming behavior of a dolphin in a bathtub"

Female rats will engage in proceptive courting behaviors if they are not confined to a cage (dogma used to be that rat females are very passive, receptive).
Mathematical biologist Joel Cohen proposed in the 1980s (http://www.rockefeller.edu/labh.../cohenje/PDFs/142Cohen.pdf, http://lab.rockefeller.edu/.../162StatConceptsAIDSSymposi...) using the method of randomized response originally proposed by Fiddler and Kleinknect in 1977 to determine actual patterns of behavior despite the potentially embarrassing nature of some questions.
Fertility does not depend on female orgasm. So why did it evolve? Monkeys and Apes have female orgasm. [Note: these studies do not receive federal grant money.] Theories: Female orgasm may facilitate fertilization (facilitation). Sperm motility is improved with vaginal secretions which are enhanced in female orgasm (not proven). Other theories: orgasm exhausts the female, so she lays down so sperm don't need to swim against gravity. Reinforcement theory: it feels good, so increases the behavior. But most studies have shown no relationship between fertility and propensity to orgasm. Female orgasm appears to play no role in reproductive success. Why are clitoral orgasms more easily brought about than vaginal orgasm? Sapolsky: it may be a spandrel! Male reproductive success is improved by orgasm and females may carry along that physiology as evolutionary baggage. Similarly nipples are spandrels in males.
What isn't unique to humans?
Non-reproductive sex: bonobo chimps, cetaceans like dolphins. In most species, the endocrinology of ovulation makes sex pleasurable (for both female and male). In humans, females do not need higher estrogen levels to have "tactile responsiveness to sexually arousing stimuli" (however, it enhances the effect).
Foreplay: bonobos have more patience with foreplay than do humans
Homosexuality & Masturbation: not just in zoos, ethological studies have documented it in other species.
Fantasy: difficult to know, but Sapolsky tells of a low-ranking juevenile baboon who masturbated after the hottest baboon female walked by (could be pheromone induced)
What is unique to humans?
Egalitarian sex: in humans, unlike marmoset monkeys, all adults are allowed to reproduce
Sex in private: in no other species is most sexual behavior done out of sight of others
Men sometimes psychopathologically confusing sexual behavior with violence
Marriage: all human cultures have some form of it: more than 90% of people wind up in each culture's equivalent of a permanent stable relationship. Even though the majority of human cultures have been polygamous, nonetheless, the vast majority of individuals have been in monogamous relationship. But there is a lot less monogamy than you might think. Alfred Kinsey questionnaires and other research shows that humans have high degrees of social monogamy but much less sexual monogamy. Between 10 and 40% of children do not have the father they are supposed to have.
Romance is viewed as a relatively new cultural invention. Sapolsky suggests a couple of centuries old. Sapolsky asserts that the notion that romance and passion should persist throughout a lifetime is only about 30-50 years old in most westernized cultures. Do you believe this? Doesn't "The Tale of Genji" and Dante's "Commedia" refute the hypothesis? Is this an arrogance of modern scholarship or does the historical record support the thesis? Wikipedia doesn't shed much light on the subject: https://en.wikipedia.org/wiki/Romance_(love). Plato's Symposium has an interesting speech by Aristophanes which suggests to me that the passion of romance was strong in Greek understanding.

Across cultures the average duration of marriage is 2-4 years (roughly corresponds to the inter-birth interval). Humans tend to be serial monogamists.
Compared to many animals, in humans sex is relatively "normal". Many species are hermaphroditic (individuals with male and female sex organs), opportunistic sex changing species (fish), parthenogenesis (reproduction without fertilization: some snakes reproduce parthenogenetically only after sex with a male whose sperm is not used)
The neurobiology of sexual behavior is heavily centered in the limbic system.
Klüver–Bucy syndrome: brain damage to the limbic system causing sex with inanimate objects, and other behavior changes.
The ventral medial hypothalamus is implicated in female sexual behavior: if stimulated you get the behavior of an ovulating female, damage it and sexual behavior goes away. It is the location of estrogen and progesterone receptors. The midbrain also appears to be involved in female sexual behavior. The lordosis reflex (female back-arching effect in hamsters) indicates special spinal pathways that do not exist in males.
The medial preoptic area is the location of testosterone and androgen receptors and is implicated in male sexual behavior (especially performance). The amygdala is involved in male sexual motivation (in addition to its role in fear, anxiety, and aggression). This may be why it is far more likely for male humans to confuse sexuality with aggression.
The autonomic nervous system is involved in penile erection (parasympathetic nervous system which is involved in growth, repair, & vegetative functions) and ejaculation (sympathetic nervous system which is involved in arousal and the fight or flight reflex). The same mechanism is behind clitoral erections in females.
Vascular v. muscular erections: vascular erections are caused by increased blood flow to the penis and blocking its escape leading to vascular engorgement. In muscular erections (such as in rodents) there is the erector levi muscle. Vascular erections last longer, muscular ones can happen quicker. Both methods use the same autonomic physiology.
In females it takes longer for the sympathetic nervous system to return to baseline after arousal (whether an orgasm or a fight).
There are many regions of the brain with sexual dimorphism (difference in size depending on gender) both in the size of a nuclei (center of neuron cell bodies where the nucleus is situated) and the number of axons (the part of a neuron that "connects" to other neurons) between centers. The INAH3 (third interstitial nucleus of the anterior hypothalamus) is a small nucleus in the hypothalamus which is about twice the size in men than women.
But much of the neuroanatomy is the same in males and females. Orgasm is the transition from parasympathetic to sympathetic nervous system activity. Premature ejaculation is when the transition from parasympathetic to sympathetic is too rapid. Women tend to have the more difficulty transitioning from parasympathetic to sympathetic (so, difficulty in reaching orgasm).
The neurobiology of anticipation, pleasure, & reward is the same in both sexes. The mesolimbic pathway is a dopaminergic pathway in the brain. The VTA (ventral tegmental area) sends a big dopaminergic projection to the NAcc (Nucleus accumbens) which then passes it all around the brain. If this pathway is depleted of dopamine, there will be a diminution of proceptivity. This frequently happens in clinical depression. Dopamine is about the anticipation of reward and then fueling the behavior needed to get the reward. If the reward is only presented half the time (intermittent reinforcement), then dopamine rises even higher than when it is a sure thing. "Maybe" is highly reinforcing! Brain scans of people presented with pornography activate the dopaminergic pathways (Sapolsky implies that males respond more than females).

The effects are subtle: if a guy sees an attractive to him female, the dopaminergic pathway activates if her eyes are directed towards him (not if she is looking elsewhere). Is this why we avoid eye contact? If the guy rates the woman as unattractive, his dopaminergic pathways activate if she is looking away. What is that about?
D1 & D2 are two dopamine receptor subtypes. In pair-bonded species (rodents), after mating the D2 receptors are down-regulated and the D1 receptors are up-regulated. D2 seems to reward mating (the attachment) and D1 rewards pair-bonding (faithfulness). In humans one study found a very small effect between the D1/D2 ratio and more stable, longer lasting relationships.
In a brain scan study, seeing a picture of one's beloved activates the dopaminergic pathways if they have been together for 2½ years, but if they were together for 5 years there is dopaminergic response instead in the activation of the AC (anterior cingulate) which is associated with empathy and comfort.
Sapolsky's college dormmate said "a relationship is the price you pay for the anticipation of it". Is dopamine the basis for this?
In general, the neurobiology of homosexuality works the same: just switch the gender of one of the individuals. What does that mean?
The role of the frontal cortex: regulating behavior, impulse control
Gratification postponement plays a large role in sexual behavior. The frontal cortex teaches you appropriate sexual behavior, it keeps you from doing things you will regret afterwards. Cortical damage can cause highly inappropriate sexual behavior (an 80 year old guy with stroke damage to his prefrontal cortex raped an 80 year old woman). Sometimes (e.g., in ungulates = hooved animal) the frontal cortex is involved in getting you to engage in terrifying courtship behaviors. Mostly the frontal cortex is about reigning in sexual behavior that would be inappropriate: it is changing the context in which the fixed action patterns occur.
What are the hormonal responses to sexual behavior?
In females, having sex increases secretion of progesterone-derived hormones (reinforcing the pleasure), testosterone and other androgens (females have only 5% of the levels of males, but levels rise after sex), oxytocin (a hormone, neurotransmitter, and a neuromodulator). In females, androgens (produced in the adrenal gland) are involved in mediating sexual motivation (arousal). In human females, removal of the adrenal glands tends to reduce sexual motivation; give them replacement androgens and their sexual arousal returns. Oxytocin plays a central role in forming attachments. Ocytocin aerosols in the nose makes them more trusting (they are more likely to agree with an argument and play games more cooperatively). Hence the new field of neuromarketing. Woah, is that OK: oxytocin coming out of my TV set before an ad comes on? Oxytocin's main role appears to be for nursing behavior. Hence the theory that monogamy may be a descendant of the neurobiology of mother-offspring attachment.
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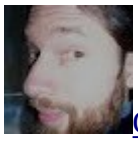 CJ Fearnley - 2014-02-15 23:53:49-0500 - Updated: 2014-03-14 23:04:16-0400
Human Sexual Behavior II. 100m Robert Sapolsky video.

Part 2 of 3 in a broad biological survey on the nature of human sexual behavior from neuroendocrinology to evolutionary history. This discussion starts with a general review of Part 1 then picks up where he left off on pheromones.

16. Human Sexual Behavior II

Releasing Stimuli for sexual behavior

Women have greater preference for the smell of males around the time of ovulation (their noses become more sensitive at this time; estrogen has receptors on olfactory neurons). Men rate the smells of females as less unpleasant when the woman is ovulating (depends on testosterone in their systems). Physiological effects: The Wellesley effect (synchronizing female menstrual cycles) is pheromone based. In rodents, the pheromones of an adult female will delay the onset of puberty of younger females and decrease the onset of induced ovulation (unless they are sisters: kin selection!). In rodents and some primates, when males smell the pheromones of dominant males it drives down their testosterone levels. In some species, the same dominant pheromones stimulates sperm production: "a coevolutionary arms race". In rodents, male pheromones stimulates onset of puberty in females and the onset of induced ovulation (such as in pigs where Boar-Mate, a variant of pig pheromone derived from truffles, is an ovulation inducer) (unless the two are siblings: kin selection). In lots of species female pheromones increases testosterone and sperm production in males.

Sapolsky describes the experiments in the paper "Effects of sexual activity on beard growth in man" by Anonymous in Nature 226, pp. 869–70, 1970 where the researcher measured beard growth after sexual activity vs. his extended periods of time alone. The effect is possibly due to pheromones, but there are many possible confounds and the sample size is n = 1.

Homosexual men prefer pheromones from men with higher testosterone levels suggesting some interesting biological underpinnings of homosexuality.

Other releasing stimuli: giraffes lick private parts to get gustatory information, auditory priming in

moose. In human females their voices get a little bit higher during ovulation which is subliminally detected by men (no conscious awareness).

The most erogenous organ in humans is the brain. Thought as a releaser: it often bypasses the world of smell and sound. Coolidge effect: variety can stimulate sexual arousal even after a male is sexually sated.

Fear suppresses sexual behavior. Chronic stress is extremely suppressive of sexual behavior. Short term stress may stimulate or suppress arousal. For example, there are plenty of historical examples of violent sexual behaviors associated with warfaring.

In most species, females only engage in proceptive behavior around the time they are ovulating. In non-human primates the relationship isn't so strict. In humans one study found heightened arousal, proceptivity, and sexual activity (including likelihood of orgasm) near day 14 (around the time of high estrogen levels and ovulation) and day 28 (more relaxed about fear of pregnancy???). The study has not been easy to replicate except the part about day 14. Women tend to wear more provocative clothing around the time of ovulation. Geoffrey Miller reported that around the time of ovulation, lap dancers get larger tips.

Estrogen increases expression of receptors for progesterone. Estrogen increases the synthesis of oxytocin. In voles (and maybe humans), social affiliation of females is enhanced at the time of ovulation. Estrogen increases sensitivity to touch in some parts of the body. Estrogen both affects the brain and the periphery to increase proceptivity around the time of ovulation. In females, androgens (male sex hormones) play a role in proceptivity, arousal and motivation.

The time of year when males mate correlates to the time of year in which testosterone levels are elevated. In humans testosterone levels increase at puberty and start decreasing after age 30 with a correlative profile of sexual behavior. More sexually active men tend to have higher testosterone levels. There is a decrease in testosterone levels (and increased vasopressin) around the time they become fathers. Testosterone in fact does cause increased male sexual behavior: after castration, there is a big drop off in male sexual behavior; then when testosterone is added back in, sexual behavior levels return. But the subtraction experiment does not eliminate male sexual behavior (in all species!). Moreover, when adding testosterone back if the level is only 10% or even 200%, the sexual behavior returns to pre-castration levels: so those brain regions that drive male sexual behavior require sufficient testosterone but are not affected by quantity. The more sexual experience before castration, the more sexual behavior is retained afterward. So there is part of male sexual behavior driven by social experience and not the hormones. If testosterone levels are increased to supraphysiological levels (1000% of normal), then you do start seeing increases in male proceptivity (an issue for anabolic steroid abusers despite the partially compensatory down regulatory response). The same testosterone levels similarly affect male aggression. In sum, testosterone is playing a modulatory role.

Melatonin is a hormone that tells the body what time of year it is: it is sensitive to light. It drives seasonal mating patterns in some species. There is some evidence for a seasonal pattern to human sexual behavior, but it is small compared to many other species.

Perinatal factors: so far, the evidence is that there is very little about early life experience that affects the quality (the way) of later sexual behavior. Early experience is not about how to be sexual (those fixed action patterns are effectively built-in), but rather about learning the appropriate social context for proceptive behavior. The same pattern holds for aggression.

Rhesus monkeys and others reared (unethically perhaps) without the social context of any parents or

peers grow up with the same sexual fixed action patterns, but without any understanding of the social context appropriate to those behaviors (e.g., the normal male fixed action pattern may occur with a towel or bowl of food or ignores the social dominance hierarchy). Kibbutz literature suggests that if you spend lots of time with someone before you both turn six, chances are that when you grow up they will not be considered a mate but more like a sibling (pseudokin).

Early 20th century literature espoused the absence of a father model for making boys gay (the idea is the father may provide training for the appropriate context for appropriate proceptive behavior). The other leading model for explaining homosexuality was the neurotic mother explanation. No evidence for either model: they are complete nonsense.

Perinatal hormone levels affect sexual behavior in many species. In humans, the jury is still out and more research is needed. The "organizational effect" of a hormone is its impact on development (on the organs of the body but also its impact on the brain). The "activational effect" of a hormone is how it activates aspects of behavior (such as the sexual behavior apparatus that develops during puberty). In rodents, sex hormones have huge organizational effects that dramatically influence sexual behavior. Some 300 species have been documented to have homo- and hetero-sexual behavior in natural settings. In primates, If and only if there is prenatal masculinization (exposure of fetus to testosterone), then testosterone will have an acute activational effect (leading to male fixed action patterns). In human females there are two known ways to become masculinized: congenital adrenal hyperplasia (a genetic disorder) where the adrenal glands pump out lots of testosterone and through the drug DES (diethylstilbestrol) which is converted to androgens. There is a higher likelihood that prenatal masculinization leads to lesbianism, but there is a strange confound: the masculinization also created weird intersexual genitals which resulted in plastic surgeries and all sorts of other social consequences as well as the hormonal effects. Sapolsky concludes that there is weak evidence that prenatal masculinization is associated with homosexual behavior as an adult (Wikipedia sites two later studies with ambiguous results).

sexual identity: what sex you feel you are
intersexual people have gender ambiguous genitalia
secondary sexual characteristics: those features that develop during puberty

In some parts of the body, testosterone (a steroid hormone with its hormone binding domain and DNA binding domain) is biochemically changed to dihydrotestosterone which behaves somewhat differently (this effect occurs in the genitalia and the locations for secondary sexual characteristics). In other parts of the body, testosterone is biochemically changed to estrogen to cause male behavior! This estrogen effect only happens in target cells (not in blood circulation) generally in the brain. What? Why don't the masculinizing effects of estrogen affect female brains causing everyone to have a male brain? Alpha-fetoprotein (AFP), the most abundant plasma protein found in the human fetus, during pregnancy it binds to estrogen breaking it down so that circulating estrogen never has an effect on the fetus (at least in rodents according to Wikipedia). So the only fetuses with estrogen are males who made it from their testosterone. Where do prenatal feminizing effects come from? Perhaps, the female brain is the default brain and it takes testosterone to masculinize it. Sapolsky argues that there are other hormones that produce the feminizing effects. The effects of gender on our brains are very complex!

Testicular-feminizing males are phenotypically female, but genetically male. It is a genetic condition caused by ineffective testosterone receptors. Although testicular-feminized males experience no masculinizing effects directly from testosterone and dihydrotestosterone, they still have testosterone in their brains biochemically changed to estrogen and therefore they have masculinized brains. Inference: testicular-feminized males develop a female sex identity due to socialization and not the masculinization of their brains. So do the prenatal hormonal effects

matter?

Genetic effects: what do genes have to do with sexual behavior?

Genes determine which gonads you make as a fetus which determines the kind of sex hormones released into the blood stream which determines what genitals you develop and the type of secondary sexual characteristics. Genes play a role in sex determination.

In monozygotic twins there is about 50% concordance with sexual orientation; in dizygotic twins it is 22%; and 9% with other siblings. So there is some suggestion that genes and/or pre-natal environment play some role in sexual orientation. Openly gay NIH scientist Dean Hamer reported finding genetic markers that were more likely to be shared between gay siblings. It was reported as "the gay gene": it is not a gene, it is a genetic marker: a statistical pattern in a genetic analysis. The marker showed no consistency: different pairs of twins shared different markers. Moreover, the finding has never been replicated despite substantial effort. Simon LeVay's INAH3 brain region size differentiator for homosexuality is scientifically compelling; Dean's genetic markers much less so.

Evolutionary History of Human Sexuality

"Organisms have sex for the good of the species." Sexual behavior is about reproduction: passing on copies of your genes. But humans and bonobo ("pigmy") chimps have non-reproductive sex. Bonobos are much different from Jane Goodall's tournament species (they are highly aggressive, fashion weapons, etc.). On the other side of the Congo, bonobos have virtually no sexual dimorphism, female dominance, and they have astonishing amounts of sex. What? A pair-bonding species (little sexual dimophism) with low aggression that is the most sexually promiscuous species on Earth? They have lots of non-reproductive sex: female not ovulating, homosexual, non-fertilizing sex (does he mean oral or anal sex?). Conclusion: sex must not be just for passing on copies of genes! Joan Roughgarden formerly at Stanford argues that there is far more non-reproductive sex in lots of species than the classical Darwinian notion of sexual selection can account for.

In bonobos sex appears to be about promoting group cohesion. Social grooming is important for social cohesion in many primate species (baboons will groom after a scare from a predator), decreasing individual tensions, reconciliation, etc. Solly Zuckerman's favored the theory that sexual behavior is purely for promoting group cohesion and decreasing violence. It became the dominant theory early in the 20th century. Sapolsky criticizes it severely: sexual behavior is the cause of huge amounts of aggression in many species.

Costs of sexual behavior: sperm do not cost much; eggs, pregnancy and post-natal care are very expensive. So sociobiology suggests that females are more selective about who they will mate with than males because of the differential costs of reproduction. Pair-bonded species like the marmosets provide an interesting exception: the male does as much child care as the female (they have twins). When the male caloric expenditure exceeds the female, you start to see cuckoldry (females abandoning their children to find a better mate) which is common in bird species. So the sociobiological argument that female selection is always more important has an important exception. In many species, the male attempts to control female reproductive behavior (is that common in humans?). In primates mate guarding is a male's attempt to keep his female away from any other guy. In humans, we find clitorectomy and chastity belts in cultures where males tend to go away for long periods of time such as nomadic pastoralists or during the Crusades.

The linear access model of reproduction suggests that male dominance rank is entirely predictive of male reproductive success: the top ranked N males get access to the N females that are in estrus (in

Baboons for example). Copulatory plugs occur in many canine (dog) species where semen hardens into a plug to block access by other males. In some fly species the penis is barbed so that after sex it detaches (he can grow a new one) and lodges in the female blocking access to other males. In some fly species there is also sperm competition where sperm use toxic substances to kill each other. In other species the male biochemically decreases the sexual attractiveness of the female subsequent to mating (by decreasing pheromones or proceptivity). "Viciously clever ways for males to control female reproductive behavior after they have left."

Females have developed counter strategies to male strategies to control female sexual behavior so the female gets more choice in the matter. Hidden ovulation in humans is a tactic to decrease the certainty of paternity (Sapolsky discounts the effects of pheromones and voice raising during ovulation which are subliminal and so consciously undetectable). One argument suggests hidden ovulation may reduce competitive infanticide while another suggests it decreases the male interest in controlling female sexual behavior because it is less clear when one needs to. Non-reproductive sex serves to fool the male when ovulation is actually occurring. Pseudo-estrus helps females in some species prevent competitive infanticide.

The leading cause of aggression in social species is male-male competition for reproductive access to females including humans. In monogamous species males produce small amounts of sperm (small testes) whereas in polygamous species males produce lots of sperm and have larger testes. Chimps have gigantic testicles per body size but not Gorillas. Humans are intermediate in testes size between pair-bonded and polygamous/tournament species.

Evolutionary aspects of female choice: In classic tournament species, females have very little choice (linear access model). By 1980s it became clear that there is little evidence for strict linear access models in actual behavior (female primatologists entered the field and started looking for evidence). Female choice was discovered! In tournament species, there is sexual dimorphism (big males), so females must be more clever than the brute force techniques of males in these species. For example, she can exhaust the guy: every time he sits down or takes a nap, she moves away forcing him to move to maintain his control over her sexual access. In baboons, a female will walk right in front of the dominant guy's worst rival again and again and again until those two fight, then she runs off to the bushes and has a "stolen copulation" with the guy she is actually interested in. There is a suggestion in the data (unproven) that female baboons like to mate with male baboons who are nice to them. Barbara Smutz coined the term intersexual friendships to describe nonsexual male-female interactions. So being a nice guy is an alternative male strategy in a tournament species since there is female choice. Paternity studies have shown that the strategy works because although nice guys mate less often, then do not suffer the injuries and complications of fighting to maintain a high dominance position. So these "alternative strategies" can work.

In orangutans, an alternative strategy for the low-ranking males who never get to mate (they are physiologically smaller) was first discovered by pioneer researcher Birutė Galdikas when she observed low-ranking males raping females (rape is defined as the violent process of mating with a female against her will). Some fish species have an alternative mating strategy where some males pretend to be female (coloration, etc.).

CJ Fearnley's post

Human Sexual Behavior II. 100m Robert Sapolsky video.
Part 2 of 3 in a broad biological survey on the nature of human sexual behavior from neuroendocrinology to evolutionary history. This discussion starts with a general review of Part 1 then picks up where he left off on pheromones.
http://www.youtube.com/watch?v=95OP9rSjxzw
Releasing Stimuli for sexual behavior
Women have greater preference for the smell of males around the time of ovulation (their noses become more sensitive at this time; estrogen has receptors on olfactory neurons). Men rate the smells of females as less unpleasant when the woman is ovulating (depends on testosterone in their systems). Physiological effects: The Wellesley effect (synchronizing female menstrual cycles) is pheromone based. In rodents, the pheromones of an adult female will delay the onset of puberty of younger females and decrease the onset of induced ovulation (unless they are sisters: kin selection!). In rodents and some primates, when males smell the pheromones of dominant males it drives down their testosterone levels. In some species, the same dominant pheromones stimulates sperm production: "a coevolutionary arms race". In rodents, male pheromones stimulates onset of puberty in females and the onset of induced ovulation (such as in pigs where Boar-Mate, a variant of pig pheromone derived from truffles, is an ovulation inducer) (unless the two are siblings: kin selection). In lots of species female pheromones increases testosterone and sperm production in males.
Sapolsky describes the experiments in the paper "Effects of sexual activity on beard growth in man" by Anonymous in Nature 226, pp. 869–70, 1970 where the researcher measured beard growth after sexual activity vs. his extended periods of time alone. The effect is possibly due to pheromones, but there are many possible confounds and the sample size is n = 1.
Homosexual men prefer pheromones from men with higher testosterone levels suggesting some interesting biological underpinnings of homosexuality.
Other releasing stimuli: giraffes lick private parts to get gustatory information, auditory priming in moose. In human females their voices get a little bit higher during ovulation which is subliminally detected by men (no conscious awareness).
The most erogenous organ in humans is the brain. Thought as a releaser: it often bypasses the world of smell and sound. Coolidge effect: variety can stimulate sexual arousal even after a male is sexually sated.
Fear suppresses sexual behavior. Chronic stress is extremely suppressive of sexual behavior. Short term stress may stimulate or suppress arousal. For example, there are plenty of historical examples of violent sexual behaviors associated with warfaring.
In most species, females only engage in proceptive behavior around the time they are ovulating. In non-human primates the relationship isn't so strict. In humans one study found heightened arousal, proceptivity, and sexual activity (including likelihood of orgasm) near day 14 (around the time of high estrogen levels and ovulation) and day 28 (more relaxed about fear of pregnancy???). The study has not been easy to replicate except the part about day 14. Women tend to wear more provocative clothing around the time of ovulation. Geoffrey Miller reported that around the time of ovulation, lap dancers get larger tips.
Estrogen increases expression of receptors for progesterone. Estrogen increases the synthesis of oxytocin. In voles (and maybe humans), social affiliation of females is enhanced at the time of ovulation. Estrogen increases sensitivity to touch in some parts of the body. Estrogen both affects the brain and the periphery to increase proceptivity around the time of ovulation. In females, androgens (male sex hormones) play a role in proceptivity, arousal and motivation.

The time of year when males mate correlates to the time of year in which testosterone levels are elevated. In humans testosterone levels increase at puberty and start decreasing after age 30 with a correlative profile of sexual behavior. More sexually active men tend to have higher testosterone levels. There is a decrease in testosterone levels (and increased vasopressin) around the time they become fathers. Testosterone in fact does cause increased male sexual behavior: after castration, there is a big drop off in male sexual behavior; then when testosterone is added back in, sexual behavior levels return. But the subtraction experiment does not eliminate male sexual behavior (in all species!). Moreover, when adding testosterone back if the level is only 10% or even 200%, the sexual behavior returns to pre-castration levels: so those brain regions that drive male sexual behavior require sufficient testosterone but are not affected by quantity. The more sexual experience before castration, the more sexual behavior is retained afterward. So there is part of male sexual behavior driven by social experience and not the hormones. If testosterone levels are increased to supraphysiological levels (1000% of normal), then you do start seeing increases in male proceptivity (an issue for anabolic steroid abusers despite the partially compensatory down regulatory response). The same testosterone levels similarly affect male aggression. In sum, testosterone is playing a modulatory role.

Melatonin is a hormone that tells the body what time of year it is: it is sensitive to light. It drives seasonal mating patterns in some species. There is some evidence for a seasonal pattern to human sexual behavior, but it is small compared to many other species.

Perinatal factors: so far, the evidence is that there is very little about early life experience that affects the quality (the way) of later sexual behavior. Early experience is not about how to be sexual (those fixed action patterns are effectively built-in), but rather about learning the appropriate social context for proceptive behavior. The same pattern holds for aggression.

Rhesus monkeys and others reared (unethically perhaps) without the social context of any parents or peers grow up with the same sexual fixed action patterns, but without any understanding of the social context appropriate to those behaviors (e.g., the normal male fixed action pattern may occur with a towel or bowl of food or ignores the social dominance hierarchy). Kibbutz literature suggests that if you spend lots of time with someone before you both turn six, chances are that when you grow up they will not be considered a mate but more like a sibling (pseudokin).

Early 20th century literature espoused the absence of a father model for making boys gay (the idea is the father may provide training for the appropriate context for appropriate proceptive behavior). The other leading model for explaining homosexuality was the neurotic mother explanation. No evidence for either model: they are complete nonsense.

Perinatal hormone levels affect sexual behavior in many species. In humans, the jury is still out and more research is needed. The "organizational effect" of a hormone is its impact on development (on the organs of the body but also its impact on the brain). The "activational effect" of a hormone is how it activates aspects of behavior (such as the sexual behavior apparatus that develops during puberty). In rodents, sex hormones have huge organizational effects that dramatically influence sexual behavior. Some 300 species have been documented to have homo- and hetero-sexual behavior in natural settings. In primates, If and only if there is prenatal masculinization (exposure of fetus to testosterone), then testosterone will have an acute activational effect (leading to male fixed action patterns). In human females there are two known ways to become masculinized: congenital adrenal hyperplasia (a genetic disorder) where the adrenal glands pump out lots of testosterone and through the drug DES (diethylstilbestrol) which is converted to androgens. There is a higher likelihood that prenatal masculinization leads to lesbianism, but there is a strange confound: the masculinization also created weird intersexual genitals which resulted in plastic surgeries and all

sorts of other social consequences as well as the hormonal effects. Sapolsky concludes that there is weak evidence that prenatal masculinization is associated with homosexual behavior as an adult (Wikipedia sites two later studies with ambiguous results).
sexual identity: what sex you feel you are
intersexual people have gender ambiguous genitalia
secondary sexual characteristics: those features that develop during puberty
In some parts of the body, testosterone (a steroid hormone with its hormone binding domain and DNA binding domain) is biochemically changed to dihydrotestosterone which behaves somewhat differently (this effect occurs in the genitalia and the locations for secondary sexual characteristics). In other parts of the body, testosterone is biochemically changed to estrogen to cause male behavior! This estrogen effect only happens in target cells (not in blood circulation) generally in the brain. What? Why don't the masculinizing effects of estrogen affect female brains causing everyone to have a male brain? Alpha-fetoprotein (AFP), the most abundant plasma protein found in the human fetus, during pregnancy it binds to estrogen breaking it down so that circulating estrogen never has an effect on the fetus (at least in rodents according to Wikipedia). So the only fetuses with estrogen are males who made it from their testosterone. Where do prenatal feminizing effects come from? Perhaps, the female brain is the default brain and it takes testosterone to masculinize it? Sapolsky suggests there are other hormones that produce the feminizing effects. The effects of gender on our brains are very complex!
Testicular-feminizing males are phenotypically female, but genetically male. It is a genetic condition caused by ineffective testosterone receptors. Although testicular-feminized males experience no masculinizing effects directly from testosterone and dihydrotestosterone, they still have testosterone in their brains biochemically changed to estrogen and therefore they have masculinized brains. Inference: testicular-feminized males develop a female sex identity due to socialization and not the masculinization of their brains. So do the prenatal hormonal effects matter?
Genetic effects: what do genes have to do with sexual behavior?
Genes determine which gonads you make as a fetus which determines the kind of sex hormones released into the blood stream which determines what genitals you develop and the type of secondary sexual characteristics. Genes play a role in sex determination.
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The leading cause of aggression in social species is male-male competition for reproductive access to females including humans. In monogamous species males produce small amounts of sperm (small testes) whereas in polygamous species males produce lots of sperm and have larger testes. Chimps have gigantic testicles per body size but not Gorillas. Humans are intermediate in testes size between pair-bonded and polygamous/tournament species.

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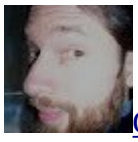 CJ Fearnley - 2014-03-07 18:45:01-0500 - Updated: 2014-03-07 18:45:01-0500
Human Sexual Behavior III & Aggression I. 100m Robert Sapolsky video.

Part 3 of 3 in a broad biological survey on the nature of human sexual behavior focuses on attractiveness. The second half of the video introduces part 1 of 4 in a broad biological survey of empathy and aggression. Extensive notes below.

17. Human Sexual Behavior III & Aggression I

Evolutionary theory suggests that the caloric expenditure of sperm (cheap) vs. eggs (expensive) implies more promiscuity on the part of males.

Female-female competition. In some pair-bonding species (such as new world monkeys and many bird species) females compete for the better "parental" males and so they are often more aggressive, have larger body size, and more prominent secondary sexual characteristics (those traits that appear in puberty).

5-20% of the population is homosexual in every human culture ever looked at. The evidence for a genetic basis for homosexuality is not established, so there may not be any role for natural selection. There are three theories for the selective advantage or neutrality of homosexuality. Hetereozygotic vigor argument (like sickle cell anemia where the homozygotic version is a disease but the heterozygotic variation protects against malaria): so it could be that homosexuality is the non-selective form but in heterozygotes it is adaptive (very little evidence). Gender-dependent argument: in one gender the genetically influenced trait is adaptive while in the other gender the trait is maladaptive and leads to homosexuality (evidence: the sisters of gay men tend to be more reproductively successful). Helper at the nest kin selection model: the individuals who are not passing on copies of their genes are helping their siblings (so both sisters & brothers would have

higher reproductive success and studies seem to mostly support this model).

Traits that are attractive.

Facial symmetry. Francis Galton developed composite photography which he used to determine the face of crime. Sapolsky belittles the technique and asserts that such composites average to a more attractive face (corroboration: http://faceresearch.org/students/averageness). The symmetry seems to be the quality that makes such composites attractive. The literature suggests that symmetry is a good indicator of health. babies at two months already prefer more symmetrical faces (rats & nonhuman primates also show a preference for symmetry). A study in Nature showed that people with more symmetrical faces were better dancers (this study was retracted by Nature in Dec 2013: http://www.nature.com/nature/journal/v438/n7071/full/nature04344.html). The faces of women when ovulating are slightly more symmetrical (http://www.nature.com/news/2004/040331/full/news040329-6.html).

Secondary sexual characteristics. Zahavi's handicap principle: the bigger more garish secondary sexual characteristics is a signal to the world of health, good immunity, or fertility (a signal that one has so much energy to conspicuously consume resources to build this garish trait to attract a mate). Sapolsky claims there is lots of evidence for the principle. Marsupial mice: those individuals with the more exaggerated secondary sexual characteristic had more fertile sperm. Immune system molecules may create the more showy trait. A study of women from many industrialized societies correlates inversely between a preference for men with big jutting jaws, high forehead, and musculature and life expectancy and economic health of that culture. Organisms do not want disease, so they want evidence of a strong immune system. They are good at detecting the smell of parasitic or other infections and avoiding those individuals. Some individuals may cheat: to uncouple their health from their secondary sexual characteristics (some bird species will put in extra energy to enhance their traits when they are sick: reproduce now or never?). Of course, counterstrategies and another coevolutionary back and forth is seen.

A problem with the handicap principle: "there is a certain way to frame things so that you never ever can get a finding that disagrees with your general stance". Vultures have sexually dimorphic faces. Male vultures faces are orange due to carotenoids from consuming ungulate feces with parasites. So dramatic secondary sexual characteristics can both be a marker of health or healthy enough despite some disease. The handicap principle remains controversial. Lions are a tournament species with strong sexual dimorphism (manes): a black mane would be most attractive based on the handicap principle, but it would heat your head in the African sun. So there are complex balancing issues.

Some secondary sexual characteristics are markers of fertility, such as, external swellings in ovulating females (estrus). Baboon males prefer females with larger swellings. Swellings are a marker of estrogen levels, greater fertility, and greater health. The biggest swellings may incur an extra 25 pounds of water weight and yet I can still run around the jungle as a marker of fitness and a source of attractiveness. Humans are concealed ovulators. Human females have no swellings, but a larger hip to waist ratio implies more fertility (child-bearing pelvises, development health augurs well for better fertility). Human males across cultures find larger hip to waist ratios more attractive. This could be a Western bias: the minimal literature on hip to waist ratio among non-Western cultures at first contact suggest that the attractiveness of the trait may be less strong. It is a widespread trait in almost every culture studied. Human females find big jutting jaws, high forehead, and musculature more attractive and these are markers for high testosterone levels during adolescence. Surprisingly, women rate rounder faces as more likable, more honest, more trustworthy, but less desirable! There are subtle differences that people are only subliminally aware of. During ovulation, women's attractiveness for these male secondary sexual characteristics

increases (as does pheromone sensitivity).

Confounds: female birds who mate with more attractive males, invest more effort in their kids who have bigger egg size, and therefore they do survive better. So is this a self-fulfilling prophesy or is it the handicap principle? Lee Dugatkin at University of Louisville (I could not find this study, could be a spurious citation): in situations where the female rejects the male, he makes the male appear to be very popular (stuffed females around the rejected male) then the female who spurned him is more likely to initiate proceptive behavior. This bandwagon effect has been shown in many species, but it may be a social contagion not a sign of fitness nor the handicap principle.

In many pair-bonded species attractiveness is shown by demonstrating parental competence (bringing worms) not markers of health or fertility. In most non-human primate species studied, females are most attractive to males (independent of estrus swelling) when they have already had a few kids which might be another marker of parental competence.

In many species and in humans, individuals tend to be attracted to others who are "just like me": homogamy (mating with someone who is homogeneous or similar in traits). In the US, greater than 90% chance that couples share the same religion, are within three years of each other, have the same ethnicity, race, the socioeconomic status (SES) of their childhood, political views; greater than 40% concordance for having similar IQ, level of education; 20-40% concordance having the same percentile for height for their sex, weight, hair color, lung capacity, width of nostrils or eyes. This is probably driven by kin selection: optimal fertility is roughly 3rd or 4th cousin. Homogeneity signals that we might be distant relatives. Studies of hunter-gatherers shows they tend to mate with people who grew up less than 40 kilometers away. In traditional agricultural societies, mates tend to grow up less than 10 kilometers away. Homogamy is widespread. In a study of 200 years of Icelandic marriages, optimal survival of offspring are for 3rd & 4th cousin marriages. In the US, younger people are more likely to make less homogamous mate choices. Perhaps as we get older we become more closed-minded? For religion, there is a secondary increase in heterogamy between 50-60. Could that be due to lack of choices, because children are now not a factor, a midlife crisis, or they waited for their parents to die?

David Buss published a wide-ranging cross-cultural study which found that in all cultures women want mates who are older than them, men looking for women younger than them, women citing a man's economic prowess as important, men having a preference for markers for fertility, both sexes rated as their highest preference for someone who is nice to them.

Aggression

Sapolsky's soccer aggressiveness made him feel wonderful.

Aggression is about knowing when to be aggressive: finding the appropriate social context. We love violence, we get excited by it, and will pay good money to see it, and we'll even gladly join in when it is the right kind of violence. The same behavior can get you all kinds of awards and "differential reproductive success" and the exact same actions with your muscles could be among the worst things that one human could do to another. Context is critical.

What about violence and aggression is unique to humans?

Sarah Hrdy found competitive infanticide in langur monkeys upending the dogma that no other species besides humans kills its own. By now there are 20-25 species that have competitive infanticide. Humans are not the only species that kills its own kind. Jane Goodall with Chimps showed that humans are not the only ones who kill in a premeditated, strategic, Machiavelian kind

of a way: females killing each other's babies, males killing males. Now we know that Chimps also make weapons for killing. Chimps are female exogamous, so the males in a group are related. Goodall observed "border patrols": the males of a group buildup a high level of excitation and then patrol the boundary of their territory and kill any males from other groups they encounter. She also documented examples of the males in one group systematically killing all of the males of a neighboring group: genocide (killing all individuals based on what group they belong to).

Reconciliation (increased likelihood of affiliative behavior after an aggresive encounter) was once thought to be uniquely human, but there are now reports of reconciliative behavior in a couple of dozen other species. This was first reported by Frans de Waal in Rhesus monkeys. It has been noted in dolphins, whales, and gorillas. Marina Cords showed the odds of reconciliation increase when it is a more important relationship: macaques show more reconciliation if they have a history of cooperating to get food. Instead of the game theory framing, she prefers to frame it as supporting a more valuable relationship. In baboons, females will reconcile but not males. Bonobos have aggression, but it is usually followed by reconciliative behavior afterwards.

Humans are not the only species with a sense of justice. Franz de Waal showed that chimps that establish a cooperative relationship for getting food, will share the food even if only one of them controls the food gathered by the cooperative effort.

Empathy is not unique to humans. In chimps, Frans de Waal found unjustified pommeling by high ranking males elicits more grooming by females in the troupe (but not if the low-ranking guy was provocative). A group from McGill in a study in Science showed that rats hearing ultrasonic alarm calls of their cagemates are more sensitive to pain than agitated rats from out of the group. Humans are the only species that can be moved by suffering on the other side of the planet or artworks or fictional characters in books or movies or even by neglected "child" lamps. Humans have "truth and reconciliation" commissions in South Africa, The Balkans, and Rwanda, sometimes there is even forgiveness. "I will let no man spoil my soul by causing me to hate him": an unprecedented psychology. Helen Prejean, a Catholic nun who ministers to the needs of death row inmates in maximum security prisons whose biography "Dead Man Walking" has been made into a feature film, opera, and a play, says "the less forgiveable the act, the more it must be forgiven; the less lovable the person, the more they must be loved": a distinctly human take on empathy with a logic no one in the animal world could understand.

Humans are less territorial and have less distinct dominance hierarchies. Top/down (despotic) hierarchies have a single aggressive individual who enforces unequal distribution of resources based on violence or threats of violence (baboons, chimps, rhesus monkeys). Bottom/up (egalitarian) hierarchy the top individual is only there via the cooperation of everyone else in the troupe (vervet monkey).

Patas monkey ethology finds almost no male-male violence, but put two males in the same cage and they will fight to the death. In the wild the social structure keeps males as far apart as possible: social cues prevent aggression from happening. Are they a violent species or not? Animal "violence" can also mean getting dinner (predation). Is it violent to eat meat? Or to kill your dinner? What constitutes violent behavior?

Rough and tumble play is ubiquitous in the animal world. Play is one of the last behaviors that goes away during famine: it is deeply hardwired. Primate studies suggest that aggressive play is not for practice, it is establishing the dominance hierarchy that will emerge later.

Unique human forms of aggression: although we can be as violent as a chimp cudgeling one another, we can be violent by doing nothing more taxing than pulling a trigger, looking the other

way, releasing a bomb from 30,000 feet, passive aggressive, damning with faint praise. Examples. Two young kids have a tussle and one breaks the other's easter egg. Teacher says paint her another, so she paints a black one "here's your stupid egg". The other bursts into tears upon seeing the egg. Cooperating with the letter of the law while doing as much violence as possible to the spirit of the law. Sapolsky's wife got cutoff in traffic and responded "I'm going after this guy" and when he is trapped at a red light goes to his open window and say "anybody who could do something like that needs one of these" and flings a lollypop at him. At Nellis Air Force Base just outside Las Vegas in an air conditioned room people control drones killing people 12,000 miles away and then go home to their wife and kids: another day at the office.

Humans are the only species that can psychopathologically confuse sexual behavior with aggressive behavior (sadism & masochism): that appears to be a unique trait.

Humans are not the only species with empathy, but we have it in unique realms.

Human empathy and aggression are very complicated behaviors.

As with sexual behavior, the limbic system is important for understanding the biology of empathy and aggression.

The amygdala with its role in fear and anxiety is important in understanding aggressive behavior. Sapolsky highlights the connection between fear and aggression in the amygdala. Animal studies show the amygdala is involved in aggression (destroy it and no more aggression). In the 1960s & 70s there were thousands of court ordered amygdalectomies (surgical lesion to destroy the amygdala on both sides of the brain) to decrease aggression (it worked, but "there would not be a whole lot of a person left afterward"). In rats, stimulation experiments produce aggressive behavior. A rare form of epilepsy where the focus of the seizure is in the amygdala (many epileptic seizures are preceded by stimulation in the region of focus: smell, sound, math equation, etc.) has seizures preceded by a sudden furiousness. So uncontrolled stimulation in the amygdala results in aggression.

Charles Whitman in 1966 climbed the famed clock tower at the University of Texas at Austin and shot 32 people before killing himself. He had a tumor in his amygdala. Brain scan studies: show something to make someone angry and note that the matabolic rate in the amygdala increases. The amygdala is larger in people with post traumatic stress disorder. People with amygdaloid lesions have difficulty detecting faces with angry emotions, they are more trusting of others, and more likely to forgive. Antonio Demasio eye tracking study: people with amygdaloid lesions make less eye contact. The amygdala directs you to look for potential fears in the world. Testosterone in males makes them look harder for scary things and lowers the threshold for response.

(May 10, 2010) Robert Sapolsky completes his talk on sexual behavior in humans as well as other species, focusing on characteristics that create attractivene...

CJ Fearnley

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Human Sexual Behavior III & Aggression I. 100m Robert Sapolsky video.
Part 3 of 3 in a broad biological survey on the nature of human sexual behavior focuses on attractiveness. The second half of the video introduces part 1 of 4 in a broad biological survey of empathy and aggression. Extensive notes below.
http://www.youtube.com/watch?v=JPYmarGO5jM
Evolutionary theory suggests that the caloric expenditure of sperm (cheap) vs. eggs (expensive) implies more promiscuity on the part of males.
Female-female competition. In some pair-bonding species (such as new world monkeys and many bird species) females compete for the better "parental" males and so they are often more aggressive, have larger body size, and more prominent secondary sexual characteristics (those traits that appear in puberty).
5-20% of the population is homosexual in every human culture ever looked at. The evidence for a genetic basis for homosexuality is not established, so there may not be any role for natural selection. There are three theories for the selective advantage or neutrality of homosexuality. Hetereozygotic vigor argument (like sickle cell anemia where the homozygotic version is a disease but the heterozygotic variation protects against malaria): so it could be that homosexuality is the non-selective form but in heterozygotes it is adaptive (very little evidence). Gender-dependent argument: in one gender the genetically influenced trait is adaptive while in the other gender the trait is maladaptive and leads to homosexuality (evidence: the sisters of gay men tend to be more reproductively successful). Helper at the nest kin selection model: the individuals who are not passing on copies of their genes are helping their siblings (so both sisters & brothers would have higher reproductive success and studies seem to mostly support this model).
Traits that are attractive.
Facial symmetry. Francis Galton developed composite photography which he used to determine the face of crime. Sapolsky belittles the technique and asserts that such composites average to a more attractive face (corroboration: http://faceresearch.org/students/averageness). The symmetry seems to be the quality that makes such composites attractive. The literature suggests that symmetry is a good indicator of health. babies at two months already prefer more symmetrical faces (rats & nonhuman primates also show a preference for symmetry). A study in Nature showed that people with more symmetrical faces were better dancers (this study was retracted by Nature in Dec 2013: http://www.nature.com/.../v438/n7071/full/nature04344.html). The faces of women when ovulating are slightly more symmetrical (http://www.nature.com/.../2004/040331/full/news040329-6.html).
Secondary sexual characteristics. Zahavi's handicap principle: the bigger more garish secondary sexual characteristics is a signal to the world of health, good immunity, or fertility (a signal that one has so much energy to conspicuously consume resources to build this garish trait to attract a mate). Sapolsky claims there is lots of evidence for the principle. Marsupial mice: those individuals with the more exaggerated secondary sexual characteristic had more fertile sperm. Immune system molecules may create the more showy trait. A study of women from many industrialized societies correlates inversely between a preference for men with big jutting jaws, high forehead, and musculature and life expectancy and economic health of that culture. Organisms do not want disease, so they want evidence of a strong immune system. They are good at detecting the smell of parasitic or other infections and avoiding those individuals. Some individuals may cheat: to uncouple their health from their secondary sexual characteristics (some bird species will put in extra energy to enhance their traits when they are sick: reproduce now or never?). Of course, counterstrategies and another coevolutionary back and forth is seen.

A problem with the handicap principle: "there is a certain way to frame things so that you never ever can get a finding that disagrees with your general stance". Vultures have sexually dimorphic faces. Male vultures faces are orange due to carotenoids from consuming ungulate feces with parasites. So dramatic secondary sexual characteristics can both be a marker of health or healthy enough despite some disease. The handicap principle remains controversial. Lions are a tournament species with strong sexual dimorphism (manes): a black mane would be most attractive based on the handicap principle, but it would heat your head in the African sun. So there are complex balancing issues.

Some secondary sexual characteristics are markers of fertility, such as, external swellings in ovulating females (estrus). Baboon males prefer females with larger swellings. Swellings are a marker of estrogen levels, greater fertility, and greater health. The biggest swellings may incur an extra 25 pounds of water weight and yet I can still run around the jungle as a marker of fitness and a source of attractiveness. Humans are concealed ovulators. Human females have no swellings, but a larger hip to waist ratio implies more fertility (child-bearing pelvises, development health augurs well for better fertility). Human males across cultures find larger hip to waist ratios more attractive. This could be a Western bias: the minimal literature on hip to waist ratio among non-Western cultures at first contact suggest that the attractiveness of the trait may be less strong. It is a widespread trait in almost every culture studied. Human females find big jutting jaws, high forehead, and musculature more attractive and these are markers for high testosterone levels during adolescence. Surprisingly, women rate rounder faces as more likable, more honest, more trustworthy, but less desirable! There are subtle differences that people are only subliminally aware of. During ovulation, women's attractiveness for these male secondary sexual characteristics increases (as does pheromone sensitivity).

Confounds: female birds who mate with more attractive males, invest more effort in their kids who have bigger egg size, and therefore they do survive better. So is this a self-fulfilling prophesy or is it the handicap principle? Lee Dugatkin at University of Louisville (I could not find this study, could be a spurious citation): in situations where the female rejects the male, he makes the male appear to be very popular (stuffed females around the rejected male) then the female who spurned him is more likely to initiate proceptive behavior. This bandwagon effect has been shown in many species, but it may be a social contagion not a sign of fitness nor the handicap principle.

In many pair-bonded species attractiveness is shown by demonstrating parental competence (bringing worms) not markers of health or fertility. In most non-human primate species studied, females are most attractive to males (independent of estrus swelling) when they have already had a few kids which might be another marker of parental competence.

In many species and in humans, individuals tend to be attracted to others who are "just like me": homogamy (mating with someone who is homogeneous or similar in traits). In the US, greater than 90% chance that couples share the same religion, are within three years of each other, have the same ethnicity, race, the socioeconomic status (SES) of their childhood, political views; greater than 40% concordance for having similar IQ, level of education; 20-40% concordance having the same percentile for height for their sex, weight, hair color, lung capacity, width of nostrils or eyes. This is probably driven by kin selection: optimal fertility is roughly 3rd or 4th cousin. Homogeneity signals that we might be distant relatives. Studies of hunter-gatherers shows they tend to mate with people who grew up less than 40 kilometers away. In traditional agricultural societies, mates tend to grow up less than 10 kilometers away. Homogamy is widespread. In a study of 200 years of Icelandic marriages, optimal survival of offspring are for 3rd & 4th cousin marriages. In the US, younger people are more likely to make less homogamous mate choices. Perhaps as we get older we become

more closed-minded? For religion, there is a secondary increase in heterogamy between 50-60. Could that be due to lack of choices, because children are now not a factor, a midlife crisis, or they waited for their parents to die?
David Buss published a wide-ranging cross-cultural study which found that in all cultures women want mates who are older than them, men looking for women younger than them, women citing a man's economic prowess as important, men having a preference for markers for fertility, both sexes rated as their highest preference for someone who is nice to them.
Aggression
Sapolsky's soccer aggressiveness made him feel wonderful.
Aggression is about knowing when to be aggressive: finding the appropriate social context. We love violence, we get excited by it, and will pay good money to see it, and we'll even gladly join in when it is the right kind of violence. The same behavior can get you all kinds of awards and "differential reproductive success" and the exact same actions with your muscles could be among the worst things that one human could do to another. Context is critical.
What about violence and aggression is unique to humans?
Sarah Hrdy found competitive infanticide in langur monkeys upending the dogma that no other species besides humans kills its own. By now there are 20-25 species that have competitive infanticide. Humans are not the only species that kills its own kind. Jane Goodall with Chimps showed that humans are not the only ones who kill in a premeditated, strategic, Machiavelian kind of a way: females killing each other's babies, males killing males. Now we know that Chimps also make weapons for killing. Chimps are female exogamous, so the males in a group are related. Goodall observed "border patrols": the males of a group buildup a high level of excitation and then patrol the boundary of their territory and kill any males from other groups they encounter. She also documented examples of the males in one group systematically killing all of the males of a neighboring group: genocide (killing all individuals based on what group they belong to).
Reconciliation (increased likelihood of affiliative behavior after an aggresive encounter) was once thought to be uniquely human, but there are now reports of reconciliative behavior in a couple of dozen other species. This was first reported by Frans de Waal in Rhesus monkeys. It has been noted in dolphins, whales, and gorillas. Marina Cords showed the odds of reconciliation increase when it is a more important relationship: macaques show more reconciliation if they have a history of cooperating to get food. Instead of the game theory framing, she prefers to frame it as supporting a more valuable relationship. In baboons, females will reconcile but not males. Bonobos have aggression, but it is usually followed by reconciliative behavior afterwards.
Humans are not the only species with a sense of justice. Franz de Waal showed that chimps that establish a cooperative relationship for getting food, will share the food even if only one of them controls the food gathered by the cooperative effort.
Empathy is not unique to humans. In chimps, Frans de Waal found unjustified pommeling by high ranking males elicits more grooming by females in the troupe (but not if the low-ranking guy was provocative). A group from McGill in a study in Science showed that rats hearing ultrasonic alarm calls of their cagemates are more sensitive to pain than agitated rats from out of the group. Humans are the only species that can be moved by suffering on the other side of the planet or artworks or fictional characters in books or movies or even by neglected "child" lamps. Humans have "truth and reconciliation" commissions in South Africa, The Balkans, and Rwanda, sometimes there is even forgiveness. "I will let no man spoil my soul by causing me to hate him": an unprecedented psychology. Helen Prejean, a Catholic nun who ministers to the needs of death row inmates in maximum security prisons whose biography "Dead Man Walking" has been made into a feature

film, opera, and a play, says "the less forgivable the act, the more it must be forgiven; the less lovable the person, the more they must be loved": a distinctly human take on empathy with a logic no one in the animal world could understand.
Humans are less territorial and have less distinct dominance hierarchies. Top/down (despotic) hierarchies have a single aggressive individual who enforces unequal distribution of resources based on violence or threats of violence (baboons, chimps, rhesus monkeys). Bottom/up (egalitarian) hierarchy the top individual is only there via the cooperation of everyone else in the troupe (vervet monkey).
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Rough and tumble play is ubiquitous in the animal world. Play is one of the last behaviors that goes away during famine: it is deeply hardwired. Primate studies suggest that aggressive play is not for practice, it is establishing the dominance hierarchy that will emerge later.
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The Biology of Morality: the roots of human aggression & empathy

Details

Due to strong interest in this topic, "The Biology of Morality: the roots of human aggression & empathy" will be repeated on Saturday 10 May (follow this link to RSVP) (https://www.meetup.com/thinkingsociety/events/179689272). Since the Saturday version is easier to get into, please RSVP to it if your schedule permits.

What can biology tell us about human aggression, empathy, and our moral behavior? Robert Sapolsky discusses these issues and more in a broad biological survey of human aggression that starts with an ethologists attention to understanding the context of the behavior itself and then examines the full timeline of biological factors that lead to the behavior from the most proximal (closest) to the most distal (furthest) factors: neurology, releasing stimuli, acute and chronic hormonal situation, cultural factors, perinatal biology and developmental environment, genetics, and the evolutionary and environmental influences.

This discussion is based on 3½ Robert Sapolsky videos in almost 6 hours of lectures. Watching the videos and reading my notes are optional, but the material is so fascinating that I invite you to delve into it and explore it more deeply noting any questions that occur to you.

Aggression I (http://www.youtube.com/watch?v=JPYmarGO5jM) (last 45 minutes of a 1h 35m video). In the second half of this video Sapolsky begins the discussion on aggression with some stories. Humans value aggression when it is in the right context. He then explores how human aggression and empathy are (and are not) unique in the animal kingdom. At the end of the video he starts an in depth discussion of the neurology of aggression with a focus on the amygdala. Read my extensive notes summarizing Sapolsky's discussion. (https://www.facebook.com/cj.fearnley/posts/10202852668653387) Read the notes of a Sapolsky fan (http://robertsapolskyrocks.weebly.com/aggression.html).

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Aggression IV (http://www.youtube.com/watch?v=BqP4_4kr7-0) (1h 42m video). This video looks briefly at hormonal and genetic effects on aggression. The majority of the video explores the cultural, ecological, environmental, and evolutionary effects of aggression and cooperation. The ending is oh so poignant! Read my extensive notes summarizing Sapolsky's discussion. (https://www.facebook.com/cj.fearnley/posts/10203239235757323) Read the notes of a Sapolsky fan. (http://robertsapolskyrocks.weebly.com/aggression-iv.html)

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I have led several previous discussions on Robert Sapolsky videos. Here is a collection of links to those events for your reference. The Uniqueness and Evolution of Humans (15 Apr 2012) (https://www.meetup.com/thinkingsociety/events/50061542/) is based on a commencement speech Sapolsky delivered. The other discussions have been based on Sapolsky's course BIO 250, HUMBIO 160: Human Behavioral Biology (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D). There were two discussions on "The Evolutionary and Genetic Bases of Human Behavior (https://www.meetup.com/thinkingsociety/events/116303742)" which covered videos 2-7 of the course on 14 Jul 2013 (https://www.meetup.com/thinkingsociety/events/116303742) and 27 Jul 2013 (https://www.meetup.com/thinkingsociety/events/128381212), two discussions on "The Biology of Learning" which covered videos 8 & 9 of the course on 10 Nov 2013 (https://www.meetup.com/thinkingsociety/events/144382142/) and 30 Nov 2013 (https://www.meetup.com/thinkingsociety/events/148844402/). There were three discussions on "Brain Science and Human Behavior (https://www.meetup.com/thinkingsociety/events/153389892)" which covered videos 10-14 of the course on 12 Jan 2014 (https://www.meetup.com/thinkingsociety/events/153389892/), 18 Jan 2014 (https://www.meetup.com/thinkingsociety/events/158107662/), and 2 Feb 2014 (https://www.meetup.com/thinkingsociety/events/161011212/). There were two discussions on "The Biology of Human Sexual Behavior (https://www.meetup.com/thinkingsociety/events/153541762/)" which covered videos 15-17 of the course on 9 March 2014 (https://www.meetup.com/thinkingsociety/events/153541762/) and 15 March 2014 (https://www.meetup.com/thinkingsociety/events/168419202/).

Comments

700
James F.

4,080 days ago

It was too much ambient noise throughout the time I stayed for hearing other members.

CJ F.

4,080 days ago

Thanks for the feedback. Our usual table in the back was taken and we decided to use the big long table. In hindsight that was a mistake. If we had used four round tables we could have made a circle and halved the distance between the two ends of the table. I won't make that mistake again.

Jyoti M.

4,080 days ago

CJ, I am going to take full responsibility for this one because I dissuaded you from joining the round tables for better group dynamics as I thought long table would serve the purpose. Between the noisy ambience and large distance from the facilitator , some of our members could not get the contents of the discussion in entirety. My sincere apologies to Mark, Martin , Tee and Vanessa who were seated at the end. Will not happen again!

James F.

4,080 days ago

Maybe a good tactic would be if one of us got down there real early and put some tables together 'in reserve' in the alcove area.

James F.

4,080 days ago

That would be a worthy morning exercise for me! Jim

CJ F.

4,080 days ago

I arrived 15 minutes early, but the usual table in the back was taken. We like that table because the sound is somewhat blocked by the alcove and curtains. Plan B should be to organize the round tables into a circle. The big table is just too long for people on one side to hear those at the other side. But I didn't realize that until afterward, unfortunately.

David S.

4,082 days ago

Really great discussion. All 13 people contributed extensively, but we were not aggeressive against each other. Very interesting behavior experments were discussed. The moderator was well prepared from the video, and led the group to the next video topic at appropiate times.

CJ F.

4,083 days ago

Tomorrow we will discuss the biological roots of morality. The 4th & final optional video by Robert Sapolsky is "Aggression IV": http://y2u.be/BqP4_4kr7-0

Pseudospeciation: psychological mechanisms to make others seem so different from us that you don't even view them as human: "us v. them". Do Hitler & bin Laden deserve the same moral considerations as other human beings?

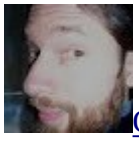
The theme of metaphor and symbol from the 2nd & 3rd videos continue as Sapolsky explores peacemaking.

Profound treatment of cooperation: there are many tools to increase cooperative behavior, but cooperation is also a prerequisite to genocide! The scariest thing on the planet is when a group of males starts to cooperate and look at others around them.

The conclusion is poignant & profound: the exact same behaviors are cheered & are "the most frightening possible thing that can happen to us". Aggressive behavior is complex!

Read my notes: https://t.co/UnCcrqBtJX

Watch "Aggression IV": http://y2u.be/BqP4_4kr7-0



CJ Fearnley - 2014-04-22 07:32:19-0400 - Updated: 2014-04-22 07:32:19-0400
Join me on Sun May 4th to discuss "The Biology of Morality: the roots of human aggression & empathy" based on Robert Sapolsky videos. What can biology tell us about human aggression, empathy, and our moral behavior?

http://www.meetup.com/thinkingsociety/events/176276012/

The Biology of Morality: the roots of human aggression & empathy

The Biology of Morality: the roots of human aggression & empathy (Repeat)

Details

What can biology tell us about human aggression, empathy, and our moral behavior? Robert Sapolsky discusses these issues and more in a broad biological survey of human aggression that starts with an ethologists attention to understanding the context of the behavior itself and then examines the full timeline of biological factors that lead to the behavior from the most proximal (closest) to the most distal (furthest) factors: neurology, releasing stimuli, acute and chronic hormonal situation, cultural factors, perinatal biology and developmental environment, genetics, and the evolutionary and environmental influences.

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http://www.youtube.com/watch?v=BqP4_4kr7-0

This topic is a repeat of the one on Sunday May 4th. (https://www.meetup.com/thinkingsociety/events/176276012/)

I have led several previous discussions on Robert Sapolsky videos. Here is a collection of links to those events for your reference. The Uniqueness and Evolution of Humans (15 Apr 2012) (https://www.meetup.com/thinkingsociety/events/50061542/) is based on a commencement speech Sapolsky delivered. The other discussions have been based on Sapolsky's course BIO 250, HUMBIO 160: Human Behavioral Biology (http://www.youtube.com/view_play_list?p=848F2368C90DDC3D). There were two discussions on "The Evolutionary and Genetic Bases of Human Behavior (https://www.meetup.com/thinkingsociety/events/116303742)" which covered videos 2-7 of the course on 14 Jul 2013 (https://www.meetup.com/thinkingsociety/events/116303742) and 27 Jul 2013 (https://www.meetup.com/thinkingsociety/events/128381212), two discussions on "The Biology of Learning" which covered videos 8 & 9 of the course on 10 Nov 2013 (https://www.meetup.com/thinkingsociety/events/144382142/) and 30 Nov 2013 (https://www.meetup.com/thinkingsociety/events/148844402/). There were three discussions on "Brain Science and Human Behavior (https://www.meetup.com/thinkingsociety/events/153389892)" which covered videos 10-14 of the course on 12 Jan 2014 (https://www.meetup.com/thinkingsociety/events/153389892/), 18 Jan 2014 (https://www.meetup.com/thinkingsociety/events/158107662/), and 2 Feb 2014 (https://www.meetup.com/thinkingsociety/events/161011212/). There were two discussions on "The Biology of Human Sexual Behavior (https://www.meetup.com/thinkingsociety/events/153541762/)" which covered videos 15-17 of the course on 9 March 2014 (https://www.meetup.com/thinkingsociety/events/153541762/) and 15 March 2014 (https://www.meetup.com/thinkingsociety/events/168419202/).

Events in Philadelphia, PA

Nadine

4,076 days ago

Thanks CJ for your work and preparation. I echo what others said. A thought provoking, lively discussion with excellent contributions from everyone. I enjoyed meeting everyone.

Dr. Janice P.

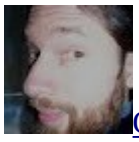
4,076 days ago

Excellent topic, wll moderated. Thanks, CJ and all for time well spent!

Will B.

4,076 days ago

Great discussion. Very lively, informed and stimulating. Everyone contributed and had something interesting to offer. C.J. did a fine job of keeping the discussion balanced and shared by all as well as bringing up questions raised by Sapolsky in the videos.

 CJ Fearnley - 2014-03-27 23:41:13-0400 - Updated: 2014-03-27 23:41:13-0400

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18. Aggression II

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amygdala = fear = anxiety = aggression

Exceptions: Williams syndrome is a poorly understood imprinted genetic disorder resulting in kids who are unbelievably facile with language and emotional expressivity yet borderline retarded

cognitively. Very affectionate, trustful, gregarious and so incredibly vulnerable to being taken advantage of. They do not react to scary faces in their amygdala. People with social phobias have the amygdala activate for any face: for them every face is a scary face. In depressed people the amygdala activates when you show them something sad suggesting that the amygdala is subtle and contextual in its functioning.

The amygdala is probably the best part of the brain for dichotomizing between us or them and responding to outgroup stimuli. The amygdala forms these dichotomies very quickly.

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Frontal cortex is involved in regulating appropriate behavior in the context of violence, aggression, competition, & cooperation. It was Walle Nauta who suggested that the wiring of the prefrontal cortex makes it part of the limbic system (the so-called emotional brain). When the choice is between something harder (yet better for you) and something easier, the frontal cortex is about doing the harder thing. The frontal cortex has many weak, diffuse projections throughout the limbic system to give more biasing to excite those pathways that are "harder". The frontal cortex provides a modulatory function. Dopamine fuels expectation and drives the frontal cortex to goal-directed behavior.

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Another case of a child with frontal cortical damage from forceps slipping at birth. Frontally disinhibited: unconstrained by society or its laws. But in this case its an upper middle class family with excellent family support, but he plays the piano way longer that is socially acceptable. So environmental differences can result in the damage manifesting in different ways.

So some states protect people (M'Naghten rules) with 99% of their frontal cortex destroyed. What about 97% or 85% or 45% damage? Or you and me who have 5 more or fewer synapses than the other in our frontal cortexes?

Epileptic seizures in the 16th century were medically explained as demonic possession punished by burning at the stake which was proven by observing if they cried when read the story of the crucifixion. Someone at the time observed that in some elderly women the lacrimal gland can atrophy making them unable to cry.

During REM sleep, the frontal cortex is almost entirely inactive which is why dreams make no sense.

The frontal cortex is the last part of the brain to be fully myelinated (the white sheath that speeds neuronal communication) and to develop its branching synapses. It is finally fully developed about age 25 hence it is the part of the brain most sculpted by environment and experience and so least constrained by genes. "It is the most definably human part of the brain."

The dopamine-driven metabolic changes (rewards and disappointments triggering rises and falls in dopamine) swing much more widely in teenagers than in adults. The teenager's brain is less regulated than the adult brain.

Supreme court decided that since the brain and its regulatory functions are not fully mature, the death penalty should not apply to kids under 18. But brain science offers no reason to assume that one's 18th birthday resolves any of the frontal cortex deficiencies of one's late teens.

The frontal cortex is the third most vulnerable brain region to aging. The substantia nigra a motor system in the brain which loses most of its neurons with aging which causes the tremors of old age and Parkinson's disease. The hippocampus loses most of its neurons with age which affects memory in the elderly. The degradation in the frontal cortex with aging explains disinhibited comments from the elderly.

People with repressive personalities (highly regimented, disciplined, controlled behavior, weak at expressing and reading emotions in others, not depressed nor anxious) have elevated resting metabolism in the frontal cortex. Sociopaths have low activation (low metabolism rates) of their frontal cortex; when they recite the months backward (requiring frontal cortex function), it takes them more work to do it than for those whose frontal cortex is generally more active.

By age five, the size and resting metabolic rate of the frontal cortex shows the substantial impact of socioeconomic status (poverty) on glucocorticoid levels (from the stress of poverty; the frontal cortex has a lot of glucocorticoid receptors which atrophy neurons with elevated levels) which inhibits neuronal growth.

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In the anterior cingulate the brain both senses pain and empathically senses the pain of another. Another metaphor / reality mixing.

Insular cortex activates when foul odors are sensed in many species. In humans, tell someone a story about someone being mistreated by someone powerful or being exploited (such as "stab you in the back" in a game) and the insular cortex activates. Moral disgust and culinary disgust co-occur in the same brain region. Every human culture has language referring to moral failures with words denoting gustatory repellent stimuli: "I'm disgusted by what you did", "it makes me nauseous", "something about this smells rotten". In another study, if people are asked to talk about moral failings, they are more likely to choose soap as a reward than other equally valuable commodities. We shoehorned our sense of moral disgust into the part of the brain that handles bad food intermixing metaphor and reality.

Jonathan Haight shows how much of human decision-making is affective: your limbic system decides before you do. The cognitive system struggles to explain what the limbic system has decided for you.

When someone does something aggressive, the sympathetic nervous system activates. You cannot tell the difference between murder and orgasm when looking at the basic brain systems. There is a

lot of non-specific activation in arousal. Love and hate are physiologically very, very similar: arousal of the sympathetic nervous system. Elie Wiesel: "The opposite of love is not hate, it's indifference". Many humans can psychopathologically confuse love and hate.

Hormones of Aggression

Why are males generally more aggressive than females? Is testosterone the cause? Testosterone plays the same role in aggression as it does in human sexual behavior. Testosterone is required for the full expression of aggressive behavior, but even after castration aggressive behavior does not go to zero (prior experience also influences the result). Whether adding 10%, 100% or 200% of normal testosterone levels back after castration, aggression returns to normal levels. Testosterone is needed but is neither necessary nor sufficient for aggression. You cannot predict aggressiveness based on testosterone levels. In Rhesus monkeys with a dominance hierarchy 1>2>3>4>5. Pump #3 with testosterone and he becomes more aggressive, but only to #4 and #5. Testosterone exaggerates (modulates or amplifies) the preexisting social structure. Raise testosterone and the amygdala has a lower threshold for activating given a potentially threatening face and at the cellular level it shortens the refractory period after a neuron fires (so that it can fire again more quickly): again, testosterone amplifies the activity.

Spotted hyenas have female dominance where females are larger, more aggressive, and have higher testosterone levels than males. Female hyenas have an enlarged clitoris the size of a male penis and compacted fat cells that look like a scrotum so you cannot tell males from females. An interesting case of a sex reversal: androgenized females. Hyena feeding order is cubs then females then males (in lions it is males then females then cubs). Masculination of the female genitilia. Generally male dominance displays involve waving their erect penis about. In hyenas, males get erections when they are terrified as a subordination gesture (similarly, low ranking females get clitoral erections). Hyena females are hormonally more male than male hyenas to achieve the sex role reversal in their social structure.

CJ Fearnley's post

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Insular cortex activates when foul odors are sensed in many species. In humans, tell someone a story about someone being mistreated by someone powerful or being exploited (such as "stab you in the back" in a game) and the insular cortex activates. Moral disgust and culinary disgust co-occur in the same brain region. Every human culture has language referring to moral failures with words denoting gustatory repellent stimuli: "I'm disgusted by what you did", "it makes me nauseous", "something about this smells rotten". In another study, if people are asked to talk about moral failings, they are more likely to choose soap as a reward than other equally valuable commodities. We shoehorned our sense of moral disgust into the part of the brain that handles bad food intermixing metaphor and reality.

Jonathan Haight shows how much of human decision-making is affective: your limbic system decides before you do. The cognitive system struggles to explain what the limbic system has decided for you.

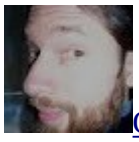
When someone does something aggressive, the sympathetic nervous system activates. You cannot tell the difference between murder and orgasm when looking at the basic brain systems. There is a lot of non-specific activation in arousal. Love and hate are physiologically very, very similar: arousal of the sympathetic nervous system. Elie Wiesel: "The opposite of love is not hate, it's indifference". Many humans can psychopathologically confuse love and hate.

Hormones of Aggression

Why are males generally more aggressive than females? Is testosterone the cause? Testosterone plays the same role in aggression as it does in human sexual behavior. Testosterone is required for the full expression of aggressive behavior, but even after castration aggressive behavior does not go to zero (prior experience also influences the result). Whether adding 10%, 100% or 200% of normal testosterone levels back after castration, aggression returns to normal levels. Testosterone is needed but is neither necessary nor sufficient for aggression. You cannot predict aggressiveness based on testosterone levels. In Rhesus monkeys with a dominance hierarchy 1>2>3>4>5. Pump #3 with testosterone and he becomes more aggressive, but only to #4 and #5. Testosterone exaggerates (modulates or amplifies) the preexisting social structure. Raise testosterone and the amygdala has a lower threshold for activating given a potentially threatening face and at the cellular level it shortens the refractory period after a neuron fires (so that it can fire again more quickly): again, testosterone amplifies the activity.

Spotted hyenas have female dominance where females are larger, more aggressive, and have higher testosterone levels than males. Female hyenas have an enlarged clitoris the size of a male penis and compacted fat cells that look like a scrotum so you cannot tell males from females. An interesting case of a sex reversal: androgenized females. Hyena feeding order is cubs then females then males (in lions it is males then females then cubs). Masculination of the female genitilia. Generally male dominance displays involve waving their erect penis about. In hyenas, males get erections when they are terrified as a subordination gesture (similarly, low ranking females get clitoral erections). Hyena females are hormonally more male than male hyenas to achieve the sex role reversal in their social structure.

youtube.com
18. Aggression II
(May 12, 2010) Robert Sapolsky continues his lectures about aggression in humans but also continues to talk about other emotions and what goes on in the brai...

 CJ Fearnley - 2014-04-23 15:21:47-0400 - Updated: 2014-04-23 15:21:47-0400
Aggression III. 105m Robert Sapolsky video.

Part 3 of 4 in a broad biological survey on empathy and aggression. The discussion drills deeper into the timeline of biological causes starting with the neurology and reviewing how the brain mixes reality and metaphor in its moral wiring. Then a fascinating and deep discussion of the role of serotonin in aggression and impulsive behavior shows how difficult it is to find cause and effect in brain science. Then there is a discussion of the releasing stimuli for aggression followed by the hormonal effects. Perhaps the most interesting part of the lecture covers the environmental influences for moral behavior: bake it into the cerebellum or you'll have to think it through! Aha, this whole series of 4 videos is about "the biology of morality": sweet!

19. Aggression III

Metaphor in the Brain (continued)

Sapolsky continues exploring how the brain mixes reality and metaphor. These effects strongly support Jonathan Haight's findings that moral reasoning is mostly after the fact rationalization for moral affect (the affective decisions of the limbic system). The dictionary (1913 Webster) says "affective" means "pertaining to or exciting emotion" and "affect" means "to influence or move, as the feelings or passions".

Anterior cingulate is a brain region (nucleus) that both senses pain and empathically senses the pain of another. Despite tremendous interest in mirror neurons which get excited if you and someone else are doing the same motor action (but not when doing it yourself or watching others), there is no proof that mirror neurons play a role in the anterior cingulate.

Serotonin is a neurotransmitter which seems to affect aggression and impulsive behavior. In animal studies less serotonin yields more impulsive and aggressive behavior. In humans, one studies the serotonin breakdown products and finds that lower levels of such products correlate with higher levels of aggression and antisocial behavior (caveats: correlation is not causation: cause and effect may be reversed; breakdown products may come from serotonin use in other parts of the brain). By driving down serotonin levels in animal studies, there is some evidence that serotonin causes impulsive and aggressive behavior. In humans giving a drug that mimics serotonin in the short term ("buzzing serotonin pathways") leads to increased metabolism in the frontal cortex except in individuals with a history of antisocial violence. This suggests serotonin supports frontal cortex inhibitions of inappropriate limbic impulses. Sociopaths have lower than normal metabolic rates in the frontal cortex.

Serotonin breakdown biochemistry: tryptophan (TH: tryptophan hydroxylase) -> 5-HTP (5-Hydroxy-L-tryptophan; enzyme TBH is not listed at https://en.wikipedia.org/wiki/Serotonin#Biosynthesis) -> Serotonin (MAO: Monoamine oxidase, COMT) -> 5-HIAA (which can be measured in bloodstream, cerebospinal fluid, or urine). Measuring serotonin levels in this indirect fashion can be problematic because there could be a problem in the breakdown process itself instead of with serotonin. Sapolsky reports that some studies failed to control for these effects and reported results which are completely uninterpretable.

The rate-limiting step is the biochemical reaction that is slower or harder to accomplish than the other steps in a biochemical pathway. TH is the rate-liming step in serotonin metabolism. There are studies showing that variations in TH in humans and primates correlate with aggression (but they are very small statistical effects). There are studies on correlations with the two variations on MAO showing a gene-environment interaction: the "bad" gene doesn't lead to more aggression unless there is also abuse.

Alcohol has lots of messy effects on the brain (no specific effect). There is no significant relationship between alcohol and aggression! Alcohol causes aggressive people to be more aggressive; it causes non-aggressive people to be more inhibited. Alcohol merely magnifies the preexisting social tendency. People who believe their blood alcohol levels have risen tend to become more aggressive: alcohol socially "allows" you to become more aggressive. It has a modulating function. Anthropological studies of Polynesian cultures who were taught drinking after WWII adopted the behavior and attitude toward alcohol of the colonial power teaching them to drink (US, UK: aggression; FR: sexual promiscuity).

Releasing stimuli for aggression

Pseudomyrmex ants respond aggressively to vibrations in their Acacia tree homes as a symbiotic protection service against herbivores. Humans have no auditory, olfactory or other direct cues for aggressive behavior. Although there is amygdal activation with exposure to the sweat of frightened individuals, it is subliminal and does not necessarily lead to aggressive behavior.

Pain is the most reliable stimulus for triggering aggressive behavior. Frustration is another trigger: a rat trained to press a lever will get frustrated when the lever stops working and bite its cagemate: displacement aggression. The stressed and frustrated rat will have high glucocorticoid levels until it bites its cagemate. Displacing aggression on somebody else in species after species is stress reducing. Male baboons occasionally have rape (forced sex with a female who actively attempts to get away and resist): Sapolsky has seen it three times: when an alpha male is displaced from its #1 position. Although such displaced males will usually just mope quietly or beat up on other lower-ranking guys occasionally they displace their frustration on females by rape. Frustration displacement may account for the increase in violence typically associated with poverty and recessions. Frustration, pain, and stress are reliable predictors of aggressive behavior. But it is a modulatory factor: only those predisposed to aggression will vent it when facing pain, frustration and stress.

John B. Calhoun studied overpopulation in rats in the 50s and reported that rats in a smaller enclosure dramatically increased aggression to the point where they started killing and cannibalizing each other. Urban sociologists expected the next generation of large cities to feature cannibalism. Everyone feared the menace of overpopulated inner cities. Eventually more careful studies were conducted which determined that aggression does not increase with overcrowding. Overcrowding modulates aggressive behavior. Calhoun and other early researchers failed to quantitatively measure the aggression and simply reported the dramatic behaviors of the few hyper-aggressive rats dealing with the stress of overcrowding and having more rats around them to vent their frustrations upon.

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Testosterone and other androgens are synthesized in the adrenal gland in females at about 5% male levels. Aggression in females is also modulated by adrenal androgens.

Perimenstral (a little before and after menses) hormones are associated with increased aggression in females. Since female baboons also display perimenstral aggressive behavior, the cause is evidently biological and so Sapolsky dismisses the cultural and psychodynamic explanations. Some

anthropologists have argued that perimenstral effects don't occur in societies that are freer about their bodies and more sexually uninhibited. Sapolsky reports that perimenstral mood shifts are pretty universal in human cultures. But there is some psychology because women told that their period is approaching, will become more irritable (self-fulfilling prophesy). The significant others of perimenstral women also become more irritable. Perimenstral behavior also correlates with depression and social withdrawal. In baboons, low-ranking females withdraw whereas high-ranking ones get irritable. Ethology (interviewing an animal in its own language) plays its profound role again!

The ratios of estrogen and progesterone drop significantly in the perimenstral period. 10% of women have severe perimenstral syndrome correlated with a large drop in progesterone around the time of menses. Progesterone binds to GABA, the main inhibitory neurotransmitter in mammals, at the binding site for minor tranquilizers (which also binds benzodiazepines such as Valium and Librium). An interpretation is that progesterone acts as a mild sedative decreasing anxiety except for that time of the month when its levels are low. In another subset of women with perimenstral syndrome, endogenous opioid levels (specifically beta-endorphin, a neurotransmitter and hormone with "nice effects on mood") decrease significantly around menses.

Glucocorticoids, epinephrine, & norepinephrine rise with fight (aggression) or flight (not aggression): activation of the sympathetic nervous system. The opposite of love is indifference. Give someone pain or physical discomfort and their sympathetic nervous system typically activates as part of stress response. In sociopaths, the response is much reduced (elevated pain thresholds with less sympathetic responsiveness to pain). Since empathy is feeling someone else's pain, this effect may explain some sociopathic behavior.

Environmental triggers of aggression: broad theories: 3 schools

1. Environment is irrelevant: Konrad Lorenz 1966 book "On Aggression" was very influential: aggression is universal (present in all individuals) and has no environmental requirements. Famous quote: "there is no love without hate". Hydraulic model of aggression: aggressive drive builds up until smaller and smaller environmental triggers will release it. If aggression is not released, it will eventually lead to spontaneous aggression. Once aggression is released, the aggressive drive is depleted starting a refractory period. Critiques: aggression is not universal nor inevitable in humans (well, at least not after seventh grade). Aggression is not self-depleting, instead it is self-reinforcing (witness crowd violence at sporting events). Aggression stimulates more aggression: it legitimizes and habituates one to it.

2. Aggression is ultimately all about frustration, pain, stress, fear, and anxiety. Extreme version of the frustration displacement model: since the amygdala is about fear and aggression, all aggression is based on some form of fear or stress. Unemployment, spousal abuse, and child abuse are correlated. In baboons, about 50% of aggression occurs after a fight or someone loses access to a resource. The poor are more subject to stress and so are more violent. Moreover, the aggression is directed at other low-ranking organisms. Negative result: during periods of famine, aggression tends to go down despite the high stress that everyone suffers. So at least in this realm, the extreme frustration displacement theory is off. "Behavioral fat": aggression occurs when there are fewer stresses (lions during zebra migration have the surplus resources to fight for dominance). Clan or retributive violence can be interpreted as competing for the one who does the last retribution.

3. Behaviorists argue that aggressive behavior can be shaped by positive and negative reinforcements. Studies looking at laws and crime rates can test the theory. When the death penalty becomes law, the number of premeditated murders does in fact decrease. However, crimes of passion and impulsive murders are not affected by the death penalty. Generally changing the

severity of punishment does not change the rate of murder.

Early environmental (developmental & upbringing) influences on aggression (and empathy, etc.) is mainly about learning the context for appropriate aggressive, empathic, compassionate, and cooperative behavior. Most of the research on this question is framed by the question: how do moral standards develop in kids? During the first few days or weeks of life babies start distinguishing between animate and inanimate things. The fusiform cortex which responds to faces does not work as well in autistic children who cannot distinguish animate from inanimate. Before the development of a self (distinguishing self from other: an ego boundary) at about one year of age, kids identify themselves with mom: they report an owwwy on their finger if mom has cut her finger.

Kids develop theory of mind (that is, the recognition that others may have different information, thoughts, and feelings than they have) between ages 3 and 5. Kids may have a good faculty for theory of mind when reading a story but when in an emotionally charged situation, they may not be effective in thinking through the differential information, thoughts, and feelings of others.

Is theory of mind a prerequisite for empathy? Is it possible to feel someone else's pain without understanding that there is a someone else who has different thoughts and feelings? The thinking is that yes theory of mind is a prerequisite for empathy. Sociopaths have an effective theory of mind faculty yet little or no empathy. So theory of mind is not a sufficient condition for empathy. But if an adult is crying, a 15 month old will often offer them their pacifier to help them feel better. Do 15 month olds have empathy without theory of mind? Perhaps, but it might also be the child is irritated by the crying and is just trying to stop it (or mimicking the parental behavior of sticking pacifiers in the mouths of upset babies). One experiment tested kids aged 3-6 months with three skits. One skit exhibited a prosocial interaction (someone helping another), another neutral behavior, and the third antisocial behavior (taking something away from someone). The kids prefer to look at the individual doing the prosocial helpful behavior. So some parts of empathy manifest before theory of mind.

Lawrence Kohlberg at Harvard built upon Swiss psychologist Jean Piaget's work of stages of cognitive development (object permanence: if you cover an object is it surprising when it gets uncovered?) to develop Kohlberg's famed stages of moral development in kids: six sequential stages falling in three categories of moral reasoning: 1) pre-conventional (you behave morally because you might get caught and either get punished or lose a reward: ego centered motivations for moral behavior), 2) conventional (social norms: shared group conventions: following the rules or laws), and post-conventional (transcendent: behaving in a way that breaks the rules or laws: there are things more important than what everyone else thinks: civil disobedience, bad laws may deserve to be broken). Studies show that not all adults advance through the stages: many stay in the pre-conventional or conventional stage into adulthood. One study looked at a girl wanting to play ball with some boys. In the pre-conventional stage they argue it is fair since she has not played. In the conventional stage, they argue it will be bad for the team, so no (instead of individual consequences using standards of norms to reason instead).

Criticisms of Kohlberg's model. Like most studies in the 60s, most of them used male subjects. Kohlberg's model fits boys better than girls. Boys are more about justice and girls are more about social affiliation (a gender critique). Moral development in other cultures doesn't seem to fit Kohlberg's model well either. People who are trained in childhood to respect ability more than effort are far more likely to use social norm moral reasoning than social justice reasoning and so are less likely to reach a post-conventional Kohlberg stage. How do kids learn that it is sometimes OK to lie? ("No, grandma, I do not like this sweater." "I already have one of those.") How do kids learn the difference between rules (less breakable: must be followed) and principles (involve judicious weighing). When do we learn about bad laws? How do we learn the difference (consistently see

distinction) between intended harm and successful harm? At earlier stages kids discount harm that doesn't actually happen.

Peer groups & community

Felton Earls at Harvard made very careful studies about how many violent acts kids were exposed to growing up, He found that those growing up in a violent community (witness or victim to violence) led to adults who are more prone to violence (they rationalize and are desensitized to it). The effect is even stronger if the violence happened in the family. Many studies now show that the effects of watching a lot of violent TV or movies or video games modulates behavior: it magnifies the effect only in those who have a pre-existing tendency to violence.

In a study comparing male homicide rates by age in three cities (one Canadian: Toronto: 50; one American: Chicago: 600; and one British: London: 30), Martin Daly and Margo Wilson found that the frequency of murder by age increases rapidly until the early 20s and then starts declining significantly by age 25. It is a maxim in criminology that there is a big decrease in criminal behavior after age 25. So although age is an important predictor, the community you grow up in is the most significant predictor of a subject's likelihood to commit murder.

Harry Harlow raising captive rhesus monkeys in isolation showed that (as with sexual behavior) their fixed action patterns are normal, but they never learned the appropriate social context for the behavior. So they show the normal behaviors of dominance and subordination, but they do not know when and to whom those displays should be given. In baboons, you inherit your rank from your mother. The social training for appropriate social behavior based on hereditary rank begins as early as the first week after birth. Similarly the Berkeley Hills hyenas took longer to establish their dominance hierarchy since they were raised without social context.

How important are parents and how important is peer group? Judith Rich Harris argues in her book "The Nurture Assumption" that peer influences are vastly more important than had been recognized. She observed that in immigrant families by age 4 or 5 the kids are developing the accent of the local community not that of their parents; they are becoming embarrassed by their parents' accent; they answer questions given in the home language using the outside language. We pick up the accent of our peer group, not our parents. "At the end of the day, parents are mostly good for determining which peer groups their kids have access to." Humans are very subject to conformity and conventionalized behavior in groups: once separated into groups we start to have disparaging beliefs about the other group; we overidentify with whatever category or group we are assigned. Within a week of working on the set of Planet of the Apes, actors sat at lunch with people from the same species that they were acting in the movie. All the actor chimps ate together even though their best friends were orangutans. Harris reinterpreted the result that kids growing up in a family without a father had a higher chance of antisocial violence as an adult: such families tend to be poorer and it is the peer socialization which leads to the effect.

Steven Levitt (economist) and John Donohue (lawyer) determined that a significant part of the decrease in the crime rate since the 1970s (now thought to be 50%) is due to the Roe v. Wade decision that legalized abortion. As each state legalized abortion, there would be a 12-15 year lag before the crime rate would drop. At the beginning of the decrease in crime, it would be due to fewer teenagers entering the criminal justice system. As time goes on there are fewer criminals in the age range that grew up with safe, legal abortion. Evidently a huge predictor of growing up to be a violent antisocial individual is being an unwanted child. Abortion reduces the number of unwanted children which significantly reduces the crime rate.

Explicit declarative learning: you learn a fact, you know that you know it, and you can consciously

use it, strategize with it, and use it in an executive way (requires hippocampus to store it)

Implicit procedural learning: your body knows it better than your head (stored in the cerebellum)

Is there a correlation between moral reasoning and moral behavior? The literature does not find a particularly good connection. Those who reach Kohlberg's post-conventional stage of moral reasoning tend not to be more likely to act heroically. The Carnegie Foundation studies those who do brave unexpected moral acts and finds they grow up in an environment with a very strong, consistent, repeated stated imperative to act morally, to act bravely, and to not care what other people think. When interviewed, such people invariably report that they didn't think (for example before running into the burning building or jumping into the freezing river), so it is implicit procedural learning that spurs them to action. Something overlearned in childhood that does not require reasoning skills and effort.

Josh Green determined that players who cheated at least once (in a game designed to make it easy to cheat and easy for the researches to know when they were cheating) had increased activity in their frontal cortex. Those who didn't cheat never had frontal cortex activation: it was an implicit pathway: they were not even tempted. Perhaps their baked in moral rules made the question verboten.

Sapolsky concludes that he thinks a lot of what moral development is about is developing implicit rules by overlearning in childhood.

CJ Fearnley's post

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Aggression III. 105m Robert Sapolsky video.
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Early environmental (developmental & upbringing) influences on aggression (and empathy, etc.) is mainly about learning the context for appropriate aggressive, empathic, compassionate, and cooperative behavior. Most of the research on this question is framed by the question: how do moral standards develop in kids? During the first few days or weeks of life babies start distinguishing between animate and inanimate things. The fusiform cortex which responds to faces does not work

as well in autistic children who cannot distinguish animate from inanimate. Before the development of a self (distinguishing self from other: an ego boundary) at about one year of age, kids identify themselves with mom: they report an owwwy on their finger if mom has cut her finger.
Kids develop theory of mind (that is, the recognition that others may have different information, thoughts, and feelings than they have) between ages 3 and 5. Kids may have a good faculty for theory of mind when reading a story but when in an emotionally charged situation, they may not be effective in thinking through the differential information, thoughts, and feelings of others.
Is theory of mind a prerequisite for empathy? Is it possible to feel someone else's pain without understanding that there is a someone else who has different thoughts and feelings? The thinking is that yes theory of mind is a prerequisite for empathy. Sociopaths have an effective theory of mind faculty yet little or no empathy. So theory of mind is not a sufficient condition for empathy. But if an adult is crying, a 15 month old will often offer them their pacifier to help them feel better. Do 15 month olds have empathy without theory of mind? Perhaps, but it might also be the child is irritated by the crying and is just trying to stop it (or mimicking the parental behavior of sticking pacifiers in the mouths of upset babies). One experiment tested kids aged 3-6 months with three skits. One skit exhibited a prosocial interaction (someone helping another), another neutral behavior, and the third antisocial behavior (taking something away from someone). The kids prefer to look at the individual doing the prosocial helpful behavior. So some parts of empathy manifest before theory of mind.
Lawrence Kohlberg at Harvard built upon Swiss psychologist Jean Piaget's work of stages of cognitive development (object permanence: if you cover an object is it surprising when it gets uncovered?) to develop Kohlberg's famed stages of moral development in kids: six sequential stages falling in three categories of moral reasoning: 1) pre-conventional (you behave morally because you might get caught and either get punished or lose a reward: ego centered motivations for moral behavior), 2) conventional (social norms: shared group conventions: following the rules or laws), and post-conventional (transcendent: behaving in a way that breaks the rules or laws: there are things more important than what everyone else thinks: civil disobedience, bad laws may deserve to be broken). Studies show that not all adults advance through the stages: many stay in the pre-conventional or conventional stage into adulthood. One study looked at a girl wanting to play ball with some boys. In the pre-conventional stage they argue it is fair since she has not played. In the conventional stage, they argue it will be bad for the team, so no (instead of individual consequences using standards of norms to reason instead).
Criticisms of Kohlberg's model. Like most studies in the 60s, most of them used male subjects. Kohlberg's model fits boys better than girls. Boys are more about justice and girls are more about social affiliation (a gender critique). Moral development in other cultures doesn't seem to fit Kohlberg's model well either. People who are trained in childhood to respect ability more than effort are far more likely to use social norm moral reasoning than social justice reasoning and so are less likely to reach a post-conventional Kohlberg stage. How do kids learn that it is sometimes OK to lie? ("No, grandma, I do not like this sweater." "I already have one of those.") How do kids learn the difference between rules (less breakable: must be followed) and principles (involve judicious weighing). When do we learn about bad laws? How do we learn the difference (consistently see distinction) between intended harm and successful harm? At earlier stages kids discount harm that doesn't actually happen.
Peer groups & community
Felton Earls at Harvard made very careful studies about how many violent acts kids were exposed to growing up, He found that those growing up in a violent community (witness or victim to

violence) led to adults who are more prone to violence (they rationalize and are desensitized to it). The effect is even stronger if the violence happened in the family. Many studies now show that the effects of watching a lot of violent TV or movies or video games modulates behavior: it magnifies the effect only in those who have a pre-existing tendency to violence.

In a study comparing male homicide rates by age in three cities (one Canadian: Toronto: 50; one American: Chicago: 600; and one British: London: 30), Martin Daly and Margo Wilson found that the frequency of murder by age increases rapidly until the early 20s and then starts declining significantly by age 25. It is a maxim in criminology that there is a big decrease in criminal behavior after age 25. So although age is an important predictor, the community you grow up in is the most significant predictor of a subject's likelihood to commit murder.

Harry Harlow raising captive rhesus monkeys in isolation showed that (as with sexual behavior) their fixed action patterns are normal, but they never learned the appropriate social context for the behavior. So they show the normal behaviors of dominance and subordination, but they do not know when and to whom those displays should be given. In baboons, you inherit your rank from your mother. The social training for appropriate social behavior based on hereditary rank begins as early as the first week after birth. Similarly the Berkeley Hills hyenas took longer to establish their dominance hierarchy since they were raised without social context.

How important are parents and how important is peer group? Judith Rich Harris argues in her book "The Nurture Assumption" that peer influences are vastly more important than had been recognized. She observed that in immigrant families by age 4 or 5 the kids are developing the accent of the local community not that of their parents; they are becoming embarrassed by their parents' accent; they answer questions given in the home language using the outside language. We pick up the accent of our peer group, not our parents. "At the end of the day, parents are mostly good for determining which peer groups their kids have access to." Humans are very subject to conformity and conventionalized behavior in groups: once separated into groups we start to have disparaging beliefs about the other group; we overidentify with whatever category or group we are assigned. Within a week of working on the set of Planet of the Apes, actors sat at lunch with people from the same species that they were acting in the movie. All the actor chimps ate together even though their best friends were orangutans. Harris reinterpreted the result that kids growing up in a family without a father had a higher chance of antisocial violence as an adult: such families tend to be poorer and it is the peer socialization which leads to the effect.

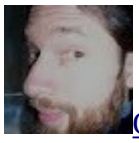
Steven Levitt (economist) and John Donohue (lawyer) determined that a significant part of the decrease in the crime rate since the 1970s (now thought to be 50%) is due to the Roe v. Wade decision that legalized abortion. As each state legalized abortion, there would be a 12-15 year lag before the crime rate would drop. At the beginning of the decrease in crime, it would be due to fewer teenagers entering the criminal justice system. As time goes on there are fewer criminals in the age range that grew up with safe, legal abortion. Evidently a huge predictor of growing up to be a violent antisocial individual is being an unwanted child. Abortion reduces the number of unwanted children which significantly reduces the crime rate.

Explicit declarative learning: you learn a fact, you know that you know it, and you can consciously use it, strategize with it, and use it in an executive way (requires hippocampus to store it)

Implicit procedural learning: your body knows it better than your head (stored in the cerebellum)

Is there a correlation between moral reasoning and moral behavior? The literature does not find a particularly good connection. Those who reach Kohlberg's post-conventional stage of moral reasoning tend not to be more likely to act heroically. The Carnegie Foundation studies those who do brave unexpected moral acts and finds they grow up in an environment with a very strong,

consistent, repeated stated imperative to act morally, to act bravely, and to not care what other people think. When interviewed, such people invariably report that they didn't think (for example before running into the burning building or jumping into the freezing river), so it is implicit procedural learning that spurs them to action. Something overlearned in childhood that does not require reasoning skills and effort.
Josh Green determined that players who cheated at least once (in a game designed to make it easy to cheat and easy for the researches to know when they were cheating) had increased activity in their frontal cortex. Those who didn't cheat never had frontal cortex activation: it was an implicit pathway: they were not even tempted. Perhaps their baked in moral rules made the question verboten.
Sapolsky concludes that he thinks a lot of what moral development is about is developing implicit rules by overlearning in childhood.

 CJ Fearnley - 2014-05-03 13:03:34-0400 - Updated: 2014-05-03 13:03:34-0400

Aggression IV. 102m Robert Sapolsky video.

Part 4 of 4 in a broad biological survey on empathy and aggression and the biological roots of human morality. Sapolsky begins by adding more detail to the discussion of perinatal effects on aggression. Then he covers the effects of genes. He predictably argues that the gene-environment interaction makes it difficult if not impossible to differentiate genetic effects. Then he gives an in depth exploration of cultural, ecological, environmental, and evolutionary effects. He concludes poignantly reminding us that the same exact behaviors can win medals or be the most horrific thing one human being can do to another.

20. Aggression IV

Perinatal Development

Organizational hormonal effects ("setting up the nervous system to respond later on to some sort of activational hormonal effect") vs. activational effects. Rodent studies of female androgenization (exposure to high levels of perinatal testosterone) show that there is a strong organizational effect resulting in more aggressive play, aggression, and less maternal behavior (activational effects) even with low adult testosterone levels. In primates, the patterns are strong but less dramatic than in rodents. In humans, female fetuses with congenital adrenal hyperplasia or those exposed to the drug DES (diethylstilbestrol) are androgenized. The literature suggests that there might be some hints of effects, but the confounds make the literature uninterpretable in Sapolsky's view.

Simon Baron-Cohen has developed the hypermale hypothesis of autism. Autism occurs more often in males. For problem-solving, males tend to use more analytical approaches to social problem-solving; females use more empathic approaches. The finger-length ratio (ratio between the length of the second and the fourth fingers), stronger spatial skills, the analytical problem-solving approach are all more pronounced in autistics. That is, normal male behavior verges on the pathology of autism.

Genetics

It used to be offensive to suggest that genes could have anything to do with aggression (pickets and

NIH funding restrictions and all that). Since hormones and receptors are all built from information in the DNA, genes can have a definite effect on behavior. But how do we distinguish a gene having to do with impulsivity versus one dealing with aggression? Since pain is a reliable releasing stimulus for aggression, the gene might have something to do with pain and not aggression. If the gene for general sympathetic nervous system arousal is more enhanced, in addition to more aggression you also see more affiliative behavior. Most of the early studies identified genes with indirect causes for more aggression.

Some genes have held up to very careful studies (namely, the serotonin synthesis and receptor genes and dopamine receptors). The effects are modulatory and subject to the environment early in life (mainly if there was abuse or stress). This is to be expected because all genes are subject to the gene-environment interaction where the genetic behavior only makes sense given its environmental context.

Sapolsky expects that the emerging field looking at different genotypes in different populations will soon be very interesting. But he doesn't feel it is mature enough to report on yet.

Even if we have identified genes increasing predisposition to aggressive behavior in some environmental context, we have no biological means to predict who will become a sociopath and who will become an aggressive monopoly or chess player. Just as we can't predict which person with damage to their frontal cortex will become a sociopath and which will be an annoying piano player.

Richard Speck broke in to the apartment of 8 student nurses in Chicago and slaughtered them in 1966. It was erroneously discovered and reported that Speck had 47 chromosomes (XYY karyotype). This started a mania about XYY and violence which proved to be totally unfounded. Aggression is no greater in XYY than normal XY males. More evidence that we ought not overvalue the genetic evidence.

Ecology & Culture and Aggression

Nomadic pastoralists (in contrast to traditional hunter/gatherers and farmers) have higher rates of violence (both in-group and out-group), more likely to have standing armies and warrior classes, leadership tends to come from the warrior class, and religions where success in war is a gateway to heaven. It makes since since animals need to be protected and groups can be widely dispersed. The American South was disproportionately settled by sheep herders from the Northern British Isles (nomadic pastoralists). These are cultures of honor: clear rules about triggers for aggression over symbolic slights. The higher murder rate in the American south occur in rural places among people who know each other (not in cities, not crimes for material). Richard E. Nisbett did a study to assess the differences in southern vs. northern culture in males: a confederate bumps past the subject in the hall and says "watch it asshole". The subject then goes into the study where their blood pressure and other physiological factors are tested. Northerners brush it off, Southerners have a massive stress response with high blood pressure and elevated stress hormone & testosterone levels.

Desert dwellers (who invented monotheism) and those living on open savannah grasslands tend to be nomadic pastoralists and they have higher rates of violence compared with rain forest dwellers who tend to be polytheistic and hunter/gatherers or small-scale farmers.

Those with cultural myths of victimization with an ethos of retribution tend to be more violent.

Altruistic punishing can be modeled in a game by giving the option of spending some of your resources to punish a cheating participant. In a study across many cultures of university students, on

average everyone spends about the same amount to punish cheaters. Antisocial punishment is where someone is punished for having been overly generous. The US, UK, and Scandinavian participants had the lowest rates of antisocial punishment; middle eastern countries and Slavic countries of the eastern block, Korea, and Turkey were in the middle; the worst such punishers were in Greece and the United Arab Emirates (UAE) where more punishment went to generous players more often than to cheaters. Why? If people started being generous it will up the ante for everyone. A deeper factor is the level of social trust (social capital) in the society.

What are the cultural and ideological environments that give rise to terrorism? Some view it as abnormal sociopathic behavior (individual dysfunction: neuropsychiatric), others view it as ideological extremes. There tends to be a consistent profile of terrorists (from the Boston Tea Party, the IRA, the Haganah in Israel): young, male, socially isolated, socially unaffiliated, relatively uneducated, history of childhood abuse. If they hadn't joined the terrorist group, they would be mugging old ladies. Middle Eastern terrorism follows a completely different profile: educated, well off people in their 30s and 40s (not young men), middle or upper class backgrounds, more women than in any previous kind of terrorism, individuals who tend to have no prior exposure to the oppression which they are fighting against, and relatively low levels of religiosity. Very surprising. Phil Zimbardo (who did the famed Stanford prison study) argues that under the right circumstances virtually anyone can do anything that is appalling. Another view is that because modern terrorism is international, only the better educated are able to get passports, navigate customs, and fly elsewhere.

Evolutionary Influences of Aggressive Behavior

In the vast majority of social species and in all human cultures, the major cause of aggression is "male-male violence over reproductive access to females". The Yanamamo have been intensely studied for decades (principally by Napoleon Chagnon). Chagnon published a highly controversial article in Science backed by decades of data that the more violent males have higher reproductive success (Sapolsky doesn't think the statistics are valid). Orangutans and other species including humans have rape. Is rape more about passing on copies of your genes or power and subjugation? Sapolsky's assessment of the field is that (except for Orangutans) it is mostly about power and subjugation.

The second most common cause of human aggression is males attacking females over denial of sexual access. The individual selection explanation applies: for a male to pass on copies of his genes he needs a "willing" female. Individual selection also accounts for female-female aggression, infanticide, and competitive infanticide.

Kin selection ("I will gladly give my life for 2 brothers or 8 cousins" as attributed to Haldane) accounts for why related individuals cooperate in circumstances of aggression. Chimps have female exogamy, so all the males in a chimp group are related and so they cooperate with each other resulting in warfare and even genocide. In most primates, kin selection explains why aggression is mainly between lineages (not within).

In humans, there is a much more fluid sense of "relatedness" or "us vs. them". A Bedouin saying explains: "It's my brothers, my cousins and I against the world; and it's my brothers and I against my cousins" (different translations are seen on the net). Humans have a relative sense of relatedness.

Daly and Wilson use a kin selection argument to explain why step-parents are more likely to abuse their stepkids than their biological father; a child is more likely to be abused by their paternal grandparent than their maternal grandparent (due to the greater certainty of paternity). Problems: the

Scandinavians cannot replicate these findings. It could be that economic stress increases violence (families with step fathers tend to have more economic stress).

Do we have prepared learning to see some us/them dichotomies better than others? Very controversial: what categories do kids divide people into? Are kids colorblind (skin color-wise)? Do they distinguish on other body characteristics?

Pseudokinship: people who feel as closely connected to each other as they would to their relatives. In military indoctrination a highly developed faculty has developed for making some people feel more related to each other than they actually are: one of the main points of military training is to forge a "band of brothers" to increase cooperation (that is, to increase the chance that you will give up your life for the person next to you).

The Maasai people in East Africa are a warrior culture that begins military training at 15 (it lasts 10 years). They live separately from everyone else, use kinship terms to refer to each other, share food with fellow warriors, and protect the herds and raid other villages. After they leave the military and get married, his wife will refer to members of his warrior cohort as her brother-in-law. The Israeli military allows a cohort of friends to join the same unit to increase pseudokinship.

In WWII, the heterogeneity of US troops meant that those of German ancestry were more related to the people they were fighting against. In Vietnam, there was a major failure of US military pseudokinship: the military moved people around so that no one was kept in a stable fighting unit. Apparently, this was done to reduce the high rates of soldiers shooting their officers.

Pseudospeciation: psychological mechanisms to make others seem so different from us that you don't even view them as human. Take Adolf Hitler, Osama bin Laden, Idi Amin Dada, Pol Pot, Heinrich Himmler, Adolf Eichmann, or Jim Jones: were they human? Do they deserve the same moral considerations as other human beings?

Examples include US WWII propaganda about ethnicities we were fighting, the 1994 Rwandan Genocide had the call to arms of "Kill the Cockroaches" (the Hutu killing the Tutsi). Another example was the Nayirah testimony in the run-up to the 1991 Gulf War which claimed that the Iraqis had removed the Kuwaiti babies from incubators to die: they aren't even human. The story was fabricated and pushed by PR firm Hill & Knowlton who was paid about $11 million by the Kuwaiti government, the measure to support military action passed by only 3 votes with 7 senators citing the fabricated story in their votes, and we went into the war with a 92% approval rating. A gigantic piece of pseudospeciation manipulation: "my god they leave babies out to die ... they are hardly even human".

After the break, Sapolsky summarizes: we've seen how individual and kin selection lead to more aggression. What evolutionary selective mechanisms lead to more cooperation, empathy, affiliation, and less violence?

In individual selection, there are alternative male strategies (be nice if you cannot be dominant). South American pair-bonded monkeys have low rates of aggression (in contrast to tournament species). Pseudokinship can also be used to decrease violence by making people feel more connected to each other. Examples of pseudokinship to support peace: in a traditional Bedouin society peacemaking effort, the rival clansmen start exploring each others' geneology until one party transparently fabricates a story about a common ancestor (aha, we're related, let's make peace). In Australian aboriginal groups when two individuals approach a water hole they start sharing their geneologies when "oh, we're relatives". Again it is a transparent effort at pseudokinship to share a vital resource without violence. After revolutions, the two sides are viewed as brothers reuniting:

using pseudokinship terms. After the French revolution everyone addressed each other in the familiar tense instead of the (illegal?) formal tense.

Elizabeth Phelps at NYU showed using fMRI that there is subliminal activation of the amygdala when a picture is flashed of someone of another race (it has been replicated a number of times). The amygdala sees us v. them in a split second. Susan Fiske at Princeton found that if some of the pictures have a big red dot and you are asked to tag them, then the amygdala does not activate on pictures of people of another race. Ask subjects to categorize the photos as below or over age 30 (treat the images as a category not an individual), now the amygdala is even more activated (us v. them priming). Finally, ask subjects to identify individuality in the photos (do they like coke or pepsi?), then the amygdala does not activate. The amygdala does not activate in people who grew up in racially and ethnically diverse neighborhoods nor for people who had a significant relationship with someone of another race.

Contact theory: aggression is decreased and affiliation is increased if you grow up with lots of contact with people from other cultures. It broadens one's sense of what is an "us". But studies have shown that if you send kids from different cultures to retreats or summer camps or other short programs together, the pseudokinship only lasts for a short while. To get a lasting inclusive "us" it is necessary to grow up in (or live for a long time in) a diverse community. More interfaces of contact between two groups doesn't guarantee less amygdala activation (less us vs. them).

Aggression can be increased because of the spatial characteristics of the interface between the two populations (there are regions where irritation, resources contention, and pockets of local unity can heighten us vs. them conflict instead of pseudokinship). The least optimal community interfaces predicted the locations of violence in the Balkans (Sapolsky only mentions Croatia & Bosnia, but there ware wars in Slovenia, & Kosovo in the 1990s too). I searched for but could not find the study Sapolsky cites. Do you know where it is?

The neurobiology of symbols in our brains can foster cooperation. Robert Axelrod at U Michigan has been showing the importance of symbols in peacemaking: being respectful of the other side's symbols makes it easier to share contested resources. "The power of symbols over rational contested resources."

Sinn Féin sent a 50th wedding anniversary gift to Ian Paisley which led to a massive breakthrough in Northern Ireland peace negotiations. The film Invictus shows how Nelson Mandela spent his time in prison becoming fluent in Afrikaans to build pseudokinship in negotiations to end apartheid. Mandela would invite the most difficult negotiator to sit next to him and offer especially considerate hospitality. Axelrod finds that Palestinian leaders say "if the Israelis would just once admit that the Palestinians got screwed in 1948 and we're sorry, then we'd be willing to make peace" and that Israeli generals say "if the Palestinians would just get the anti-semitic garbage out of their schoolbooks, we'd be willing to make peace". Respecting symbols and the legitimacy of the history is more important (and sometimes more difficult) than sharing the contested resources.

I think Sapolsky is slightly misusing Garrett Hardin's "tragedy of the commons" here. A tragedy of the commons occurs when a common pool (shared) resource is knowingly and regretfully but unavoidably degraded by a community. Sapolsky is exapting the term to apply to broader competitive situations as well.

How can cooperation evolve? In the prisoner's dilemma (a game designed to show why cooperation might fail even when it is in both parties' best interest) the tit-for-tat strategy starts off cooperating and then either cooperates or defects based on the other player's last action. One way to get cooperation going is with a founder population who due to kin selection in a geographically isolated

area develop cooperation which they can introduce and fix in the larger population because of its competitive benefits (this is the modern version of group selection). The phenomenon generalizes: whenever a cooperating population emerges amid a sea of non-cooperators, there is competitive pressure for cooperation. In New York City in the 1980s, Korean and Lebanese immigration increased. The Korean community opened many fruit/vegetable stands and the Lebanese regular grocery stores. The community of extant groceries complained that they were at an unfair disadvantage because the owners of the immigrant groceries were cooperating with each other with low-interest loans and other help. A community of trust can outcompete the previously entrenched larger non-cooperative population. "Those people are cheating, they are cooperating with each other, so either join in or you will be driven to extinction."

Important factors in fostering cooperation in game theory: 1) repetition: with repeated rounds punishment for cheating increases the chances for cooperation to start. Sapolsky emphasizes that the number of rounds cannot be known beforehand. If we all know this is the last round, then there is no "shadow of the future" to encourage cooperation and cheating is strategically justified. But knowing everyone will cheat in the last round, they will cheat in the second to last round and indeed all rounds will quickly succumb to the strategic cheater. 2) open book play: other players can know your record from previous rounds. Reputation encourages cooperation. 3) multiple unsynchronized games: there can be a psychological bleedover from cooperative games into other games where it would be less likely to develop (more like the real world). 4) altruistic punishment: if you can expend some of your resources to reduce their resources, then cooperation is more likely to emerge. 4) second party altruistic punishing: if an independent 3rd party can punish cheaters (an outside enforcer), cooperation is more likely to emerge even faster. 5) Secondary altruistic punishing: if an outside enforcer doesn't punish a cheater, the parties can punish the outside enforcer and cooperation can emerge even faster still (expectation of reporting honor code violations). 6) Secession: if a party can withdraw (secede) from a dyad (a pair of players) or from the whole game, then cooperation can emerge even faster.

Sapolsky cautions cooperation doesn't always lead to less aggression. Indeed when kin selection results in Chimpanzee males building strong cooperation, you end up with border patrols and even genocide. One of the scariest things on this planet is when a bunch of males start cooperating seriously because then they start to look at their neighbors. It seems that a prerequisite for genocide between groups is a reduction in homicide within a group. Does the decreasing homicide rate in the US put us at risk for committing the next genocide?

Sapolsky then tells stories of cooperation emerging in the trench warfare of WWI. On Christmas day 1914 a truce was agreed. Troops from both sides emerged from the trenches to play soccer, exchange gifts, singing and drinking. The officers couldn't get the troops to return to their jobs of killing each other for a few days. But that is an outside force establishing the cooperation. In WWI spontaneous cooperation ("truces") also arose between the two sides. How do you generate a reciprocally altruistic relationship with the enemy troops hidden from view in the next trench over who don't even speak my language? You have your best gunner fire repeatedly at the same tree 20 yards beyond the trenches. This communicates, our guy is really good and we are choosing not to put it in your trench. If the other side has their best gunner do the same thing, you now have a flaming war with shells constantly exploding but no one getting hurt due to the spontaneously negotiated non-aggression pact. The phenomenon is well documented and developed multiple times along the fronts of WWI. The only thing that stopped it from spreading was orders from higher up to attack or face court-martial.

Sapolsky ends poignantly. He tells of the 1979 story of when he was about 21 in the first year of his graduate research in East Africa. At that time Uganda's nightmare dictator Idi Amin miscalculated a border intrusion into Tanzania which resulted in the Tanzania army counterattacking and liberating

all of Uganda forcing Amin into exile. The day after the Tanzanians gained control of the southern part of Uganda by reaching the Kenyan border, Sapolsky decided to tour the war zone in Uganda. He figured seeing some violence would do good things for his 20 year old brain neurochemistry. After getting sufficiently scared, he decided to leave once he visited the source of the White Nile in a town he calls Tororo (but I don't see why he thought it the source of the white nile; maybe he meant Jinja, Uganda?). He tells of seeing a Ugandan soldier whose bloated body was tossed by the waves with his hands tied behind his back, strangled, and drowned. He felt a storm of emotions: "good, that's what you deserve being in Amin's army. No, wait a moment, this was probably some poor guy who was forced to do it. And thinking, no, I know what I think of soldiers who are just following orders. And thinking I ought to get a lot closer to see what is happening. And thinking, no, I better get as far away from here as possible." He stood there for an hour and half until some Tanzanian soldiers chased him away.

33 years after witnessing the dead Ugandan soldier, Sapolsky thinks he lectures longer and longer about aggression because of that soldier in Uganda: rational, professorial people who respect thought and think if you lecture on a subject long enough it will go away and will cure world aggression: "if only people can be lectured about the frontal cortex it will solve world violence. ... But the problem is that aggression is such a messy set of behaviors ... the same exact behaviors and depending on the context it could be something that could get a medal for someone, someone you'd want to mate with, vote for, reward, cheer on, join in, and in another setting it is the most frightening possible thing that can happen to us. And it is the same behaviors in all those cases. ... Violence is always going to be the hardest subject for us to understand biologically. And for that reason that it's always going to be the one we have to try hardest to understand."

CJ Fearnley

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http://www.youtube.com/watch?v=BqP4_4kr7-0
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Richard Speck broke in to the apartment of 8 student nurses in Chicago and slaughtered them in 1966. It was erroneously discovered and reported that Speck had 47 chromosomes (XYY karyotype). This started a mania about XYY and violence which proved to be totally unfounded. Aggression is no greater in XYY than normal XY males. More evidence that we ought not overvalue the genetic evidence.

Ecology & Culture and Aggression

Nomadic pastoralists (in contrast to traditional hunter/gatherers and farmers) have higher rates of violence (both in-group and out-group), more likely to have standing armies and warrior classes, leadership tends to come from the warrior class, and religions where success in war is a gateway to heaven. It makes since since animals need to be protected and groups can be widely dispersed. The American South was disproportionately settled by sheep herders from the Northern British Isles (nomadic pastoralists). These are cultures of honor: clear rules about triggers for aggression over symbolic slights. The higher murder rate in the American south occur in rural places among people who know each other (not in cities, not crimes for material). Richard E. Nisbett did a study to assess the differences in southern vs. northern culture in males: a confederate bumps past the subject in the hall and says "watch it asshole". The subject then goes into the study where their blood pressure and other physiological factors are tested. Northerners brush it off, Southerners have a massive stress response with high blood pressure and elevated stress hormone & testosterone levels.

Desert dwellers (who invented monotheism) and those living on open savannah grasslands tend to be nomadic pastoralists and they have higher rates of violence compared with rain forest dwellers who tend to be polytheistic and hunter/gatherers or small-scale farmers.
Those with cultural myths of victimization with an ethos of retribution tend to be more violent. Altruistic punishing can be modeled in a game by giving the option of spending some of your resources to punish a cheating participant. In a study across many cultures of university students, on average everyone spends about the same amount to punish cheaters. Antisocial punishment is where someone is punished for having been overly generous. The US, UK, and Scandinavian participants had the lowest rates of antisocial punishment; middle eastern countries and Slavic countries of the eastern block, Korea, and Turkey were in the middle; the worst such punishers were in Greece and the United Arab Emirates (UAE) where more punishment went to generous players more often than to cheaters. Why? If people started being generous it will up the ante for everyone. A deeper factor is the level of social trust (social capital) in the society.
What are the cultural and ideological environments that give rise to terrorism? Some view it as abnormal sociopathic behavior (individual dysfunction: neuropsychiatric), others view it as ideological extremes. There tends to be a consistent profile of terrorists (from the Boston Tea Party, the IRA, the Haganah in Israel): young, male, socially isolated, socially unaffiliated, relatively uneducated, history of childhood abuse. If they hadn't joined the terrorist group, they would be mugging old ladies. Middle Eastern terrorism follows a completely different profile: educated, well off people in their 30s and 40s (not young men), middle or upper class backgrounds, more women than in any previous kind of terrorism, individuals who tend to have no prior exposure to the oppression which they are fighting against, and relatively low levels of religiosity. Very surprising. Phil Zimbardo (who did the famed Stanford prison study) argues that under the right circumstances virtually anyone can do anything that is appalling. Another view is that because modern terrorism is international, only the better educated are able to get passports, navigate customs, and fly elsewhere.
Evolutionary Influences of Aggressive Behavior
In the vast majority of social species and in all human cultures, the major cause of aggression is "male-male violence over reproductive access to females". The Yanamamo have been intensely studied for decades (principally by Napoleon Chagnon). Chagnon published a highly controversial article in Science backed by decades of data that the more violent males have higher reproductive success (Sapolsky doesn't think the statistics are valid). Orangutans and other species including humans have rape. Is rape more about passing on copies of your genes or power and subjugation? Sapolsky's assessment of the field is that (except for Orangutans) it is mostly about power and subjugation.
The second most common cause of human aggression is males attacking females over denial of sexual access. The individual selection explanation applies: for a male to pass on copies of his genes he needs a "willing" female. Individual selection also accounts for female-female aggression, infanticide, and competitive infanticide.
Kin selection ("I will gladly give my life for 2 brothers or 8 cousins" as attributed to Haldane) accounts for why related individuals cooperate in circumstances of aggression. Chimps have female exogamy, so all the males in a chimp group are related and so they cooperate with each other resulting in warfare and even genocide. In most primates, kin selection explains why aggression is mainly between lineages (not within).
In humans, there is a much more fluid sense of "relatedness" or "us vs. them". A Bedouin saying explains: "It's my brothers, my cousins and I against the world; and it's my brothers and I against

my cousins" (different translations are seen on the net). Humans have a relative sense of relatedness.
Daly and Wilson use a kin selection argument to explain why step-parents are more likely to abuse their stepkids than their biological father; a child is more likely to be abused by their paternal grandparent than their maternal grandparent (due to the greater certainty of paternity). Problems: the Scandinavians cannot replicate these findings. It could be that economic stress increases violence (families with step fathers tend to have more economic stress).
Do we have prepared learning to see some us/them dichotomies better than others? Very controversial: what categories do kids divide people into? Are kids colorblind (skin color-wise)? Do they distinguish on other body characteristics?
Pseudokinship: people who feel as closely connected to each other as they would to their relatives. In military indoctrination a highly developed faculty has developed for making some people feel more related to each other than they actually are: one of the main points of military training is to forge a "band of brothers" to increase cooperation (that is, to increase the chance that you will give up your life for the person next to you).
The Maasai people in East Africa are a warrior culture that begins military training at 15 (it lasts 10 years). They live separately from everyone else, use kinship terms to refer to each other, share food with fellow warriors, and protect the herds and raid other villages. After they leave the military and get married, his wife will refer to members of his warrior cohort as her brother-in-law. The Israeli military allows a cohort of friends to join the same unit to increase pseudokinship.
In WWII, the heterogeneity of US troops meant that those of German ancestry were more related to the people they were fighting against. In Vietnam, there was a major failure of US military pseudokinship: the military moved people around so that no one was kept in a stable fighting unit. Apparently, this was done to reduce the high rates of soldiers shooting their officers.
Pseudospeciation: psychological mechanisms to make others seem so different from us that you don't even view them as human. Take Adolf Hitler, Osama bin Laden, Idi Amin Dada, Pol Pot, Heinrich Himmler, Adolf Eichmann, or Jim Jones: were they human? Do they deserve the same moral considerations as other human beings?
Examples include US WWII propaganda about ethnicities we were fighting, the 1994 Rwandan Genocide had the call to arms of "Kill the Cockroaches" (the Hutu killing the Tutsi). Another example was the Nayirah testimony in the run-up to the 1991 Gulf War which claimed that the Iraqis had removed the Kuwaiti babies from incubators to die: they aren't even human. The story was fabricated and pushed by PR firm Hill & Knowlton who was paid about $11 million by the Kuwaiti government, the measure to support military action passed by only 3 votes with 7 senators citing the fabricated story in their votes, and we went into the war with a 92% approval rating. A gigantic piece of pseudospeciation manipulation: "my god they leave babies out to die ... they are hardly even human".
After the break, Sapolsky summarizes: we've seen how individual and kin selection lead to more aggression. What evolutionary selective mechanisms lead to more cooperation, empathy, affiliation, and less violence?
In individual selection, there are alternative male strategies (be nice if you cannot be dominant). South American pair-bonded monkeys have low rates of aggression (in contrast to tournament species). Pseudokinship can also be used to decrease violence by making people feel more connected to each other. Examples of pseudokinship to support peace: in a traditional Bedouin society peacemaking effort, the rival clansmen start exploring each others' geneology until one party transparently fabricates a story about a common ancestor (aha, we're related, let's make peace). In

Australian aboriginal groups when two individuals approach a water hole they start sharing their geneologies when "oh, we're relatives". Again it is a transparent effort at pseudokinship to share a vital resource without violence. After revolutions, the two sides are viewed as brothers reuniting: using pseudokinship terms. After the French revolution everyone addressed each other in the familiar tense instead of the (illegal?) formal tense.

Elizabeth Phelps at NYU showed using fMRI that there is subliminal activation of the amygdala when a picture is flashed of someone of another race (it has been replicated a number of times). The amygdala sees us v. them in a split second. Susan Fiske at Princeton found that if some of the pictures have a big red dot and you are asked to tag them, then the amygdala does not activate on pictures of people of another race. Ask subjects to categorize the photos as below or over age 30 (treat the images as a category not an individual), now the amygdala is even more activated (us v. them priming). Finally, ask subjects to identify individuality in the photos (do they like coke or pepsi?), then the amygdala does not activate. The amygdala does not activate in people who grew up in racially and ethnically diverse neighborhoods nor for people who had a significant relationship with someone of another race.

Contact theory: aggression is decreased and affiliation is increased if you grow up with lots of contact with people from other cultures. It broadens one's sense of what is an "us". But studies have shown that if you send kids from different cultures to retreats or summer camps or other short programs together, the pseudokinship only lasts for a short while. To get a lasting inclusive "us" it is necessary to grow up in (or live for a long time in) a diverse community. More interfaces of contact between two groups doesn't guarantee less amygdala activation (less us vs. them).

Aggression can be increased because of the spatial characteristics of the interface between the two populations (there are regions where irritation, resources contention, and pockets of local unity can heighten us vs. them conflict instead of pseudokinship). The least optimal community interfaces predicted the locations of violence in the Balkans (Sapolsky only mentions Croatia & Bosnia, but there ware wars in Slovenia, & Kosovo in the 1990s too). I searched for but could not find the study Sapolsky cites. Do you know where it is?

The neurobiology of symbols in our brains can foster cooperation. Robert Axelrod at U Michigan has been showing the importance of symbols in peacemaking: being respectful of the other side's symbols makes it easier to share contested resources. "The power of symbols over rational contested resources."

Sinn Féin sent a 50th wedding anniversary gift to Ian Paisley which led to a massive breakthrough in Northern Ireland peace negotiations. The film Invictus shows how Nelson Mandela spent his time in prison becoming fluent in Afrikaans to build pseudokinship in negotiations to end apartheid. Mandela would invite the most difficult negotiator to sit next to him and offer especially considerate hospitality. Axelrod finds that Palestinian leaders say "if the Israelis would just once admit that the Palestinians got screwed in 1948 and we're sorry, then we'd be willing to make peace" and that Israeli generals say "if the Palestinians would just get the anti-semitic garbage out of their schoolbooks, we'd be willing to make peace". Respecting symbols and the legitimacy of the history is more important (and sometimes more difficult) than sharing the contested resources.

I think Sapolsky is slightly misusing Garrett Hardin's "tragedy of the commons" here. A tragedy of the commons occurs when a common pool (shared) resource is knowingly and regretfully but unavoidably degraded by a community. Sapolsky is exapting the term to apply to broader competitive situations as well.

How can cooperation evolve? In the prisoner's dilemma (a game designed to show why cooperation might fail even when it is in both parties' best interest) the tit-for-tat strategy starts off cooperating

and then either cooperates or defects based on the other player's last action. One way to get cooperation going is with a founder population who due to kin selection in a geographically isolated area develop cooperation which they can introduce and fix in the larger population because of its competitive benefits (this is the modern version of group selection). The phenomenon generalizes: whenever a cooperating population emerges amid a sea of non-cooperators, there is competitive pressure for cooperation. In New York City in the 1980s, Korean and Lebanese immigration increased. The Korean community opened many fruit/vegetable stands and the Lebanese regular grocery stores. The community of extant groceries complained that they were at an unfair disadvantage because the owners of the immigrant groceries were cooperating with each other with low-interest loans and other help. A community of trust can outcompete the previously entrenched larger non-cooperative population. "Those people are cheating, they are cooperating with each other, so either join in or you will be driven to extinction."

Important factors in fostering cooperation in game theory: 1) repetition: with repeated rounds punishment for cheating increases the chances for cooperation to start. Sapolsky emphasizes that the number of rounds cannot be known beforehand. If we all know this is the last round, then there is no "shadow of the future" to encourage cooperation and cheating is strategically justified. But knowing everyone will cheat in the last round, they will cheat in the second to last round and indeed all rounds will quickly succumb to the strategic cheater. 2) open book play: other players can know your record from previous rounds. Reputation encourages cooperation. 3) multiple unsynchronized games: there can be a psychological bleedover from cooperative games into other games where it would be less likely to develop (more like the real world). 4) altruistic punishment: if you can expend some of your resources to reduce their resources, then cooperation is more likely to emerge. 4) second party altruistic punishing: if an independent 3rd party can punish cheaters (an outside enforcer), cooperation is more likely to emerge even faster. 5) Secondary altruistic punishing: if an outside enforcer doesn't punish a cheater, the parties can punish the outside enforcer and cooperation can emerge even faster still (expectation of reporting honor code violations). 6) Secession: if a party can withdraw (secede) from a dyad (a pair of players) or from the whole game, then cooperation can emerge even faster.

Sapolsky cautions cooperation doesn't always lead to less aggression. Indeed when kin selection results in Chimpanzee males building strong cooperation, you end up with border patrols and even genocide. One of the scariest things on this planet is when a bunch of males start cooperating seriously because then they start to look at their neighbors. It seems that a prerequisite for genocide between groups is a reduction in homicide within a group. Does the decreasing homicide rate in the US put us at risk for committing the next genocide?

Sapolsky then tells stories of cooperation emerging in the trench warfare of WWI. On Christmas day 1914 a truce was agreed. Troops from both sides emerged from the trenches to play soccer, exchange gifts, singing and drinking. The officers couldn't get the troops to return to their jobs of killing each other for a few days. But that is an outside force establishing the cooperation. In WWI spontaneous cooperation ("truces") also arose between the two sides. How do you generate a reciprocally altruistic relationship with the enemy troops hidden from view in the next trench over who don't even speak my language? You have your best gunner fire repeatedly at the same tree 20 yards beyond the trenches. This communicates, our guy is really good and we are choosing not to put it in your trench. If the other side has their best gunner do the same thing, you now have a flaming war with shells constantly exploding but no one getting hurt due to the spontaneously negotiated non-aggression pact. The phenomenon is well documented and developed multiple times

along the fronts of WWI. The only thing that stopped it from spreading was orders from higher up to attack or face court-martial.
Sapolsky ends poignantly. He tells of the 1979 story of when he was about 21 in the first year of his graduate research in East Africa. At that time Uganda's nightmare dictator Idi Amin miscalculated a border intrusion into Tanzania which resulted in the Tanzania army counterattacking and liberating all of Uganda forcing Amin into exile. The day after the Tanzanians gained control of the southern part of Uganda by reaching the Kenyan border, Sapolsky decided to tour the war zone in Uganda. He figured seeing some violence would do good things for his 20 year old brain neurochemistry. After getting sufficiently scared, he decided to leave once he visited the source of the White Nile in a town he calls Tororo (but I don't see why he thought it the source of the white nile; maybe he meant Jinja, Uganda?). He tells of seeing a Ugandan soldier whose bloated body was tossed by the waves with his hands tied behind his back, strangled, and drowned. He felt a storm of emotions: "good, that's what you deserve being in Amin's army. No, wait a moment, this was probably some poor guy who was forced to do it. And thinking, no, I know what I think of soldiers who are just following orders. And thinking I ought to get a lot closer to see what is happening. And thinking, no, I better get as far away from here as possible." He stood there for an hour and half until some Tanzanian soldiers chased him away.
33 years after witnessing the dead Ugandan soldier, Sapolsky thinks he lectures longer and longer about aggression because of that soldier in Uganda: rational, professorial people who respect thought and think if you lecture on a subject long enough it will go away and will cure world aggression: "if only people can be lectured about the frontal cortex it will solve world violence. ... But the problem is that aggression is such a messy set of behaviors ... the same exact behaviors and depending on the context it could be something that could get a medal for someone, someone you'd want to mate with, vote for, reward, cheer on, join in, and in another setting it is the most frightening possible thing that can happen to us. And it is the same behaviors in all those cases. ... Violence is always going to be the hardest subject for us to understand biologically. And for that reason that it's always going to be the one we have to try hardest to understand."

Beyond Reductionism: The Biology of Complexity, Chaos & Emergence

Details

Is reductionism inadequate to explain many important phenomena in biology and other sciences? We may define reductionism as the idea that systems can be decomposed into more elementary parts which combine under definite rules to produce the whole. How does reductionism fail in biological systems? How and why is reductionsim still useful in the sciences including biology? What is the nature of the successes and failures of reductionism in science?

How do aperiodic deterministic systems show that predictiveness is impossible in many systems? If variability is inherent in some systems, how can we think of such dynamical systems scientifically? If predictiveness in science fails, how can we understand the world? In fractal systems (scale-free),

can their measurable "dimension" characterize their variability? Can science explain the aperiodic and variable after all?

What is the nature of the new ideas of chaoticism and complex systems which attempt to describe how complex properties emerge from components whose combinations are too unpredictable to encompass precisely?

How can the emergence of complexity develop from simple and unintelligent parts?

What can we learn about the nature of science from the development of complexity science as first documented for the general reader in James Glick's Pulitzer Prize winning book "Chaos: Making a New Science"?

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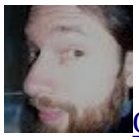
http://upload.wikimedia.org/wikipedia/en/thumb/f/f2/Chaos_Gleick_OReB.jpg/156px-Chaos_Gleick_OReB.jpg

 CJ Fearnley - 2015-04-26 12:57:41-0400 - Updated: 2015-04-26 12:57:41-0400
Reductionism is the idea that we can understand systems by decomposing them into more elementary parts which add up to produce a whole. Although it is one of the most successful ideas in science, it fails and breaks down in biology and many other complex and chaotic systems. How can we understand the strengths and limitations of reductionism? How can we glipmse the reality of those complex, chaotic, and emergent systems where reductionism fails to explain system behavior? The resources and description for this event will guide us in exploring this fascinating story about some profound limitations in modern science.

http://www.meetup.com/thinkingsociety/events/191317422/

Beyond Reductionism: The Biology of Complexity, Chaos & Emergence

CJ Fearnley

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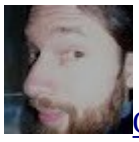 CJ Fearnley - 2015-04-13 18:05:15-0400 - Updated: 2015-04-13 18:05:15-0400
In this hour and a half video, Robert Sapolsky surveys the weaknesses (and the strengths) of reductionism in science. We learn that biological systems are complex, nonlinear, aperiodic systems where reductionism breaks down. That doesn't mean that reductionism is useless, just that we need to understand its limitations and we need better models for understanding scale-free and fractal systems in nature. The video discusses the butterfly effect, the Lorenzian waterwheel and other models of such complex systems.

http://www.youtube.com/watch?v=_njf8jwEGRo

This lecture is based on ideas from James Gleick's book "Chaos: Making a New Science" which is required reading for the course "Human Behavioral Biology" of which this is lecture 21.

Reductionism

I largely disagree with Sapolsky's caricature of the so-called "dark ages". Learning was not nonexistent in the medieval period. Scholarship has survived from the Carolingian and Ottonian

renaissances. The 12th century renaissance and the conquest of Toledo in 1085 shows that civilization in Europe was very actively engaged in the constant flux we call history. Sapolsky is correct that there was incredible ignorance about explaining causality in the world. Even though science has improved its approaches and become ever increasingly incisive, it is probable that Enlightenment and Modern science are only a few epsilon ahead of medieval science (except in terms of arrogance of which they indubitably excel). Look up Johannes Scotus Eriugena (c.815-c.877) and Lanfranc (c.1005-1089). Minor nit: the Alhambra is in Grenada, not Toledo.

Also, We should not forget Alhazen's pathbreaking contribution early in the 1000s of recommending systematic observation and experimental tests for science (which Dante (c.1265–1321) in his great "Comedìa" seemed to intimate). Finally, I'll note that the Islamic world (and China's great Tang dynasty) was triumphant and ascending in the period sometimes referred to as the "dark ages" which further points out the dubiousness of that designation.

Sapolsky claims that reductionism is "the single most important concept in all of science in the last 500 years". To make further progress, must science revise it basic approach to understanding?

Reductionism: the idea one can understand complex things by breaking them into their component parts. The concept of linearity or additivity suggests that the parts combine to form and explain the complex whole by simple addition.

Corollary: if you know the starting state of a system, you have 100% predictability of its future states. Conversely if you know the whole system and its parts, you can deduce its starting state.

So under the assumption of reductionism extrapolation and prediction is possible and powerful for understanding. A blueprint or road map is needed for the extrapolations: you have to know the rules or laws of addition or combination of parts into the whole.

Measurements have variability which the reductive world view interprets as noise in the system which needs to be controlled. That is, noise represents instrument error: it isn't "real". The reductive world view suggests that by looking into the component parts with a better and better microscope (breaking the component parts into their components and so forth), you can reduce the noise (less variability should be observed) and improve predictions.

"Sitting way down at the bottom of all these reductive processes there is an iconic and absolute idealized norm as to what the answer is ... Variability is discrepancy from seeing what the actual true measure is."

But reductionism breaks down in biological systems.

1st example: Hubel & Wiesel (1981 Nobel prize) discovered the functionality of the layered visual system. Neurons in the retina corresponded to the simplest parts of the visual cortex (a 1-1 mapping: they could recognize dots). Neurons in the second layer of the visual cortex can recognize straight lines. A third layer can respond to curves. A triumph of reductive science. But the logical reductive implication is that still higher layers of the visual cortex would recognize more complex patterns which were referred to as "grandmother neurons". Although a few such neurons have been found (you can read about "sparse" neurons such as the Jennifer Aniston neuron here: http://www.scientificamerican.com/article/brain-cells-searching-for-jennifer-aniston-neuron/), because of the combinatorial explosion of possibilities it is impossible for there to be many of them.

Experimental efforts to identify these higher layers in the brain have failed. "There aren't enough neurons in the brain to do facial recognition in a point-to-point reductive manner." There aren't

enough neurons to recognize the faces of everyone you know from every possible angle. Current research focuses on the non-reductive approach of neural networks which attempts to identify how patterns of information are encoded in large networks of neurons.

Bifurcating systems are scale-free. They model dendrites of neurons (dendritic trees), the circulatory and pulmonary system. You can't code for such bifurcating systems in a point-for-point reductive way wherein each gene specifies each bifurcation.

The importance of randomness, such as Brownian motion leading to unequal distributions of mitochondria (background information on transcription factors: https://plus.google.com/104222466367230914966/posts/KbfGBETCeP3 transposons: https://plus.google.com/104222466367230914966/posts/BK4xp8PFxav) in daughter cells, is not accounted for in a perfect prediction system based on reductive parts analysis. "Chance is throwing off this ability to know the starting state and know what this complex system is going to be."

Ivan Chase (http://www.pnas.org/content/99/8/5744.full) studied dominance hierarchies in fish and found that randomness plays a role in individual pairings which makes transitivity fail in group dynamics. "The dyadic pairing dominance outcomes has zero predictability over what the actual dominance hierarchy is going to be like. ... Chance plays a role as well: chance interactions of individuals affects their behavior. Reductionism breaks down again.

The most interesting biological systems cannot be explained with classical reductionism.

Chaoticism

Non-linear systems or non-additive systems are those which cannot be explained by a reductive science of breaking them into their component parts.

Aperiodic deterministic systems: you need to fully compute each step in the system to predict its behavior: you cannot skip steps or you lose predictability because the behavior of the system does not follow a clear periodic pattern. Periodicity implies reductionism.

Nondeterministic systems are random: there is no rule to determine the next step in the system. They are not chaotic.

On page 27 of James Gleick's book "Chaos: Making a New Science", the Lorenzian waterwheel (http://www.math.cornell.edu/~lipa/mec/lesson1.html) is described. A waterwheel with holes in the bottom to release water. As the flow of water increases the system progresses from boring (all the water flows out before any movement can begin), to periodic (perfectly predictable), to an increasingly complex period doubling behavior, to chaotic (completely unpredictable: "a pattern that never repeats") behavior. That is, the direction of the wheel can change at unpredictable times in the process. Heat/convection systems can have behavior akin to the waterwheel model. James York's famous paper "Period Three Implies Chaos" showed that odd periodicity leads to chaos (Gleick, p. 73).

Sapolsky explains that previous generations of scientists observed the turbulent/chaotic regime, but focused on the aspects that were periodic, predictable and reductive. That is, they broke off their studies just where reductionism broke down leading to a biased and incorrect view that the world is reductive when in fact almost all of the interesting cases live in the chaotic regime.

This is a profound point about science: when you control for variables in your experiments, you may miss the real complexity in the system by designing experiments that only look at the

reductive, predictable parts of the system.

When periodic systems are perturbed, they eventually return to their periodic behavior. An attractor is a point in a representation of behavior that equilibrates toward that center. Strange attractors in complex systems never return to the same point but oscillate around a center in a butterfly-like pattern. In periodic systems, variability from the attractor is noise. In chaotic systems, variability is the essence and nature of the system: there is no noise, just uniqueness.

The butterfly effect: when tiny differences in initial conditions produce large and unpredictable effects.

The variability in the strange attractors entail the butterfly effect. But the profound conclusion is that the system is really inherently variable in its nature and essence. So strange attractors have no predictability: no matter how accurate the instrumentation, the variability and noise stays prevalent (it is a scale-free system). Philosophically we conclude that there is no correct idealized answer. If there is an idealized answer, it is that noise and unpredictability are fundamental and inherent. The noise and variability is the system.

Complex systems in our world, especially biological ones, are inherently complex and are not subject to reductive analysis. Their "noise" is the system itself!

A fractal is a complex pattern that is scale-free (the idea of self-similarity may be clearer). The appearance and complexity of a fractal is the same no matter how closely you examine it. More formally, a fractal represents fractional dimensionality. In a fractal the amount of variability is the same no matter the resolution of the measurement.

The circulatory system, pulmonary system, neural dendrites, and the branches of a tree are all biological fractals. Fractal genes: genes giving instructions independent of scale.

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"Variability is the system rather than discrepancy from the system."

Reductionism is still very useful for biological systems "if you are not very picky, if you are not very precise". On the average, science and medicine can identify incisive results. But they do not apply to every case because then you enter the world of the nonperiodic, nonlinear, fractal systems. For example, reductive science can accurately show that its warmer in June than January even though we cannot predict the temperature three years out on any given day. If you only care about what is happening on the average to compare therapies or understand general behaviors of Universe, then reductionism gives useful results. Reductionism works for a general predictive sense. But if "you really want to understand the systems, it is anything but reductionism"!

CJ Fearnley's post

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In this hour and a half video, Robert Sapolsky surveys the weaknesses (and the strengths) of reductionism in science. We learn that biological systems are complex, nonlinear, aperiodic systems where reductionism breaks down. That doesn't mean that reductionism is useless, just that we need to understand its limitations and we need better models for understanding scale-free and fractal systems in nature. The video discusses the butterfly effect, the Lorenzian waterwheel and other models of such complex systems.
http://www.youtube.com/watch?v=_njf8jwEGRo
This lecture is based on ideas from James Gleick's book "Chaos: Making a New Science" which is required reading for the course "Human Behavioral Biology" of which this is lecture 21.
Reductionism
I largely disagree with Sapolsky's caricature of the so-called "dark ages". Learning was not nonexistent in the medieval period. Scholarship has survived from the Carolingian and Ottonian renaissances. The 12th century renaissance and the conquest of Toledo in 1085 shows that civilization in Europe was very actively engaged in the constant flux we call history. Sapolsky is correct that there was incredible ignorance about explaining causality in the world. Even though science has improved its approaches and become ever increasingly incisive, it is probable that Enlightenment and Modern science are only a few epsilon ahead of medieval science (except in terms of arrogance of which they indubitably excel). Look up Johannes Scotus Eriugena (c.815-c.877) and Lanfranc (c.1005-1089). Minor nit: the Alhambra is in Grenada, not Toledo.
Also, We should not forget Alhazen's pathbreaking contribution early in the 1000s of recommending systematic observation and experimental tests for science (which Dante (c.1265–1321) in his great "Comedìa" seemed to intimate). Finally, I'll note that the Islamic world (and China's great Tang dynasty) was triumphant and ascending in the period sometimes referred to as the "dark ages" which further points out the dubiousness of that designation.
Sapolsky claims that reductionism is "the single most important concept in all of science in the last 500 years". To make further progress, must science revise it basic approach to understanding?
Reductionism: the idea one can understand complex things by breaking them into their component parts. The concept of linearity or additivity suggests that the parts combine to form and explain the complex whole by simple addition.
Corollary: if you know the starting state of a system, you have 100% predictability of its future states. Conversely if you know the whole system and its parts, you can deduce its starting state.
So under the assumption of reductionism extrapolation and prediction is possible and powerful for understanding. A blueprint or road map is needed for the extrapolations: you have to know the rules or laws of addition or combination of parts into the whole.
Measurements have variability which the reductive world view interprets as noise in the system which needs to be controlled. That is, noise represents instrument error: it isn't "real". The reductive world view suggests that by looking into the component parts with a better and better microscope (breaking the component parts into their components and so forth), you can reduce the noise (less variability should be observed) and improve predictions.

"Sitting way down at the bottom of all these reductive processes there is an iconic and absolute idealized norm as to what the answer is ... Variability is discrepancy from seeing what the actual true measure is."
But reductionism breaks down in biological systems.
1st example: Hubel & Wiesel (1981 Nobel prize) discovered the functionality of the layered visual system. Neurons in the retina corresponded to the simplest parts of the visual cortex (a 1-1 mapping: they could recognize dots). Neurons in the second layer of the visual cortex can recognize straight lines. A third layer can respond to curves. A triumph of reductive science. But the logical reductive implication is that still higher layers of the visual cortex would recognize more complex patterns which were referred to as "grandmother neurons". Although a few such neurons have been found (you can read about "sparse" neurons such as the Jennifer Aniston neuron here: http://www.scientificamerican.com/.../brain-cells.../), because of the combinatorial explosion of possibilities it is impossible for there to be many of them.
Experimental efforts to identify these higher layers in the brain have failed. "There aren't enough neurons in the brain to do facial recognition in a point-to-point reductive manner." There aren't enough neurons to recognize the faces of everyone you know from every possible angle. Current research focuses on the non-reductive approach of neural networks which attempts to identify how patterns of information are encoded in large networks of neurons.
Bifurcating systems are scale-free. They model dendrites of neurons (dendritic trees), the circulatory and pulmonary system. You can't code for such bifurcating systems in a point-for-point reductive way wherein each gene specifies each bifurcation.
The importance of randomness, such as Brownian motion leading to unequal distributions of mitochondria (background information on transcription factors: https://plus.google.com/104222466367230.../posts/KbfGBETCeP3 transposons: https://plus.google.com/104222466367230.../posts/BK4xp8PFxav) in daughter cells, is not accounted for in a perfect prediction system based on reductive parts analysis. "Chance is throwing off this ability to know the starting state and know what this complex system is going to be."
Ivan Chase (http://www.pnas.org/content/99/8/5744.full) studied dominance hierarchies in fish and found that randomness plays a role in individual pairings which makes transitivity fail in group dynamics. "The dyadic pairing dominance outcomes has zero predictability over what the actual dominance hierarchy is going to be like. ... Chance plays a role as well: chance interactions of individuals affects their behavior. Reductionism breaks down again.
The most interesting biological systems cannot be explained with classical reductionism.
Chaoticism
Non-linear systems or non-additive systems are those which cannot be explained by a reductive science of breaking them into their component parts.
Aperiodic deterministic systems: you need to fully compute each step in the system to predict its behavior: you cannot skip steps or you lose predictability because the behavior of the system does not follow a clear periodic pattern. Periodicity implies reductionism.
Nondeterministic systems are random: there is no rule to determine the next step in the system. They are not chaotic.
On page 27 of James Gleick's book "Chaos: Making a New Science", the Lorenzian waterwheel (http://www.math.cornell.edu/~lipa/mec/lesson1.html) is described. A waterwheel with holes in the bottom to release water. As the flow of water increases the system progresses from boring (all the water flows out before any movement can begin), to periodic (perfectly predictable), to an increasingly complex period doubling behavior, to chaotic (completely unpredictable: "a pattern

that never repeats") behavior. That is, the direction of the wheel can change at unpredictable times in the process. Heat/convection systems can have behavior akin to the waterwheel model. James York's famous paper "Period Three Implies Chaos" showed that odd periodicity leads to chaos (Gleick, p. 73).

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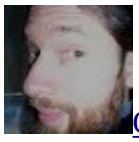 CJ Fearnley - 2015-04-29 20:18:23-0400 - Updated: 2015-05-09 08:33:45-0400
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Quality, excellence, complexity and adaptive optimization can emerge from a large quantity of simple elements operating with simple rules. "The simpler the constituent parts, the better. ... The more simple the building blocks, the better. ... More random interactions make for better more adaptive networks. ... That's how you stumble onto optimal solutions. Randomness is a good thing. ... [A]t the time that we're making new neurons in the cortex, that's when you induce the transposable events in the genome, that's where you juggle the DNA producing randomness. ... Randomness is a good thing. Randomness adds to the excellence of networks."

Gradients of attraction and repulsion provide a lot of the optimization in these systems. Nearest neighbor interactions with simple rules are vitally important. "Generalists work better than specialists. Generalists are more likely to come up with these adaptive outcomes." These emergent properties are where the complexity of human brains and their behavior comes from: but a lot more work needs to be done to really get a handle on how to think about these emergent properties.

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You can get complex adaptive optimized systems without top-down blueprints.

He implies throughout that reductionism has serious limitations and although we do not yet know how to get beyond reductionism in science (especially in the lab), these preliminary results from thinking about biology from this new complex systems / chaoticism perspective help us to understand better what makes us human and how the Universe actually operates.

Detailed notes below.

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Cellular automata are a great way to see the principles of chaos (Melanie Mitchell defines "chaos" as sensitive dependence on initial conditions), complex systems, the butterfly effect, etc. They use simple binary rules (filled in "cells" or not) to generate the next generation from a given starting state. In most cases, the rules bottom out to "all off" or "extinction". Many of the rules and the starting states that "succeed" (meaning do not go extinct), look very similar to each other after some number of generations. That is, there is a convergence to a small number of patterns. Which means that you cannot predict the starting state given the "mature" pattern. Also, given the starting state and the rule, it is impossible to predict what it will look like in 20 generations without actually calculating it out. That is, the starting state gives no predictive power for describing the mature state. Starting states that are asymmetrical tend to produce more complex patterns than symmetrical ones. You can read more about cellular automata at

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Analogously, we find that very simple patterns can generate different patterns of sea shells. In Africa and the Andes the very few plants that survive in the glacial equatorial mountains (at the 15,000 foot level), look very similar but are taxonomically (biologically) unrelated. So convergence onto common forms that survive in glacial equatorial mountains can happen from distinct starting points. Similarly we see convergence in animals and plants from arid regions. Biology seems to exhibit the same behaviors as do cellular automata: "slight differences magnify enormously (butterfly effects)".

Emergence: "ways in which you can code for a lot of the complexity in the natural world with small numbers of simple rules".

Neural networks: "information is not coded in a single molecule, a single synapse, a single neuron ... instead, information is coded in networks / in patterns of neural activation". He does an exercise with Paul Gauguin, Claude Monet, Vincent van Gogh, and Henri Marie Raymond de Toulouse-Lautrec-Monfa (and Picasso) as abstract expressionists, to try to illustrate how neural networks work: the more connected neurons "understand" abstract expressionism better.

Computers can effectively do sequential analytical calculations: The human brain can do parallel processing looking for patterns and similarities in a neural network where each neuron is at the intersection of many inputs. This gives the world of similarity and metaphor. Creativity, in this model, would be very broad networks finding connections and relationships in the neural network: "A broader network in some way is going to have wiring that is more divergent at the intersection of a bunch of networks that are acting in a convergent way." In the associational cortex (which no one understands very well), you find neurons that are multimodal in their responses: "all sorts of things stimulate them". They are neither sensory neurons (in the first few layers of sensory processing) nor are they grandmother neurons that respond to just one thing.

Karl Lashley was searching for engrams (a postulated representation of memory) storing individual facts by destroying parts of the brains of experimental animals. "He couldn't make the information disappear" until he destroyed broader areas. He concluded in his famous paper "In search of the engram" that there could be no such thing as memory. Which Sapolsky takes as evidence that neurons work in networks and not in localized structures. Early on in Alzheimer's when there are just a few neurons killed, the memories are just harder to recall. Individual memories don't die, the network is weakened by the losses. Memories appear to be stored in networks of neurons.

Fractal genes: "genes whose instructions are scale-free". The rule grow until five times longer than the width then bifurcate is a simple fractal rule to grow tree-like patterns. Thought experiment: if protein stability were a function of length (for example, when structuring a tube), at a certain length relative to width, the proteins could stop cohering until the frayed ends form a bifurcation split.

There is no cell in your body that is more than five cells away from a blood vessel. The circulatory system comprises less than 5% of body mass. It is a system that is "everywhere" but uses almost no space. The cantor set (see p. 93 of James Gleick's "Chaos" or http://en.wikipedia.org/wiki/Cantor_set) is an infinite process of cutting out the middle third of a line and then the middle third of each remaining piece and so on ad infinitum. The result is "an infinitely large number of objects, lines, that take up an infinitely small amount of space". It has a fractal dimension between 0 (the dimension of a point) and 1 (the dimension of a line).

The Koch snowflake (see Gleick p. 99 or http://en.wikipedia.org/wiki/Koch_snowflake): cut out the middle third of a line segment and replace it with a triangle whose edges are 1/3 the length of the

original segment. This results in the impossible situation of an object with an infinite perimeter in a finite space. It's fractal dimension is between 1 and 2 (the dimension of a plane). It gives an idea of how we can have a large surface area but using a negligible amount of space.

The Menger sponge (see Gleick p. 101 or http://en.wikipedia.org/wiki/Menger_sponge) in the limit case (at infinity) is an object with an infinite amount of surface area but no volume. It's fractal dimension lies between 2 and 3 (the dimension of ordinary space).

These three examples, the cantor set, the Koch snowflake, and the Menger sponge give an idea about how the body solves the packing problem for the circulatory system and other biological systems "that is everywhere and taking up virtually no space". They are examples of "how you can use a fractal system to solve the packing problem."

Fractal mutations: a mutation that has consequences that are scale-free. Kallmann syndrome: stuff that is wrong with midline structures in the body such as the nasal septum (separating the nostrils), in the hypothalamus, and in the septum of the heart.

Paul Green (1931-1998), developmental biologist, explained how when you heat a disk with a softer interior than the surface, you get a double saddle shape in the resultant form. "So that's how you get a potato chip." Similarly when plants bifurcate with shoots coming out, example after example have a double saddle shape. The form may be a mathematical necessity with no specification in the genetics. "It is an emergent property of the physical constraints of the system."

In 1906 Francis Galton observed at a county fair that the median guess of the weight of an ox was very accurate even though most of the guesses were not. This has become a famous example for the "wisdom of the crowds". On the quiz show "Who Wants to Be a Millionaire", contestants often choose their answer with the aide of the average guess of the audience which tends to be correct over 90% of the time. Prediction markets: average the predictions of a number of experts to get a more accurate guess. "Put a lot of somewhat decent experts together on a problem and they will be more accurate than almost any one single amazing expert." Provided they really are experts and provided their biases are randomly distributed. A group of ants moving a food source to the nest may use a wisdom of the crowds kind of average to act in concert to head there directly even though each ant tends to wander a bit.

Field of emergent complexity. A small number of rules governing the behavior of many "simple" participants (agent based systems). When they act in sufficient numbers with these nearest neighbor rules, a complex adaptive system emerges. It is a bottom up organization as no agent (ant in Sapolsky's example) has any instructions or notion of what needs to be done at the aggregate, emergent level.

Travelling salesman problem: visit each of n randomly spaced locales in the least effort (distance or time). It cannot be solved mathematically (the combinatorial complexity makes listing all possibilities impossible). We can program virtual ants with "swarm intelligence" so that after many iterations, a pretty efficient solution emerges. The idea is for the ants to travel from location to location randomly but they leave a pheromone trail as they go. Because the pheromone steadily evaporates, the less efficient paths will have less pheromone and gradually more and more ants will follow the pheromone trails of the shorter paths and reinforce it with their own pheromone. This is not a wisdom of the crowds example because the ants have no concept about shortest distance and finding an efficient path. It is an emergent swarm intelligence that isn't present at the level of the agents in the system.

Sapolsky suggests that bees in relocating their colony will use a similar method to move to where

the better food source is by swarming toward those locations that elicit the longer waggle dance (which implies a better food source; more on the bees waggle dance: http://www.youtube.com/watch?v=bFDGPgXtK-U).

Another approach is to have agents that attract or repel one another (for example magnetic poles). So if different shops are attracted to each other but repelled by identical shops, an agent based model will emerge with clusters as business districts where the forces of attraction and repulsion balance. An urban plan can be developed in this way. Hmm, maybe that is how our cities actually formed: by a complex of agent-based actions of humans as mindless ants acting in their world?

Neural growth can be modelled on similar attraction and repulsion rules. Certain attractors cause a neural projection to be sent toward it while repulsive signals cause projections to be sent in the other direction.

Sapolsky may be referencing this study in Science "Rules for Biologically Inspired Adaptive Network Design" http://www.sciencemag.org/content/327/5964/439.abstract which is summarized here http://blogs.discovermagazine.com/80beats/2010/01/22/brainless-slime-mold-builds-a-replica-tokyo-subway/ which he says shows that these agent-based systems can more accurately than skilled humans map out complex systems like subway systems.

Since molecules can have + and - charges on them, building proteins and other molecules can assemble according to these models.

Harold C. Urey and Stanley L. Miller experiment (http://en.wikipedia.org/wiki/Miller%E2%80%93Urey_experiment): synthesized complex organic compounds including amino acids from simple molecules by adding electricity. The experiment suggests that with the right ingredients and lightening, life could have formed spontaneously on the early Earth. Later research showed that the electricity might not be needed: just having charged molecules and random mixing will eventually result in complex organic compounds.

Power law distributions have a decaying rate of occurrence. For example less severe earthquakes occur much more frequently than major devastating earthquakes such as the 7.8 one in Nepal or the 9.0 one in Japan in 2011. The same power law distribution is observed in distance to the other party in phone calls, the distance that dollar bills travel (he may be referring to Brockmann's paper: http://rocs.northwestern.edu/research/wgstory_assets/brockmann2006nature.pdf). The number of links between web sites, complexity of proteins, the rate that people send e-mails, the amount of separation to Kevin Bacon all follow a power law distribution. Such distributions are intrinsically fractal: the pattern is similar at different scales and for different subjects.

The distribution of neural projections in the brain follows a power law which seems to be an efficient way to get stable interactions amongst clusters of nodes (neurons) but also has a few long distance connections. Sapolsky suggests that data from fetal neural development supports the power law distribution behavior.

Some evidence suggests that the projection profile of neurons in the cortex of autistic children follows a steeper than normal power law distribution so that there are more close-by projections and too few long distance connections. This may produce modules of function that are isolated from others. Autism may be a lack of integration between brain functions. Could it be a disease in the shape of the power law distribution of neural connections? Even though there are a perfectly normal number of neurons and probably a normal number of connections between neurons, we might have a malady due to the distribution of those connections.

Male brains also have a steeper power law distribution with more modular (local) connections and fewer connections to distant regions. Due to this, the average corpus callosum is thicker in women than in men (even thinner in the autistic). The corpus callosum is a wide, flat bundle of neural fibers beneath the cortex that connects the two hemispheres of the brain in most mammals.

Instead of the old world of expert reviews, we now live in a bottom up, self-correcting world of Amazon and Netflix ratings built on attraction and repulsion rules (likes and dislikes) wherein good recommendations emerge from a cacophony of voices (inputs). Wikipedia being one of the preeminent examples. A study in Nature (http://www.nature.com/nature/journal/v438/n7070/full/438900a.html) found that the hard-nosed facts in Wikipedia were nearly as accurate as those in Encyclopaedia Britannica. The wisdom of the crowds can produce a self-correcting, accurate adaptive system with no blueprint and no top-down control. Weighted wisdom of the crowds sites appear to generate even better results. A significant drawback to these systems is a bias toward conformity, that is, facts or opinions with large variance (including outliers) may not bottom up emerge accurately.

How does the nervous system wire up its power law distribution of neural projections? Swarm intelligence: radial glial cells form a pioneer generation of radially oriented cells. Then neurons in later generations act as "random wanderers" preferentially growing along the radial glial cells. This process produces the optimal power law distribution all from simple local rules.

"Looking at a single neuron you can't tell which species it came from". Humans and fruit flys are indistinguishable. We have the same neurotransmitters, ion channels, action potentials as flys and worms ("minor details are different"). The difference is that we have more neurons. Fun anecdote: Kasparov vs. Deep Blue: the first time a computer beat a top-level chess grandmaster. Kasparov said "with enough quantity [computational power] you invent quality". It is the quantity of neurons in the human nervous system that gives us our more nuanced mental faculties despite the fact that our brains have the same basic neuronal building blocks as all other animals (and insects).

Now that the human and chimp genomes have been sequenced, we can compare them. Humans and chimps share 98% of their DNA. What's the 2% that differ? They disproportionately code for transcription factors (background information on transcription factors: https://plus.google.com/104222466367230914966/posts/KbfGBETCeP3), splicing enzymes, and non-coding regions. Humans have about 1000 fewer olfactory receptors than do chimps due to pseudo-genes that do not express in humans: that's about half the difference between chimps and humans! There were some changes due to morphology and bone development (probably related to our bipedalism), there was one change in genes affecting hair development (humans are less hairy), and some differences in some mating related genes. There were very few differences related to brain development: in humans neural progenitor cells undergo an increased number of rounds of cell division than in chimps: our only significant neural difference from chimp brains is the number of cells in our brains! Qualitatively chimps have the same kind of brain cells as we do.

Sapolsky predicts that within our lifetime, there we will a revolution that collapses some government effectuated from our living rooms with a bottom-up Internet initiative with no physical demonstration or even bloodshed.

CJ Fearnley

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Analogously, we find that very simple patterns can generate different patterns of sea shells. In Africa and the Andes the very few plants that survive in the glacial equatorial mountains (at the 15,000 foot level), look very similar but are taxonomically (biologically) unrelated. So convergence onto common forms that survive in glacial equatorial mountains can happen from distinct starting points. Similarly we see convergence in animals and plants from arid regions. Biology seems to exhibit the same behaviors as do cellular automata: "slight differences magnify enormously (butterfly effects)".

Emergence: "ways in which you can code for a lot of the complexity in the natural world with small numbers of simple rules".
Neural networks: "information is not coded in a single molecule, a single synapse, a single neuron ... instead, information is coded in networks / in patterns of neural activation". He does an exercise with Paul Gauguin, Claude Monet, Vincent van Gogh, and Henri Marie Raymond de Toulouse-Lautrec-Monfa (and Picasso) as abstract expressionists, to try to illustrate how neural networks work: the more connected neurons "understand" abstract expressionism better.
Computers can effectively do sequential analytical calculations: The human brain can do parallel processing looking for patterns and similarities in a neural network where each neuron is at the intersection of many inputs. This gives the world of similarity and metaphor. Creativity, in this model, would be very broad networks finding connections and relationships in the neural network: "A broader network in some way is going to have wiring that is more divergent at the intersection of a bunch of networks that are acting in a convergent way." In the associational cortex (which no one understands very well), you find neurons that are multimodal in their responses: "all sorts of things stimulate them". They are neither sensory neurons (in the first few layers of sensory processing) nor are they grandmother neurons that respond to just one thing.
Karl Lashley was searching for engrams (a postulated representation of memory) storing individual facts by destroying parts of the brains of experimental animals. "He couldn't make the information disappear" until he destroyed broader areas. He concluded in his famous paper "In search of the engram" that there could be no such thing as memory. Which Sapolsky takes as evidence that neurons work in networks and not in localized structures. Early on in Alzheimer's when there are just a few neurons killed, the memories are just harder to recall. Individual memories don't die, the network is weakened by the losses. Memories appear to be stored in networks of neurons.
Fractal genes: "genes whose instructions are scale-free". The rule grow until five times longer than the width then bifurcate is a simple fractal rule to grow tree-like patterns. Thought experiment: if protein stability were a function of length (for example, when structuring a tube), at a certain length relative to width, the proteins could stop cohering until the frayed ends form a bifurcation split.
There is no cell in your body that is more than five cells away from a blood vessel. The circulatory system comprises less than 5% of body mass. It is a system that is "everywhere" but uses almost no space. The cantor set (see p. 93 of James Gleick's "Chaos" or http://en.wikipedia.org/wiki/Cantor_set) is an infinite process of cutting out the middle third of a line and then the middle third of each remaining piece and so on ad infinitum. The result is "an infinitely large number of objects, lines, that take up an infinitely small amount of space". It has a fractal dimension between 0 (the dimension of a point) and 1 (the dimension of a line).
The Koch snowflake (see Gleick p. 99 or http://en.wikipedia.org/wiki/Koch_snowflake): cut out the middle third of a line segment and replace it with a triangle whose edges are 1/3 the length of the original segment. This results in the impossible situation of an object with an infinite perimeter in a finite space. It's fractal dimension is between 1 and 2 (the dimension of a plane). It gives an idea of how we can have a large surface area but using a negligible amount of space.
The Menger sponge (see Gleick p. 101 or http://en.wikipedia.org/wiki/Menger_sponge) in the limit case (at infinity) is an object with an infinite amount of surface area but no volume. It's fractal dimension lies between 2 and 3 (the dimension of ordinary space).
These three examples, the cantor set, the Koch snowflake, and the Menger sponge give an idea about how the body solves the packing problem for the circulatory system and other biological systems "that is everywhere and taking up virtually no space". They are examples of "how you can use a fractal system to solve the packing problem."

Fractal mutations: a mutation that has consequences that are scale-free. Kallmann syndrome: stuff that is wrong with midline structures in the body such as the nasal septum (separating the nostrils), in the hypothalamus, and in the septum of the heart.
Paul Green (1931-1998), developmental biologist, explained how when you heat a disk with a softer interior than the surface, you get a double saddle shape in the resultant form. "So that's how you get a potato chip." Similarly when plants bifurcate with shoots coming out, example after example have a double saddle shape. The form may be a mathematical necessity with no specification in the genetics. "It is an emergent property of the physical constraints of the system."
In 1906 Francis Galton observed at a county fair that the median guess of the weight of an ox was very accurate even though most of the guesses were not. This has become a famous example for the "wisdom of the crowds". On the quiz show "Who Wants to Be a Millionaire", contestants often choose their answer with the aide of the average guess of the audience which tends to be correct over 90% of the time. Prediction markets: average the predictions of a number of experts to get a more accurate guess. "Put a lot of somewhat decent experts together on a problem and they will be more accurate than almost any one single amazing expert." Provided they really are experts and provided their biases are randomly distributed. A group of ants moving a food source to the nest may use a wisdom of the crowds kind of average to act in concert to head there directly even though each ant tends to wander a bit.
Field of emergent complexity. A small number of rules governing the behavior of many "simple" participants (agent based systems). When they act in sufficient numbers with these nearest neighbor rules, a complex adaptive system emerges. It is a bottom up organization as no agent (ant in Sapolsky's example) has any instructions or notion of what needs to be done at the aggregate, emergent level.
Travelling salesman problem: visit each of n randomly spaced locales in the least effort (distance or time). It cannot be solved mathematically (the combinatorial complexity makes listing all possibilities impossible). We can program virtual ants with "swarm intelligence" so that after many iterations, a pretty efficient solution emerges. The idea is for the ants to travel from location to location randomly but they leave a pheromone trail as they go. Because the pheromone steadily evaporates, the less efficient paths will have less pheromone and gradually more and more ants will follow the pheromone trails of the shorter paths and reinforce it with their own pheromone. This is not a wisdom of the crowds example because the ants have no concept about shortest distance and finding an efficient path. It is an emergent swarm intelligence that isn't present at the level of the agents in the system.
Sapolsky suggests that bees in relocating their colony will use a similar method to move to where the better food source is by swarming toward those locations that elicit the longer waggle dance (which implies a better food source; more on the bees waggle dance: http://www.youtube.com/watch?v=bFDGPgXtK-U).
Another approach is to have agents that attract or repel one another (for example magnetic poles). So if different shops are attracted to each other but repelled by identical shops, an agent based model will emerge with clusters as business districts where the forces of attraction and repulsion balance. An urban plan can be developed in this way. Hmm, maybe that is how our cities actually formed: by a complex of agent-based actions of humans as mindless ants acting in their world?
Neural growth can be modelled on similar attraction and repulsion rules. Certain attractors cause a neural projection to be sent toward it while repulsive signals cause projections to be sent in the other direction.

Sapolsky may be referencing this study in Science "Rules for Biologically Inspired Adaptive Network Design" http://www.sciencemag.org/content/327/5964/439.abstract which is summarized here http://blogs.discovermagazine.com/.../brainless-slime.../ which he says shows that these agent-based systems can more accurately than skilled humans map out complex systems like subway systems.
Since molecules can have + and - charges on them, building proteins and other molecules can assemble according to these models.
Harold C. Urey and Stanley L. Miller experiment (http://en.wikipedia.org/wiki/Miller%E2%80%93Urey_experiment): synthesized complex organic compounds including amino acids from simple molecules by adding electricity. The experiment suggests that with the right ingredients and lightening, life could have formed spontaneously on the early Earth. Later research showed that the electricity might not be needed: just having charged molecules and random mixing will eventually result in complex organic compounds.
Power law distributions have a decaying rate of occurrence. For example less severe earthquakes occur much more frequently than major devastating earthquakes such as the 7.8 one in Nepal or the 9.0 one in Japan in 2011. The same power law distribution is observed in distance to the other party in phone calls, the distance that dollar bills travel (he may be referring to Brockmann's paper: http://rocs.northwestern.edu/.../brockmann2006nature.pdf). The number of links between web sites, complexity of proteins, the rate that people send e-mails, the amount of separation to Kevin Bacon all follow a power law distribution. Such distributions are intrinsically fractal: the pattern is similar at different scales and for different subjects.
The distribution of neural projections in the brain follows a power law which seems to be an efficient way to get stable interactions amongst clusters of nodes (neurons) but also has a few long distance connections. Sapolsky suggests that data from fetal neural development supports the power law distribution behavior.
Some evidence suggests that the projection profile of neurons in the cortex of autistic children follows a steeper than normal power law distribution so that there are more close-by projections and too few long distance connections. This may produce modules of function that are isolated from others. Autism may be a lack of integration between brain functions. Could it be a disease in the shape of the power law distribution of neural connections? Even though there are a perfectly normal number of neurons and probably a normal number of connections between neurons, we might have a malady due to the distribution of those connections.
Male brains also have a steeper power law distribution with more modular (local) connections and fewer connections to distant regions. Due to this, the average corpus callosum is thicker in women than in men (even thinner in the autistic). The corpus callosum is a wide, flat bundle of neural fibers beneath the cortex that connects the two hemispheres of the brain in most mammals.
Instead of the old world of expert reviews, we now live in a bottom up, self-correcting world of Amazon and Netflix ratings built on attraction and repulsion rules (likes and dislikes) wherein good recommendations emerge from a cacophony of voices (inputs). Wikipedia being one of the preeminent examples. A study in Nature (http://www.nature.com/.../v438/n7070/full/438900a.html) found that the hard-nosed facts in Wikipedia were nearly as accurate as those in Encyclopaedia Britannica. The wisdom of the crowds can produce a self-correcting, accurate adaptive system with no blueprint and no top-down control. Weighted wisdom of the crowds sites appear to generate even better results. A significant drawback to these systems is a bias toward conformity, that is, facts or opinions with large variance (including outliers) may not bottom up emerge accurately.

How does the nervous system wire up its power law distribution of neural projections? Swarm intelligence: radial glial cells form a pioneer generation of radially oriented cells. Then neurons in later generations act as "random wanderers" preferentially growing along the radial glial cells. This process produces the optimal power law distribution all from simple local rules.
"Looking at a single neuron you can't tell which species it came from". Humans and fruit flys are indistinguishable. We have the same neurotransmitters, ion channels, action potentials as flys and worms ("minor details are different"). The difference is that we have more neurons. Fun anecdote: Kasparov vs. Deep Blue: the first time a computer beat a top-level chess grandmaster. Kasparov said "with enough quantity [computational power] you invent quality". It is the quantity of neurons in the human nervous system that gives us our more nuanced mental faculties despite the fact that our brains have the same basic neuronal building blocks as all other animals (and insects).
Now that the human and chimp genomes have been sequenced, we can compare them. Humans and chimps share 98% of their DNA. What's the 2% that differ? They disproportionately code for transcription factors (background information on transcription factors: https://plus.google.com/104222466367230.../posts/KbfGBETCeP3), splicing enzymes, and non-coding regions. Humans have about 1000 fewer olfactory receptors than do chimps due to pseudo-genes that do not express in humans: that's about half the difference between chimps and humans! There were some changes due to morphology and bone development (probably related to our bipedalism), there was one change in genes affecting hair development (humans are less hairy), and some differences in some mating related genes. There were very few differences related to brain development: in humans neural progenitor cells undergo an increased number of rounds of cell division than in chimps: our only significant neural difference from chimp brains is the number of cells in our brains! Qualitatively chimps have the same kind of brain cells as we do.
Sapolsky predicts that within our lifetime, there we will a revolution that collapses some government effectuated from our living rooms with a bottom-up Internet initiative with no physical demonstration or even bloodshed.

The Biology of Language

Details

What properties are common to all human languages? What can we learn about the biology of our language from people with brain defects such as Williams syndrome where people whose brain is so damaged that they have low IQs yet they are fluent in language? What does sign language tell us about the nature of our language faculty? Is our view of the world influenced by our language? What are the biological and cultural implications of the imminent loss of the countless languages that are no longer being taught to children?

Is language a motor skill (controlling breath, lips, and tongue or hands in the case of sign language)? What cognitive role is there in language? How do we learn language? What are the language centers of the brain?

What do we know about the genetics of language? How did language evolve? Can apes and chimpanzees learn sign language?

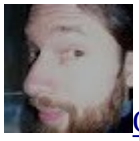
This topic will be based on two optional video lectures by Robert Sapolsky about the biology of language.

• Nearly 2 hour video by Robert Sapolsky on Language. Read Jon Dakins' notes summarizing the lecture. (http://www.robertsapolskyrocks.com/language.html) Read CJ Fearnley's detailed notes summarizing and commenting on the lecture. (https://www.facebook.com/cj.fearnley/posts/10206547472061163)

https://www.youtube.com/watch?v=SIOQgY1tqrU

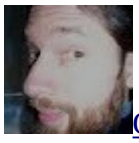
• Only the first 23 minutes of the following video are on the biology of Language (we will not discuss the second part of the video on Schizophrenia at this meetup). Read Jon Dakin's notes summarizing the lecture (http://www.robertsapolskyrocks.com/schizophrenia.html). Read CJ Fearnley's detailed notes summarizing and commenting on the first 23 minutes of the lecture (https://www.facebook.com/cj.fearnley/posts/10206555305977006).

https://www.youtube.com/watch?v=nEnklxGAmak

 CJ Fearnley - 2015-06-28 11:18:25-0400 - Updated: 2015-06-28 11:18:25-0400
Biology can offer us unique clues to understand one of humanity's most profound and mysterious faculties, the ability to communicate through language. What are the universals of human language? What do we know about how the brain processes language? Is sign language different in any fundamental way from spoken language? What are the implications that roughly half of the approximately 7,000 languages on the planet are not being taught to children? Is our view of the world influenced by our language? Is a lost language a loss to humanity's ability to understand the world?

The link asks more questions and documents just over two hours of video material of Robert Sapolsky exploring the biology of language:
http://www.meetup.com/thinkingsociety/events/222860826/

The Biology of Language

 CJ Fearnley - 2015-06-28 11:18:25-0400 - Updated: 2015-06-28 11:18:25-0400

Biology can offer us unique clues to understand one of humanity's most profound and mysterious faculties, the ability to communicate through language. What are the universals of human language? What do we know about how the brain processes language? Is sign language different in any fundamental way from spoken language? What are the implications that roughly half of the approximately 7,000 languages on the planet are not being taught to children? Is our view of the world influenced by our language? Is a lost language a loss to humanity's ability to understand the world?

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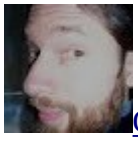
The Biology of Language

 CJ Fearnley - 2015-07-10 10:58:51-0400 - Updated: 2015-07-10 10:58:51-0400

Robert Sapolsky on Language: this video starts an exploration of human language. It covers language as a biological behavior and looks at universals of the roughly 6,000 or so human languages estimated to exist. It also explores the neurobiology of language, the acquisition and development of language, and a comparison with animal communication. The exploration of animal communication allows Sapolsky to suggest an answer to the question of what is unique about human language in the animal kingdom.

A deficiency of Sapolsky's biology-focused presentation is that it misses the tenor of the importance of language in human culture. So I'm motivating my notes with Wade Davis' poetic definition of language: "A language, by definition, is a flash of the human spirit. It's a vehicle through which the soul of each particular culture comes into the material realm. Every language [...] is an old growth forest of the mind, a watershed of thought, an ecosystem of social, spiritual, political possibilities."

That is even more poignant when you consider the fact that 90% of human languages are expected to go extinct this century. One of the most devastating signs of the rapacious side of globalization and religious missionaries.

http://www.youtube.com/watch?v=SIOQgY1tqrU

Behavior of Language. Universals across all human languages.

Semanticity: breakup of the continua of sounds into discrete "buckets" or units of meaning (words).

Embedded clauses: to express conditionals and circumstances.

Recursion (generativity): there is a potentially infinite number of combinations of words.

Displacement from emotions: ability to talk about the past and future; here and there; emotionally distant circumstances. Animal communication is overwhelmingly about their current emotional state.

Arbitrariness of language: words are not tied inextricably to their meaning: an arbitrary relationship between the signal and the message.

Meta-communication: the ability to talk about language.

Motherese or baby-talk: speaking to children in a higher-pitch voice, slow and clear, emphasizing melodic structure and vowels, repeating phrases, and focusing on the baby's face.

Is it about emotion or instruction? Pet talk is less slow and clear and doesn't focus on the face so it is not about instruction.

Is language more about cognition or the conceptual structure or about the generation and reception of sound (motor system)?

Gallaudet University in Washington, D.C. "was the first school for the advanced education of the deaf and hard of hearing in the world" (quote from https://en.wikipedia.org/wiki/Gallaudet_University).

Sign language suggests that language is mostly a cognitive faculty: it has many of the properties of language but uses different brain systems. Observe that at around 9 months, deaf and hearing babies begin to babble (practice fragments of whatever communication system they are exposed to). Pathologies of the brain affect ASL speakers in similar ways to people using spoken language.

Prosody refers to aspects of language like tone and emphasis that are not encoded in grammar or vocabulary. Sign language also has prosody.

Deaf people where ASL is their native language, use their auditory cortex when they "listen" to signs. Is that really evidence that language is about the underlying cognitive structures?

There are regional accents, poetry, and puns in ASL even though it doesn't use lips, tongue, or ears. If ASL is a second language it is "stored" in the same brain regions as a second spoken language.

Neurobiology of language.

How modular is the function of language in the brain? How separate is language from the rest of the cognitive functionality of the brain?

Williams syndrome is a genetic disease of very expressive, warm, affectionate, and verbally adept people whose IQs are near the borderline of intellectual disability. Their effective language skills imply that language is a separate faculty of the brain and not just one of many skills the brain develops. In addition, there are people with normal IQs who have various heritable language disabilities. Further supporting the idea that language is a modular function of the brain.

Nevertheless, people with Williams although fluent with words, do not communicate much meaning in their sentences. Moreover, many of the people with other heritable language disabilities are at the lower measures of intellectual function which suggests that language is probably connected in with general brain function (not so modular).

In conclusion, there is some evidence that language is a general function of the brain with quite a bit of localizations. Probably another "both/neither" rather than a clear cut situation.

The language function of the brain is lateralized (located on one side of the brain) to the left brain in 90% of people. In general, prosody is localized to the right brain in 90% of people.

The Wada test uses barbiturates to test for lateralized language and memory brain functions. Modern brain imaging will probably obsolesce this test.

Aphasia is the general term for being unable to communicate effectively.

There are three main regions of the brain associated with language: Broca's area, Wernicke's area and the arcuate fasciculus.

Broca's area is the edge of the brain region that handles motoric function (moving your body). It is about moving your lips and larynx. A Broca's aphasia or production aphasia affects the ability to produce language.

Wernicke's area is near the auditory processing region and is about language comprehension. A Wernicke's aphasia subject often generates a meaningless word salad (https://en.wikipedia.org/wiki/Word_salad).

The arcuate fasciculus is the collection of axons connecting Wernicke's with Broca's and so connects comprehension to language production.

Almost all stroke damage affects all three regions, but sometimes localized damage will affect one area or piece within it giving us clues to localized function.

Deaf people who are native speaker's of ASL use these same areas of the brain for production and comprehension and the communication between them. It appears that ASL and verbal language people use the same regions in the same ways. Whistled languages also use the same neurobiological centers in the brain.

Alexia: inability to read language.

Agraphia: inability to express language in writing.

Pictograph languages like Chinese have slightly different localization centers.

A fourth region of the brain, the basal ganglia, is used for motor control which is needed for both spoken and signed languages. Gesticulation appears to be tied into the motor output of language even with blind speakers which suggests that emotional inputs could be connected in the basal ganglia. Why?

The rest of the limbic system (see my notes on a video on this part of the brain: https://plus.google.com/104222466367230914966/posts/MW1pq8XzT33) is deeply connected to all these areas. Speech therapists will often recommend emotive singing for production aphasias since activating the emotional limbic system can sometimes help speaker's overcome damage in Broca's area. Tourette syndrome sometimes involves cursing uncontrollably which implies limbic inputs to language production. Talking can often help us feel better (the basis for psychotherapy is that there are deep connections between language and emotion). Stimulation of the brain can sometimes result in an emotional utterance; stimulate the same region in a rhesus monkey and it will give a call. A lot of the emotional input is directed to the right hemisphere where prosody is generally centered.

Other primates have very small "thickenings" where the Broca's and Wernicke's areas are located, but basically humans are the only species with these brain structures. There is evidence in lateralized facial expressions that some primates have a connection between their limbic systems and expressions.

Language acquisition and development.

Roger Brown and colleagues published a landmark study "A First Language: The Early Years" which detailed the early stages of language development in children. At 15-20 months of age, children start saying combinations of words which they've never heard before. There are days when we can learn 10 new words a day so that by 18 one's vocabulary is in the 60,000 word range.

BF Skinner, leader of the behaviorist school of psychology (which emphasized that reward and punishment shapes learning language and all other human behavior), and Noam Chomsky, a famous linguist, debated their views on language acquisition in the 1960s and 70s. Chomsky argued that kids can generalize the rules of language to acquire its generative structure despite imperfect, partial

examples from others. This "poverty of stimulus" argument suggests that there must be an innate faculty of language to explain the nature and speed with which language is acquired. There must be some prepared learning (a form of instinct in ethology; see https://plus.google.com/104222466367230914966/posts/YUnRBsoio66 for more on instinct, ethology, behaviorism and Skinner).

In addition to prepared learning, there appears to be statistical learning, that is, picking up rules from statistical patterns. There are studies showing that really young kids appear to notice statistical patterns in language.

"Any kid can learn any language." Even though different languages have different vowels and consonants. Between 1-3 months of age babies show roughly equal response to phonemes, the building blocks of sounds in a language. "As you begin to acquire your own [language], you start to be unable to hear the difference between other sounds".

By 3 months, kids can distinguish language from nonsense. Between 3-6 months, they start tuning in to vowel sounds. By 8-9 months, babbling starts and they can recognize word combinations. By 11 months they can generate vowels with consonants following soon thereafter.

In most cultures kids pick up language by observation. Only recently has language been explicitly taught to kids by presenting them with simple words and grammatical structures. But there is not yet good evidence that this helps them acquire language more effectively.

A second language learned after age 12 or so typically involves having a distinct accent. When people learn multiple languages before age 6, the languages are housed in overlapping regions of Broca's and Wernicke's. Languages learned later are housed in peripheral parts of those areas. Brain damage effects give the evidence: a language learned after age 6 might be affected while not other languages.

Sapolsky says it is widely believed in the field that "Languages are invented by kids". This surprises me: Klingon and Esperanto and Arabic (suggested by Richard Bulliet) and written Turkish are adult-designed languages. What about poets and other writers: don't they change the language? What about Public Speakers, Comedians, theater and TV, etc.? Don't each of us invent our language all the time? It isn't just kids. But is it really predominantly kids?

He cites evidence from Nicaraguan schools for the deaf where the kids invented their sign language. He suggests they invented Nicaraguan sign language, but is that a dialect or a whole language? Can languages get invented in whole cloth by kids? He said the Nicaraguan had teachers, so it was a joint project at least, right? He argues that older people rarely learn the new language being developed by kids. The transmission goes from older kids to younger ones. But didn't the teachers learn and adopt this new sign language?

Apparently it took 3 generations of students to devise a fully formed grammar and the first generation kids never fully adopted the more sophisticated form.

What are the dynamics of language development at the societal level?

Neurology of language acquisition

At 12-16 weeks of age in the fetus, evidence of lateralization in gene expression is found some of which appear to have a connection to families with language disorders. By 30 weeks, the structural asymmetry of the brain is observable. Metabolic lateralization takes several years after birth before

it is pronounced. Kids tend to myelinate their Wernicke's area about 9 months of age which is 3 months before they activate Broca's (about 1 year).

More on myelination in these notes:
https://plus.google.com/104222466367230914966/posts/MW1pq8XzT33

Judith Rich Harris emphasized the importance of peer instead of parental influences in children especially in language acquisition: kids grow up with the accent of the community around them and not the accent of their parents. Peers are extremely important in human social development. We seem to be wired to pick up language from our slightly older peers. Kids don't want to talk to their parents in the home language. Values in the local culture and language are very important: Tolstoy got upset when his nurse addressed him in an informal verb tense.

More about Judith Rich Harris' work is in my notes on Sapolsky's lecture "Behavioral Genetics I":
https://plus.google.com/104222466367230914966/posts/1vyQNjwemgX

In some languages, words for kinship relationships inherently involve the speaker's relationship. Why does this imply more intertwined social relations?

In egocentric languages, like English, directions are given relative to one's body ("here" or "there"). In many other languages, directions are a function of external landmarks. Aboriginal Australians use compass directions. Lera Boraditsky tells how this requires them to employ a system of dead reckoning to know where true South is at all times. For English speakers we need a compass! In English stories progress from left to right. In these aboriginal languages, stories begin in the East where the Sun rises. So the IQ test for placing tiles depends on the compass direction in which the player faces, not left or right!

Sapir-Whorf hypothesis: the nature of your thought is deeply shaped by the language you speak. Sapolsky talks about two Amazonian tribes whose language affects their ability to do math. The Pirahã have no words for ordinal or cardinal numbers: their counting is effectively 1, 2, many (http://www.sciencedaily.com/releases/2008/07/080714111940.htm); the Munduruku have a more complex system, but they only have cardinal numbers through 5 (https://en.wikipedia.org/wiki/Munduruku_people#Language). They are not a cognitively challenged people because they demonstrate an astute understanding of plants and other advanced capabilities needed to survive in the rainforests of the Amazon.

Since early in the course, Sapolsky has been emphasizing the importance of the ethology (animal behavior) perspective: "ethology is interviewing an animal in its own language". But now he observes that that soundbite must be taken very cautiously "because communication is so intertwined with mind, with values, with meaning". So it is very problematical "to interview an animal in its own language". Most people who speak multiple languages find that their emotive styles, expressivity, analyticalicity, etc., are different in their different languages.

How are thought and language related? Even though these examples suggest that language shapes our thinking. For words to have been invented in the first place requires an a priori thinking pattern that gets captured in the invented language. Is language a culture's capturing of important-to-them thinking patterns? But language is so complex and expressive does it allow the expansion of thought into domains that could not be accessed without language? Is language simply a tool for thought? Did thought just invent language to communicate its content? But the act of listening itself suggests language evokes thought.

Is the vicious circle between thought and language resolvable or are they like yin and yang

inextricably intertwined aspects of the human experience? Will the connection between thought and language always be tantalizingly ineffable? Or do we just need to invent the right word (or experiment) to communicate and clarify this relationship?

Animal communication

Some animals show evidence for semanticity (that discrete sounds can have meaning). For example, Vervant monkeys have discrete calls (sounds) for danger above or danger below. A word is a stable concept transmitted independent of emotion. These calls are words (Wiktionary: "The smallest unit of language which has a particular meaning and can be expressed by itself; the smallest discrete, meaningful unit of language."). Chickens also have unique calls for different kinds of dangers and some synonyms.

Multimodal communication (such as facial expression and calls) are observed, for example, by showing an incongruous facial expression and a call affecting a response in an observing animal.

Intentionality is also seen among animals: For example, vervant monkeys give alarm calls more readily when their relatives are around (squirrels too). He mentions that this assumes some theory of mind faculties (the recognition that there are other actors who can have different information than I do: see https://plus.google.com/104222466367230914966/posts/YUnRBsoio66).

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Can animals do language? Can they communicate with nearly the same facility as humans? Do they have enough cognitive capacity or are their smaller Wernicke's and Broca's areas inherently limiting of their ability to master anything like the complexity and richness of human language?

CJ Fearnley

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Robert Sapolsky on Language: this video starts an exploration of human language. It covers language as a biological behavior and looks at universals of the roughly 6,000 or so human languages estimated to exist. It also explores the neurobiology of language, the acquisition and development of language, and a comparison with animal communication. The exploration of animal communication allows Sapolsky to suggest an answer to the question of what is unique about human language in the animal kingdom.
A deficiency of Sapolsky's biology-focused presentation is that it misses the tenor of the importance of language in human culture. So I'm motivating my notes with Wade Davis' poetic definition of language: "A language, by definition, is a flash of the human spirit. It's a vehicle through which the soul of each particular culture comes into the material realm. Every language [...] is an old growth forest of the mind, a watershed of thought, an ecosystem of social, spiritual, political possibilities." That is even more poignant when you consider the fact that 90% of human languages are expected to go extinct this century. One of the most devastating signs of the rapacious side of globalization and religious missionaries.
http://www.youtube.com/watch?v=SIOQgY1tqrU
Behavior of Language. Universals across all human languages.
Semanticity: breakup of the continua of sounds into discrete "buckets" or units of meaning (words).
Embedded clauses: to express conditionals and circumstances.
Recursion (generativity): there is a potentially infinite number of combinations of words.
Displacement from emotions: ability to talk about the past and future; here and there; emotionally distant circumstances. Animal communication is overwhelmingly about their current emotional state.
Arbitrariness of language: words are not tied inextricably to their meaning: an arbitrary relationship between the signal and the message.
Meta-communication: the ability to talk about language.
Motherese or baby-talk: speaking to children in a higher-pitch voice, slow and clear, emphasizing melodic structure and vowels, repeating phrases, and focusing on the baby's face.
Is it about emotion or instruction? Pet talk is less slow and clear and doesn't focus on the face so it is not about instruction.

Is language more about cognition or the conceptual structure or about the generation and reception of sound (motor system)?
Gallaudet University in Washington, D.C. "was the first school for the advanced education of the deaf and hard of hearing in the world" (quote from https://en.wikipedia.org/wiki/Gallaudet_University).
Sign language suggests that language is mostly a cognitive faculty: it has many of the properties of language but uses different brain systems. Observe that at around 9 months, deaf and hearing babies begin to babble (practice fragments of whatever communication system they are exposed to).
Pathologies of the brain affect ASL speakers in similar ways to people using spoken language.
Prosody refers to aspects of language like tone and emphasis that are not encoded in grammar or vocabulary. Sign language also has prosody.
Deaf people where ASL is their native language, use their auditory cortex when they "listen" to signs. Is that really evidence that language is about the underlying cognitive structures?
There are regional accents, poetry, and puns in ASL even though it doesn't use lips, tongue, or ears.
If ASL is a second language it is "stored" in the same brain regions as a second spoken language.
Neurobiology of language.
How modular is the function of language in the brain? How separate is language from the rest of the cognitive functionality of the brain?
Williams syndrome is a genetic disease of very expressive, warm, affectionate, and verbally adept people whose IQs are near the borderline of intellectual disability. Their effective language skills imply that language is a separate faculty of the brain and not just one of many skills the brain develops. In addition, there are people with normal IQs who have various heritable language disabilities. Further supporting the idea that language is a modular function of the brain.
Nevertheless, people with Williams although fluent with words, do not communicate much meaning in their sentences. Moreover, many of the people with other heritable language disabilities are at the lower measures of intellectual function which suggests that language is probably connected in with general brain function (not so modular).
In conclusion, there is some evidence that language is a general function of the brain with quite a bit of localizations. Probably another "both/neither" rather than a clear cut situation.
The language function of the brain is lateralized (located on one side of the brain) to the left brain in 90% of people. In general, prosody is localized to the right brain in 90% of people.
The Wada test uses barbiturates to test for lateralized language and memory brain functions.
Modern brain imaging will probably obsolesce this test.
Aphasia is the general term for being unable to communicate effectively.
There are three main regions of the brain associated with language: Broca's area, Wernicke's area and the arcuate fasciculus.
Broca's area is the edge of the brain region that handles motoric function (moving your body). It is about moving your lips and larynx. A Broca's aphasia or production aphasia affects the ability to produce language.
Wernicke's area is near the auditory processing region and is about language comprehension. A Wernicke's aphasia subject often generates a meaningless word salad (https://en.wikipedia.org/wiki/Word_salad).
The arcuate fasciculus is the collection of axons connecting Wernicke's with Broca's and so connects comprehension to language production.
Almost all stroke damage affects all three regions, but sometimes localized damage will affect one area or piece within it giving us clues to localized function.

Deaf people who are native speaker's of ASL use these same areas of the brain for production and comprehension and the communication between them. It appears that ASL and verbal language people use the same regions in the same ways. Whistled languages also use the same neurobiological centers in the brain.
Alexia: inability to read language.
Agraphia: inability to express language in writing.
Pictograph languages like Chinese have slightly different localization centers.
A fourth region of the brain, the basal ganglia, is used for motor control which is needed for both spoken and signed languages. Gesticulation appears to be tied into the motor output of language even with blind speakers which suggests that emotional inputs could be connected in the basal ganglia. Why?
The rest of the limbic system (see my notes on a video on this part of the brain: https://plus.google.com/104222466367230.../posts/MW1pq8XzT33) is deeply connected to all these areas. Speech therapists will often recommend emotive singing for production aphasias since activating the emotional limbic system can sometimes help speaker's overcome damage in Broca's area. Tourette syndrome sometimes involves cursing uncontrollably which implies limbic inputs to language production. Talking can often help us feel better (the basis for psychotherapy is that there are deep connections between language and emotion). Stimulation of the brain can sometimes result in an emotional utterance; stimulate the same region in a rhesus monkey and it will give a call. A lot of the emotional input is directed to the right hemisphere where prosody is generally centered.
Other primates have very small "thickenings" where the Broca's and Wernicke's areas are located, but basically humans are the only species with these brain structures. There is evidence in lateralized facial expressions that some primates have a connection between their limbic systems and expressions.
Language acquisition and development.
Roger Brown and colleagues published a landmark study "A First Language: The Early Years" which detailed the early stages of language development in children. At 15-20 months of age, children start saying combinations of words which they've never heard before. There are days when we can learn 10 new words a day so that by 18 one's vocabulary is in the 60,000 word range.
BF Skinner, leader of the behaviorist school of psychology (which emphasized that reward and punishment shapes learning language and all other human behavior), and Noam Chomsky, a famous linguist, debated their views on language acquisition in the 1960s and 70s. Chomsky argued that kids can generalize the rules of language to acquire its generative structure despite imperfect, partial examples from others. This "poverty of stimulus" argument suggests that there must be an innate faculty of language to explain the nature and speed with which language is acquired. There must be some prepared learning (a form of instinct in ethology; see https://plus.google.com/104222466367230.../posts/YUnRBsoio66 for more on instinct, ethology, behaviorism and Skinner).
In addition to prepared learning, there appears to be statistical learning, that is, picking up rules from statistical patterns. There are studies showing that really young kids appear to notice statistical patterns in language.
"Any kid can learn any language." Even though different languages have different vowels and consonants. Between 1-3 months of age babies show roughly equal response to phonemes, the building blocks of sounds in a language. "As you begin to acquire your own [language], you start to be unable to hear the difference between other sounds".

By 3 months, kids can distinguish language from nonsense. Between 3-6 months, they start tuning in to vowel sounds. By 8-9 months, babbling starts and they can recognize word combinations. By 11 months they can generate vowels with consonants following soon thereafter.
In most cultures kids pick up language by observation. Only recently has language been explicitly taught to kids by presenting them with simple words and grammatical structures. But there is not yet good evidence that this helps them acquire language more effectively.
A second language learned after age 12 or so typically involves having a distinct accent. When people learn multiple languages before age 6, the languages are housed in overlapping regions of Broca's and Wernicke's. Languages learned later are housed in peripheral parts of those areas. Brain damage effects give the evidence: a language learned after age 6 might be affected while not other languages.
Sapolsky says it is widely believed in the field that "Languages are invented by kids". This surprises me: Klingon and Esperanto and Arabic (suggested by Richard Bulliet) and written Turkish are adult-designed languages. What about poets and other writers: don't they change the language? What about Public Speakers, Comedians, theater and TV, etc.? Don't each of us invent our language all the time? It isn't just kids. But is it really predominantly kids?
He cites evidence from Nicaraguan schools for the deaf where the kids invented their sign language. He suggests they invented Nicaraguan sign language, but is that a dialect or a whole language? Can languages get invented in whole cloth by kids? He said the Nicaraguan had teachers, so it was a joint project at least, right? He argues that older people rarely learn the new language being developed by kids. The transmission goes from older kids to younger ones. But didn't the teachers learn and adopt this new sign language?
Apparently it took 3 generations of students to devise a fully formed grammar and the first generation kids never fully adopted the more sophisticated form.
What are the dynamics of language development at the societal level?
Neurology of language acquisition
At 12-16 weeks of age in the fetus, evidence of lateralization in gene expression is found some of which appear to have a connection to families with language disorders. By 30 weeks, the structural asymmetry of the brain is observable. Metabolic lateralization takes several years after birth before it is pronounced. Kids tend to myelinate their Wernicke's area about 9 months of age which is 3 months before they activate Broca's (about 1 year).
More on myelination in these notes:
https://plus.google.com/104222466367230.../posts/MW1pq8XzT33
Judith Rich Harris emphasized the importance of peer instead of parental influences in children especially in language acquisition: kids grow up with the accent of the community around them and not the accent of their parents. Peers are extremely important in human social development. We seem to be wired to pick up language from our slightly older peers. Kids don't want to talk to their parents in the home language. Values in the local culture and language are very important: Tolstoy got upset when his nurse addressed him in an informal verb tense.
More about Judith Rich Harris' work is in my notes on Sapolsky's lecture "Behavioral Genetics I":
https://plus.google.com/104222466367230.../posts/1vyQNjwemgX
In some languages, words for kinship relationships inherently involve the speaker's relationship. Why does this imply more intertwined social relations?
In egocentric languages, like English, directions are given relative to one's body ("here" or "there"). In many other languages, directions are a function of external landmarks. Aboriginal Australians use compass directions. Lera Boraditsky tells how this requires them to employ a system of dead

reckoning to know where true South is at all times. For English speakers we need a compass! In English stories progress from left to right. In these aboriginal languages, stories begin in the East where the Sun rises. So the IQ test for placing tiles depends on the compass direction in which the player faces, not left or right!

Sapir-Whorf hypothesis: the nature of your thought is deeply shaped by the language you speak. Sapolsky talks about two Amazonian tribes whose language affects their ability to do math. The Pirahã have no words for ordinal or cardinal numbers: their counting is effectively 1, 2, many (http://www.sciencedaily.com/rele.../2008/07/080714111940.htm); the Munduruku have a more complex system, but they only have cardinal numbers through 5 (https://en.wikipedia.org/wiki/Munduruku_people#Language). They are not a cognitively challenged people because they demonstrate an astute understanding of plants and other advanced capabilities needed to survive in the rainforests of the Amazon.

Since early in the course, Sapolsky has been emphasizing the importance of the ethology (animal behavior) perspective: "ethology is interviewing an animal in its own language". But now he observes that that soundbite must be taken very cautiously "because communication is so intertwined with mind, with values, with meaning". So it is very problematical "to interview an animal in its own language". Most people who speak multiple languages find that their emotive styles, expressivity, analyticalicity, etc., are different in their different languages.

How are thought and language related? Even though these examples suggest that language shapes our thinking. For words to have been invented in the first place requires an a priori thinking pattern that gets captured in the invented language. Is language a culture's capturing of important-to-them thinking patterns? But language is so complex and expressive does it allow the expansion of thought into domains that could not be accessed without language? Is language simply a tool for thought? Did thought just invent language to communicate its content? But the act of listening itself suggests language evokes thought.

Is the vicious circle between thought and language resolvable or are they like yin and yang inextricably intertwined aspects of the human experience? Will the connection between thought and language always be tantalizingly ineffable? Or do we just need to invent the right word (or experiment) to communicate and clarify this relationship?

Animal communication

Some animals show evidence for semanticity (that discrete sounds can have meaning). For example, Vervant monkeys have discrete calls (sounds) for danger above or danger below. A word is a stable concept transmitted independent of emotion. These calls are words (Wiktionary: "The smallest unit of language which has a particular meaning and can be expressed by itself; the smallest discrete, meaningful unit of language."). Chickens also have unique calls for different kinds of dangers and some synonyms.

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The Biology of Mental Illness: Schizophrenia and Depression

Details

What is the meaning and what are the implications of our modern biological understanding of schizophrenia and depression? What we can learn about our own minds from the clues provided when our biology goes seriously wrong as in these two serious yet poorly understood forms of mental illness?

Does better understanding how the biology of our minds can go wrong help us better understand our own minds?

What is schizophrenia? From the biology of schizophrenia (that is from the nature of the behavior itself, the chemistry of neurotransmitters, the neuroanatomy of the brain, genetic and environmental factors, etc), what can we learn about those afflicted and about our own minds? Is there a clear distinction between mental illness and mental health? What comprises the difference?

What is major depression? What can we learn about depressed people and our own minds from a better understanding of the biology of depression?

What are the implications of the role of major stress in triggering the onset of both schizophrenia and depression?

What are the benefits and challenges in trying to understand the biology of depression and schizophrenia?

These questions occur to me as I reflect on the following two sobering Robert Sapolsky videos exploring the biological depths of schizophrenia and depression.

• 1h 40m Robert Sapolsky video on the biology of schizophrenia (skip past the first 23 minutes which concern the topic of language). Read Jon Dakin's notes on Sapolsky's lecture on schizophrenia (http://robertsapolskyrocks.weebly.com/schizophrenia.html). Read CJ Fearnley's notes on Sapolsky's lecture on schizophrenia (https://www.facebook.com/cj.fearnley/posts/10206876666570820).

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• This 15 minute video of Elyn Saks so poignantly describes schizophrenia, that it is worth watching as well:

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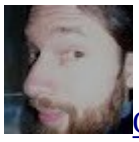
Events in Philadelphia, PA

 CJ Fearnley - 2015-08-30 09:46:12-0400 - Updated: 2015-08-30 09:46:12-0400
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http://www.meetup.com/thinkingsociety/events/223325873/

The Biology of Mental Illness: Schizophrenia and Depression

CJ Fearnley

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The Biology of Mental Illness: Schizophrenia and Depression (Repeat)

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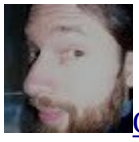
This is a repeat of the September 12th meetup (https://www.meetup.com/thinkingsociety/events/223325873/).

 CJ Fearnley - 2015-08-24 20:18:40-0400 - Updated: 2015-08-24 20:18:40-0400
Robert Sapolsky explores our biological understanding of Schizophrenia in this 1h 40 minute video (except the first 23 minutes which concludes a discussion on language). These notes summarize just the material after the topic switches to schizophrenia.

Schizophrenia is a technical term that has nothing to do with ordinary everyday usage. Schizophrenia is a disease of disordered thought, inappropriate emotion, inappropriate attribution of things. Schizophrenia is heterogeneous: the disordered thoughts manifest differently in different individuals and the biology almost certainly has multiple causative pathways.

http://y2u.be/nEnklxGAmak

Some of the different forms of schizophrenia include paranoid schizophrenia, a thought disorder of persecution, catatonic schizophrenia where a person is immobile for long periods of time, and schizoaffective disorders which is a mixture of depression and schizophrenia.

Schizophrenia is a disease of cognitive abnormalities: abnormal sequential thought or loose associations (tangential thinking). In schizophrenia, sequential thinking is greatly impaired. For example, in a sentence about boxers they confusedly slip back and forth between thinking about the dog and the sport. Schizophrenics follow loose associations like these and lose the thread of a conversation.

Schizophrenia entails a consistent problem with abstraction: they cannot fathom which level of abstraction (concrete, second hand story, parable) is involved in a story. They always interpret things far more concretely than would be appropriate. So in the test "what is common among an apple, an orange, and a banana?" Schizophrenics might say they are all multisyllabic or that each has letters forming closed loops. They are not able to step back and see the abstraction "fruit". Other examples: if you ask "what's on your mind", they might answer "hair". Or if asked "can I take your picture?", they say "I have no picture to give you". They can't understand parables which involve metaphor ("birds of a feather flock together", "a rolling stone gathers no moss", "loose lips sink ships").

I often err on the side of being too concrete, don't we all? Do we all have at least a mild form of Schizophrenia? Sapolsky emphasizes that it is a complex and subtle diagnosis. Is schizophrenia characterized by being unable to get the thread of abstraction or metaphor even on the third or forth try? But Sapolsky suggests that they have clear-headed days and psychotic breaks. So their performance is probably uneven. As is mine!

Schizophrenia entails delusions and beliefs in things that cannot be, belief in having participated in historical events that cannot be. Also paranoia: what apples, oranges and bananas have in common is that they are all wired for sound. "If the world is making so little sense to you, it is a world that is very threatening."

Schizophrenia entails structured hallucinations "hearing voices". Most hallucinations are auditory, but they are structured as voices and not random sounds. The most common voice heard is of Jesus followed by Satan followed by the head of state.

Schizophrenia entails social withdrawal, abnormal social affiliation, ostracized, apathy (there is some damping of the autonomic nervous system; see these notes for more about the ANS: https://plus.google.com/104222466367230914966/posts/EKwuB88TvAf).

I feel I too have a lack of social affiliation, but maybe mine isn't abnormal? I was ostracized in school. Some of these symptoms are almost universal. How can we understand the subtleties of this affliction? How do we integrate the whole of what Sapolsky says without erring by misinterpreting pieces of his description?

Schizophrenia has often been associated with violence, but their rates of committing violence are lower than in society in general except for self-mutilation, most often involving damaging their genitals. Wikipedia seems to disagree: https://en.wikipedia.org/wiki/Schizophrenia#Violence

In the 60s some argued that schizophrenia was a blessing. Sapolsky pushes back hard: "All you

need to do is be schizophrenic or know someone who is or have a family member and you will see there are no hidden blessings. This is not a disease of hidden compensations and more insight into the world. This is one of the most horrific ways that biology can go wrong. And one of the best demonstrations of it is that half of Schizophrenics attempt suicide."

During periods of remission, the chance of suicide increases suggesting that "the more often you have periods of where you are clear headed enough to see what your life is like the rest of the time, the more likely you are to try to kill yourself."

More and more people are thinking the core of the disease is disordered thinking.

Schizophrenia tends to manifest in late adolescence or early adulthood (it never(?) emerges after age 30). Schizophrenia is centered in the frontal cortex which is only just finishing development at this time. The first "psychotic break" tends to be associated with major stress. It seems that a fragile frontal cortex suffering stress can breakdown. With aging the positive symptoms (loose associations, hallucinations) lessen and the negative symptoms come to predominate (withdrawal, etc.).

Schizophrenia is not associated with gender or class, but once the affliction manifests there tends to be a downward economic spiral for those affected. "The majority of people living on streets in this country are individuals with schizophrenia."

Because schizophrenia is fundamentally a disease where everyone else thinks you're not thinking normally, there is a slippery slope to all sorts of hidden agendas and abuse. To "get rid of someone", the powerful can often simply ascribe a diagnosis of schizophrenia.

Can we avoid falling into the strangleholds of ideology?

One approach is to look at schizophrenics in different cultures. Sapolsky tells a story of Maasai woman with schizophrenia 25 years ago. In their culture men spend 10 years around puberty in warrior clans "pillaging the neighbors and getting killed in return". They settle down around age 25 taking their first wife ("typically a 13 year old"). Until recently their life expectancy was 30. "This is a culture where people believe in all sorts of things that we would view as being paranormal." So, poignantly, Sapolsky reports the real problem with the Maasai schizophrenic: "She hears voices at the wrong time."

Can we accept and try to understand individuals whose alternative thinking is just different from ours let alone those whose thinking is clinically disordered?

The Maasai are no more tolerant of mental illness than we are.

Can we learn to tolerate others with mental illness and various forms of disordered thinking? How could we even try to do that?

There are a large variety of ways to be "normal" in any culture, it takes a lot of understanding of the diversity of "normal" before one can competently make a diagnosis that someone's thinking is not normal or schizophrenic. There have been many creative artists with schizophrenia, but Sapolsky argues that it wasn't schizophrenia that made their creativity it is what destroyed their careers.

How could I prove to a psychologist that my brain works normally? There doesn't appear to be a definitive test for schizophrenia. How do we deal with such a fuzzy ailment, with its difficult diagnosis, and imperfect treatments with significant side effects?

The dopamine hypothesis: the dominant model for explaining schizophrenia: an excess of dopamine in the synapses in the frontal cortex to help manage executive function. Dopamine breakdown products (in bloodstream and cerebospinal fluid) are elevated in schizophrenics. All of the classic drugs (neuroleptics or antipsychotics such as Haldol, Thorazine, etc.) that help with schizophrenia block dopamine receptors. When schizophrenics receive dopamine or drugs that activate dopamine receptors, their symptoms get worse. Postmortem, there are elevated numbers of dopamine receptors in the frontal cortex.

Dopamine is also involved in fine motor control in the substantia nigra (involved with the basal ganglia). These dopaminergic neurons degrade in old age giving the characteristic hand tremors of the elderly and they die in Parkinson's disease. L-DOPA is a precursor to dopamine and can treat Parkinson's and Encephalitis lethargica. Since L-DOPA is delivered via the bloodstream, it doesn't just go to the substantia nigra where it is needed. The elevated L-DOPA leads to elevated dopamine in the rest of the brain which can cause psychotic breaks. Amphetamines cause a rapid dumping of dopamine resulting in a transient schizophrenic episode. Overmedicated schizophrenics develop the symptoms of Parkinson's. Tardive dyskinesia refers to individuals who look like they have Parkinson's. But there is at least one antipsychotic drug that increases dopamine and helps schizophrenics.

Seratonin is chemically structured almost identically to many hallucinogens such as LSD & mescaline, & psilocybin. Each fits into and activates seratonin receptors. The hallucinogens cause signaling in the post-synaptic neurons that typically receive a signal from the pre-synaptic neuron but in this case they are "hearing voices". So seratonin has been implicated in schizophrenic hallucinations.

Glutamate which is the chief excitatory neurotransmitter in the vertebrate nervous system has been implicated in schizophrenia: activation of some glutamate receptors from PCP (Phencyclidine or angel dust) resembles symptoms of schizophrenia. This has led many to argue for a glutamate role in schizophrenia. One study suggested PCP leads to increased receptors for seratonin.

Brain metabolism during hallucination is widespread except for the primary sensory cortexes (the visual and auditory cortexes) --- which is similar to the brain activity during dreaming. When schizophrenics are given memory tasks and metabolism in the hippocampus (involved in learning & memory and turning off the stress response) does not increase as much as in other individuals.

Structural abnormalities in the brains of schizophrenics. First he explains the challenges of identifying these: even rapid autopsy teams take 30 minutes to extract a brain for analysis after death, the brains of elderly schizophrenics may manifest symptoms of malnutrition and other effects of the disease and its treatment instead of evidence of schizophrenia itself. Brain imaging helps solve some of these problems.

In schizophrenics there is enlargement of the ventricles in the brain where cerebospinal fluid (CSF) is produced which in turn results in compression of the cortex, particularly the frontal cortex. Schizophrenics have fewer hippocampal neurons and some of them are misaligned. Sapolsky says "this is not going to make for a whole lot of solid sequential thought if you have neurons pointing the wrong direction." In the frontal cortex, some studies have counted fewer neurons and fewer glial cells (non-neural cells in the brain). Reelin, a protein involved in cortical maturation, is deficient in schizophrenics (another clue that it is a disease of the maturation of the frontal cortex between late adolescence and age 25). There is atrophy of the thalamus.

What about the genetics?

Seymour Kety's adoption studies are discussed in these notes
https://plus.google.com/u/0/104222466367230914966/posts/1vyQNjwemgX

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If someone has schizophrenia, their identical twin has a 50% chance of also being afflicted; a full sibling will have a 25% chance; a half sibling 12%; a random person 1-2%. So there is a large genetic component for schizophrenia. Other relatives of schizophrenics also experience a higher rate of "mild versions of thought disorder".

In the 1980s the first genetic markers for schizophrenia were identified. But each study identified a different marker and none have been replicated.

The modern approach of studying actual genes has identified some correlates. Some variants of the enzyme that degrades dopamine have a small statistical correlation to schizophrenia. Sapolsky reports that three recent studies have identified genes affecting the MHC or major histocompatibility complex (which is involved in pheromones and cell signaling and the immune system). The DISC1 (Disrupted in schizophrenia 1) is a gene that has been implicated, but there hasn't been much progress in understanding how and why.

Many studies suggest that although some genes may not be different in schizophrenics, copy number variations (different numbers of copies of a gene) is frequently different in schizophrenics. But again there is little consistency in the findings. Sapolsky summarizes: "It's not a disease it's a whole bunch of heterogeneous ones and there's going to be all sorts of different genetic components to it."

"People who were fetuses during the Dutch hunger winter have a higher than expected rate of schizophrenia." Same with the Chinese famine of 1959-1961. Birth trauma, a brief hypoxia (reduced oxygen), are all associated with an increased incidence of schizophrenia. Rats exposed prenatally to high levels of glucocorticoids have elevated dopamine levels in their frontal cortex. Monochorionic twins (identical twins that share the same placenta) are more correlated with schizophrenia than dichorionic twins. Because of the complex of correlates to stress, Sapolsky opines that more stress leads to more schizophrenia.

In the 1950s, the learned opinion of psychologists was that abnormal parenting (or mothering: those were sexist times) was considered to be the cause of schizophrenia. This was called schizophrenic mothering. Generally they identified "conflicting emotional messages" or "conflicting double bind" or "raising kids with distorted contradictory fragmented emotional demands" as leading to schizophrenia. In the early 1950s the first neuroleptics were used in treating schizophrenia and the view that it was caused by parenting not only waned but resulted in an outpouring of regret from many of those psychologists realizing "my god, what have we done" as biochemistry demonstrated that pathological psychology isn't necessarily caused by bad people.

In families of schizophrenics there is a somewhat higher correlation for "communication deviance" meaning a fragmented, telescoped, broken phrase style of communication. When schizophrenics explain a Rorschach blot to their close family, there family members can often can pick out the correct pattern (control families cannot; family members can only pick out patterns correctly within the family). One possible explanation is that these families have compensated for the thought disorder.

Schizophrenics have on average been exposed to certain viruses in the third trimester of pregnancy (perinatal stress). They have more retroviral DNA in their genome than the average person. They have an elevated history of neonatal infections.

Toxoplasma gondii reproduces in cats. Infected rodents infected with toxoplasma gondii like the smell of cats increasing their chance of predation. A protozoan that changes behavior of a species in support of its life cycle. 30-50% of the global human population has been infected. Humans infected have a higher rate of mild neuropsychological disinhibition, that is, some impact on frontal cortex function. Infected humans have a higher than expected rate of car accidents, higher rates of suicide, higher rates of a certain degree of impusivity. "Toxoplasma exposure increases the risk of schizophrenia." There is evidence of increased infection of mothers during pregnancy, increased levels of antibodies to Toxoplasma, increased association between cats and schizophrenia. Many of the findings have been replicated.

Perhaps the MHC genetic effects relate to these kinds of infections, but no one knows.

How do you put together adolescent stress with prenatal infections with enlarged ventricles with some genetic abnormalities? There is no integrated model of schizophrenia yet.

The evolution of schizophrenia. In the animal world you see depression and meloncholia, but if they had the kind of disordered thinking of schizophrenia they'd probably become prey pretty quickly. So there are no animal precedents for schizophrenia.

Schizophrenics have much less reproductive success than their unaffected siblings, yet it persists at the 1-2% rate in every culture. Historical records suggest schizophrenia has been extant for a long time.

Are there circumstances in which schizophrenia is adaptive, or advantageous and increase reproductive success? Schizophrenics tend to have lower rates of lung and esophageal cancers. There are some suggestions that mild forms of schizophrenia could have adaptive qualities. But he omits the details.

Did Sapolsky convince you that schizophrenia is a disease? What are the implications of treating it as a disease? Is it just to get more funding for research? Can the disease status help us help schizophrenics? Can it help us understand how to help homeless schizophrenics? Does a disease status help us remove the stigma of schizophrenia? As non-psychologists, can we help them be the best they can be? Do you have more compassion for the schizophrenic after watching the video? What can we do with what we learned from Sapolsky?

CJ Fearnley

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Robert Sapolsky explores our biological understanding of Schizophrenia in this 1h 40 minute video (except the first 23 minutes which concludes a discussion on language). These notes summarize just the material after the topic switches to schizophrenia.

Schizophrenia is a technical term that has nothing to do with ordinary everyday usage.

Schizophrenia is a disease of disordered thought, inappropriate emotion, inappropriate attribution of things. Schizophrenia is heterogeneous: the disordered thoughts manifest differently in different individuals and the biology almost certainly has multiple causative pathways.

http://y2u.be/nEnklxGAmak

Some of the different forms of schizophrenia include paranoid schizophrenia, a thought disorder of persecution, catatonic schizophrenia where a person is immobile for long periods of time, and schizoaffective disorders which is a mixture of depression and schizophrenia.

Schizophrenia is a disease of cognitive abnormalities: abnormal sequential thought or loose associations (tangential thinking). In schizophrenia, sequential thinking is greatly impaired. For example, in a sentence about boxers they confusedly slip back and forth between thinking about the dog and the sport. Schizophrenics follow loose associations like these and lose the thread of a conversation.

Schizophrenia entails a consistent problem with abstraction: they cannot fathom which level of abstraction (concrete, second hand story, parable) is involved in a story. They always interpret things far more concretely than would be appropriate. So in the test "what is common among an apple, an orange, and a banana?" Schizophrenics might say they are all multisyllabic or that each has letters forming closed loops. They are not able to step back and see the abstraction "fruit". Other examples: if you ask "what's on your mind", they might answer "hair". Or if asked "can I take your picture?", they say "I have no picture to give you". They can't understand parables which involve metaphor ("birds of a feather flock together", "a rolling stone gathers no moss", "loose lips sink ships").

I often err on the side of being too concrete, don't we all? Do we all have at least a mild form of Schizophrenia? Sapolsky emphasizes that it is a complex and subtle diagnosis. Is schizophrenia characterized by being unable to get the thread of abstraction or metaphor even on the third or forth try? But Sapolsky suggests that they have clear-headed days and psychotic breaks. So their performance is probably uneven. As is mine!

Schizophrenia entails delusions and beliefs in things that cannot be, belief in having participated in historical events that cannot be. Also paranoia: what apples, oranges and bananas have in common is that they are all wired for sound. "If the world is making so little sense to you, it is a world that is very threatening."

Schizophrenia entails structured hallucinations "hearing voices". Most hallucinations are auditory, but they are structured as voices and not random sounds. The most common voice heard is of Jesus followed by Satan followed by the head of state.

Schizophrenia entails social withdrawal, abnormal social affiliation, ostracized, apathy (there is some damping of the autonomic nervous system; see these notes for more about the ANS: https://plus.google.com/104222466367230.../posts/EKwuB88TvAf).

I feel I too have a lack of social affiliation, but maybe mine isn't abnormal? I was ostracized in school. Some of these symptoms are almost universal. How can we understand the subtleties of this affliction? How do we integrate the whole of what Sapolsky says without erring by misinterpreting pieces of his description?

Schizophrenia has often been associated with violence, but their rates of committing violence are lower than in society in general except for self-mutilation, most often involving damaging their

genitals. Wikipedia seems to disagree: https://en.wikipedia.org/wiki/Schizophrenia#Violence
In the 60s some argued that schizophrenia was a blessing. Sapolsky pushes back hard: "All you need to do is be schizophrenic or know someone who is or have a family member and you will see there are no hidden blessings. This is not a disease of hidden compensations and more insight into the world. This is one of the most horrific ways that biology can go wrong. And one of the best demonstrations of it is that half of Schizophrenics attempt suicide."
During periods of remission, the chance of suicide increases suggesting that "the more often you have periods of where you are clear headed enough to see what your life is like the rest of the time, the more likely you are to try to kill yourself."
More and more people are thinking the core of the disease is disordered thinking.
Schizophrenia tends to manifest in late adolescence or early adulthood (it never(?) emerges after age 30). Schizophrenia is centered in the frontal cortex which is only just finishing development at this time. The first "psychotic break" tends to be associated with major stress. It seems that a fragile frontal cortex suffering stress can breakdown. With aging the positive symptoms (loose associations, hallucinations) lessen and the negative symptoms come to predominate (withdrawal, etc.).
Schizophrenia is not associated with gender or class, but once the affliction manifests there tends to be a downward economic spiral for those affected. "The majority of people living on streets in this country are individuals with schizophrenia."
Because schizophrenia is fundamentally a disease where everyone else thinks you're not thinking normally, there is a slippery slope to all sorts of hidden agendas and abuse. To "get rid of someone", the powerful can often simply ascribe a diagnosis of schizophrenia.
Can we avoid falling into the strangleholds of ideology?
One approach is to look at schizophrenics in different cultures. Sapolsky tells a story of Maasai woman with schizophrenia 25 years ago. In their culture men spend 10 years around puberty in warrior clans "pillaging the neighbors and getting killed in return". They settle down around age 25 taking their first wife ("typically a 13 year old"). Until recently their life expectancy was 30. "This is a culture where people believe in all sorts of things that we would view as being paranormal." So, poignantly, Sapolsky reports the real problem with the Maasai schizophrenic: "She hears voices at the wrong time."
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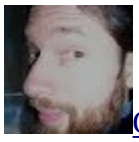 CJ Fearnley - 2015-09-09 17:33:23-0400 - Updated: 2015-09-09 17:33:23-0400
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Elyn's delusions include the sensation that she has killed hundreds of thousands of people with her thoughts. Yikes!

"The schizophrenic mind is not split but shattered"

OMG: up to 20 hours in mechanical restraints: arms and legs tied down

Does this still go on?

Every week in the US 1-3 people die in restraints. Are we so barbaric?

Why would we strap people to beds for hours?

"I don't think force is effective as treatment and I think using force is a terrible thing to do to another human person with a terrible illness."

Her experiment testing the hypothesis that "the less medicine, the less defective". Evidently

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We all deserve a life of meaning and depth

"There are not schizophrenics. There are people with schizophrenia

Schizophrenics are people not diagnoses!

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"We must stop criminalizing mental illness"

"The humanity we all share is more important than the mental illness we may not"

We all want to work and to love.

http://www.ted.com/talks/elyn_saks_seeing_mental_illness

A tale of mental illness — from the inside

CJ Fearnley

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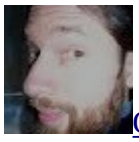 CJ Fearnley - 2015-09-07 13:11:36-0400 - Updated: 2015-09-07 13:11:36-0400
In this 52 minute lecture Robert Sapolsky argues that "depression is the worst disease you can get". About 15% of us will experience a major depression in our lives. The World Health Organization (WHO) reports that depression is the #4 cause of disability on this planet and it is projected to reach #2 by 2025 after obesity/diabetes related diseases. The talk surveys the biology and psychology of depression.

His conclusion is sobering: A major depression is as real a biological disorder as is diabetes. But people tend not to talk about psychiatric disorders. Depression is one of the hardest diseases for people to admit to despite the fact that it is so widespread.

http://www.youtube.com/watch?v=NOAgplgTxfc

"Depression is absolutely crippling" and its widely pervasive.

Sapolsky is mainly referring to major depression not the kind of being bummed for a few days after getting bad news. More serious legitimate bad news such as the death of a loved one, loss of a job, etc., can lead to serious impairment for several weeks until you come out the other end. The most serious forms of depression involve not coming out the other end after months of serious impairment. Once suffering a major depression, the triggers for a recurrence can be much less serious than the first time.

Sapolsky's one sentence definition of a major depression "it's a biochemical disorder with a genetic component with early experience influences where somebody can't appreciate sunsets." It is surprising that since humans can derive pleasure and satisfaction out of terrible experiences like dying of cancer, "what could possibly be worse than a disease whose defining symptom is the inability to feel pleasure"?

Symptoms

Anhedonia: the inability to feel pleasure

The grief and guilt in a major depression can become so severe that they take on a delusional quality.

Self-injury is a common symptom of major depression, suicide being the extreme form.

Psychomotor retardation: everything is exhausting; even thinking about things can be exhausting resulting in a paralyzed state. This is not the stage of the disease when self-injury is a risk. Usually self-injuries happen after one starts to have more energy to actually do something about how horrible one is feeling.

Vegetative symptoms show that the bodies of depressives work differently. Sleep: depressives often wake up early and their whole structure of sleeping is out of wack; appetite decrease (for most of us we eat more when we are somewhat depressed); stress response: overactivation of the sympathetic

nervous systems with elevated adreneline and glucocorticoids (the body is acting like it was just gored by an elephant but it can't get out of bed).

Rhythmic patterns to a person's depression are common

Seasonal affective disorders (only getting depressed in the Winter)

Neurotransmitters are the chemical signals neurons use to communicate with other neurons across the space between them (the synapse). There are probably hundreds of neurotransmitters, but only a few are implicated in depression.

MAO inhibitors were the first antidepressants in the 1960s. They suppress the activity of enzymes that break down norepinephrine (and epinephrine, serotonin, and dopamine). Their antidepressant activity suggests that the problem in depression is too little norepinephrine to support synaptic function.

Tricyclic antidepressants block the reabsorption (recycling) of norepinephrine further supporting the theory of inadequate neurotransmitter function in depression (the norepinephrine hypothesis).

Reserpine was used for treating high blood pressure because it depletes norepinephrine with the side effect of causing depression.

Norepinephrine affects the pleasure circuit in the brain (known from both rat studies and unanesthetized brain surgery patients). So shortages of norepinephrine (at least in that part of the brain) can cause anhedonia.

But the antidepressive effects of these treatments took weeks to have an impact. Then dopamine was discovered to be even more important than norepinephrine in the pleasure pathway.

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https://plus.google.com/104222466367230914966/posts/MW1pq8XzT33

The cortex is able to control the limbic system through abstract thoughts and impressions. At some level depression is the cortex whispering to the rest of the brain that you are feeling as bad as if assaulted by a predator. "On a very simplistic level, what a depression is is the cortex having too many sad thoughts and getting the rest of the brain to go along with it."

A cingulotomy is a surgical procedure to sever a pathway between the cortex and the limbic system and people get less depressed (it is only used in desperate cases after all other therapies and their combinations have been tried and the person continues to harm themselves every few months).

Hormones

Thyroid hormones are about maintaining metabolism. A shortage of thyroid hormones (hypothyroidism) causes a major depression. Hashimoto's disease is an auto-immune disease attacking the thyroid and results in depression. 20% of major depressions are undiagnosed hypothyroidism instead.

Women suffer major depression at about twice the rate of men. The most likely times women suffer depression are after giving birth (postpartem depression), around the time of their period, and around the time of menopause. All of these situations are characterized by major biological changes. Also, on the average women ruminate more about their emotions. Estrogen and progesterone, and most likely their ratio, appear to be the hormones that can knock the rest of the brain out of wack causing a depression in women.

Stress hormones. Adreneline is vastly overrated according to Sapolsky. He thinks the glucocorticoids are much more important (he's devoted the last 30 years of his life to them), Glucocorticoids come out of the adrenal glands. Hydrocortisone or cortisol in humans. They are secreted when we are under stress.

Half the people with depression have elevated glucocorticoid levels. People exposed to elevated glucocorticoid levels are at a greater risk of a major depression. Most people come out of stress induced depressions with no long term impact. But after the third or fourth stress induced depression, the person is at a higher risk of suffering further major depressions without major stressors triggering them.

Cushing's syndrome or hypercortisolism is a disease where people secrete lots of glucocorticoids. People with Cushing's fall into depressions. Lengthly treatments with glucocorticoids depletes the brain of dopamine and leads to more major depressions.

Only 30-40% of depressives can be treated with this biological knowledge. The psychology of depression, Sapolsky argues, is essential for helping the rest of depressives.

Sigmund Freud asked what is the difference between mourning (being depressed for a few weeks) and melancholia (major depression)? Freud suggested that melancholia is roiling in the ambiguities of love and loss being unable to put the negative feelings in the background. Sound bite: "depression is aggression turned inward".

Experimental psychology suggests that as you feel more stress and activate the stress response system for longer times, the chance of suffering stress-related diseases including major depressions is surprising small UNLESS you don't have outlets for the frustration caused by the stressor, and/or you feel you have no control over what's happening, and/or any predictability for the stressor, and/or you don't have anyone with a shoulder to cry on. Depression is pathological extremes of these factors that challenge our ability to cope with stress. The cognitive psychology soundbite for depression is "learned helplessness". Depression is making what's wrong into the whole world: feeling that you have no control and that it is hopeless and I'm helpless to do anything about it.

One of the most reliable findings in the epidemiology of depression is lose a parent to death before

age 10, and the risk of a major depression is higher. Kids at that age are learning about cause and effect and the loss teaches them that there are awful things that you cannot control bringing you one step closer to the cliff of learned helplessness that is a major depression.

Stress is the intersection of the modern biological understanding of depression with the psychology.

Depression has some degree of heritability implying that it is a genetic disorder. Depression runs in families. Identical twins have a 50% chance of having a depression if their twin has one. Full siblings have a 25% chance. Person off the street only has a 2% incidence of depression. So there is a genetic component. But since identical twins have a 50% chance of NOT getting a depression if their twin does, that implies that genes are no more important than other factors.

Genes for depression are not about inevitability, they are about vulnerability. A major study showed that having the bad version of a serotonin regulating gene means you are no more likely to have a major depression unless you also have a history of major life stressors. The effect is 30-fold greater. "This is a gene that is relevant to how readily we pick ourselves up after life has dumped us on our rear ends. How readily we recover from stressors." Glucocorticoids regulate the function of this gene.

Conclusion: A major depression is as real a biological disorder as is diabetes. But people tend not to talk about psychiatric disorders. Depression is one of the hardest diseases for people to admit to despite the fact that it is so widespread.

CJ Fearnley

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In this 52 minute lecture Robert Sapolsky argues that "depression is the worst disease you can get". About 15% of us will experience a major depression in our lives. The World Health Organization (WHO) reports that depression is the #4 cause of disability on this planet and it is projected to reach #2 by 2025 after obesity/diabetes related diseases. The talk surveys the biology and psychology of depression.
His conclusion is sobering: A major depression is as real a biological disorder as is diabetes. But people tend not to talk about psychiatric disorders. Depression is one of the hardest diseases for people to admit to despite the fact that it is so widespread.
http://www.youtube.com/watch?v=NOAgplgTxfc
"Depression is absolutely crippling" and its widely pervasive.
Sapolsky is mainly referring to major depression not the kind of being bummed for a few days after getting bad news. More serious legitimate bad news such as the death of a loved one, loss of a job, etc., can lead to serious impairment for several weeks until you come out the other end. The most serious forms of depression involve not coming out the other end after months of serious impairment. Once suffering a major depression, the triggers for a recurrence can be much less serious than the first time.
Sapolsky's one sentence definition of a major depression "it's a biochemical disorder with a genetic component with early experience influences where somebody can't appreciate sunsets." It is surprising that since humans can derive pleasure and satisfaction out of terrible experiences like

dying of cancer, "what could possibly be worse than a disease whose defining symptom is the inability to feel pleasure"?

Symptoms

Anhedonia: the inability to feel pleasure

The grief and guilt in a major depression can become so severe that they take on a delusional quality.

Self-injury is a common symptom of major depression, suicide being the extreme form.

Psychomotor retardation: everything is exhausting; even thinking about things can be exhausting resulting in a paralyzed state. This is not the stage of the disease when self-injury is a risk. Usually self-injuries happen after one starts to have more energy to actually do something about how horrible one is feeling.

Vegetative symptoms show that the bodies of depressives work differently. Sleep: depressives often wake up early and their whole structure of sleeping is out of wack; appetite decrease (for most of us we eat more when we are somewhat depressed); stress response: overactivation of the sympathetic nervous systems with elevated adreneline and glucocorticoids (the body is acting like it was just gored by an elephant but it can't get out of bed).

Rhythmic patterns to a person's depression are common

Seasonal affective disorders (only getting depressed in the Winter)

Neurotransmitters are the chemical signals neurons use to communicate with other neurons across the space between them (the synapse). There are probably hundreds of neurotransmitters, but only a few are implicated in depression.

MAO inhibitors were the first antidepressants in the 1960s. They suppress the activity of enzymes that break down norepinephrine (and epinephrine, serotonin, and dopamine). Their antidepressant activity suggests that the problem in depression is too little norepinephrine to support synaptic function.

Tricyclic antidepressants block the reabsorption (recycling) of norepinephrine further supporting the theory of inadequate neurotransmitter function in depression (the norepinephrine hypothesis).

Reserpine was used for treating high blood pressure because it depletes norepinephrine with the side effect of causing depression.

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The Biology of Religiosity

Details

What are the biological roots of our religious sense? What does it mean to have religious qualities deeply embedded in our human nature and imposed by our biology? Do we need them? What can we make of the fact that each of our decision's to be religious or irreligious is one of the most defining decisions of our lives yet may be due to a neurotransmitter hiccup or a genetic influence? What does it mean that some people's biology causes them to lose faith yet for others their biology builds their faith?

Why is religion correlated with a longer, happier life and fewer depressions? Is it illogical and unhealthy to be an atheist? Why is having a strong religious sense correlated with prestige and power in cultures all around the world? Is our religiosity an adaptive trait from an evolutionary perspective? Does biology compel even atheists to recognize the profound adaptiveness of religiosity?

The metamagical thinking of shamans and prophets appears to be associated with schizotypalism, an adaptive cousin of the debilitating and maladaptive mental illness schizophrenia. Is religious belief based on loose associations such as those that are common in schizotypals? How can we distinguish the greatness of prophets such as Abraham, Zoroaster, Jesus, and Muhammad from those who get it wrong like David Koresh, Jim Jones, and Charles Manson? What does "get it wrong" or "get it right" mean?

What does it mean when we learn that the same behavior that is valued and praised as religious ritual (principally, self-cleansing, food preparation, entering and leaving significant places, and numerology) is exactly the same behavior in people afflicted with obsessive-compulsive disorder (OCD)? Mild OCD behaviors are essential to succeed in school, in business, and in life, so is the mild form of OCD adaptive?

Did Martin Luther have OCD? Can it be that the most significant event in European history in the last millenium (the protestant reformation initiated by Luther) be associated with someone who today might be in therapy? Does it take a borderline mental person who gets it right to invent profound new ways of seeing the world? Do great advancements always require a form of religiosity or just imagination? Is there a difference between religiosity and imagination?

What does it mean that our religiosity is closely associated with the mental illnesses of schizophrenia and obsessive-compulsive disorder? Can religiosity be a highly adaptive form of being not quite mentally ill? Does that explain mental illness? Or explain religiosity? By treating those who are slightly mentally ill, are we potentially losing our ability to have great new prophets?

Do animal studies that achieve "superstitious conditioning" and "temporal lobe personality" in humans (a temporal lobe epilepsy associated with an increased concern with religious and philosophical subjects) suggest that the hippocampus is where the metamagical faculty of cause and effect is centered in the brain? Do our individual differences in this brain center account for much of our religiosity? What does it mean that genetic, hormonal, and other random factors affecting the development of one's hippocampus may cause the tenor of one's religiosity (including our irreligiosity)?

Is our religiosity simply a function our a special case brain developing in our heads under the influences of genetics, hormones, and life experiences (most of which are beyond our control)?

Here is an exquisite (but optional) video resource with Stanford Biologist Robert Sapolsky to contextualize and inform our discussion:

• In this profound and challenging hour and twenty minute video, Robert Sapolsky lays out the biological bases of religiosity:

http://www.youtube.com/watch?v=4WwAQqWUkpI

Read my (CJ Fearnley) notes on Sapolsky's video (https://plus.google.com/u/0/104222466367230914966/posts/PkFLNEonRJH). Read dj Busby's notes on the video (http://astronasty.blogspot.com/2011/11/sapolsky-religion-lecture-dissected.html). Read Bret Wright's article in the Colorado Springs Independent on Sapolsky's take on brains and religion (http://www.csindy.com/coloradosprings/neurobiologist-robert-sapolsky-spreads-the-good-word-about-brains-and-religion/Content?oid=2652816). Read Josh Jones' review of the video (http://www.openculture.com/2014/12/robert-sapolsky-explains-the-biological-basis-of-religiosity.html).

The next two videos supplement the main video above informing this discussion. They add context and texture to Sapolsky's views, but are less substantial.

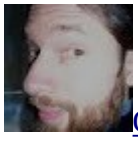
• In this short 4 minute video, Karen Song interviews Robert Sapolsky about his views on religiosity:

http://www.youtube.com/watch?v=VEiD3N4zeyM

• In this short 8 minute video, Karen Song completes her interview of Robert Sapolsky and his views on religiosity:

http://www.youtube.com/watch?v=-Fm_09fPvR4

Read dj Busby's notes on a portion of this video (http://astronasty.blogspot.com/2011/11/atheists-nostalgia-for-religion.html).

 CJ Fearnley - 2015-12-27 09:15:56-0500 - Updated: 2015-12-27 09:15:56-0500

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http://www.meetup.com/thinkingsociety/events/227484917/

The Biology of Religiosity

CJ Fearnley

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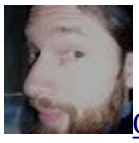 CJ Fearnley - 2015-12-14 16:43:33-0500 - Updated: 2016-01-04 00:05:37-0500

The Biological Underpinnings of Religiosity a 1h 22m lecture by Robert Sapolsky for his 2002 Human Behavioral Biology course.

Sapolsky's profound conclusion is a question: If biological "abnormalities" are responsible for many of the significant developments in religious history, what does that say about being human?

http://www.youtube.com/watch?v=4WwAQqWUkpI

What is the adaptive value of mental illnesses such as schizophrenia? Creativity may be enhanced in manic depression. But Sapolsky claims that there is no adaptive benefit to schizophrenia, it is maladaptive.

This lecture builds on a previous one on schizophrenia. My notes for that lecture from the 2010 edition of "Human Behavioral Biology" are at https://plus.google.com/104222466367230914966/posts/2cnR76GTzFF (Note: the 2010 edition does not include a lecture on religiosity, fortunately this 2002 lecture is available).

Review of a basic medical genetics concept: "some of the time a genetic trait which is god-awful in one setting may have some advantages elsewhere." The gene for sickle cell anemia is awful in one setting, but can protect from malaria in another setting. "There is no such thing as a bad gene, there is only a bad gene-environment interaction."

In full-blown Tay–Sachs disease (predominately found in Ashkenazi Jews) there is complete cortical failure resulting in death by around age 4; in a partial expression of the gene there is resistance to tuberculosis. This genetic trait may explain the belief that Jews were safe from tuberculosis and poisoning the Christian wells resulting in numerous pogroms in the medieval period.

Cystic fibrosis is the most common genetic disorder that is life threatening: the lungs fill with fluid often resulting in death by age 20. But in the partial expression version, the exact mechanism of fluid in the lungs protects you from dehydration from cholera.

The point pertinent to evolutionary theory is that if there are enough people with the partial expression protecting them, it can be evolutionarily advantageous to have the occasional full-blown pathological case in a cousin.

My notes on Seymour Kety's adoption studies in Denmark from previous Sapolsky lectures:
https://plus.google.com/104222466367230914966/posts/1vyQNjwemgX
https://plus.google.com/104222466367230914966/posts/2cnR76GTzFF

Kety and his team of psychiatrists in their decade of research in Denmark interviewed families of adoptees looking for schizophrenics. From their extensive data about the family members of schizophrenics, they were able to conclude that schizoptypal personality occurs at a higher than expected rate in the family members of schizophrenics. Schizotypalism is a mild genetic version of schizophrenia. Modern studies suggest that the genetics of schizotypalism are associated with mild tendencies toward the dopamine role in schizophrenia. Of course, there is a range of degrees of schizophrenia and schizotypalism: they live on a broad continuum. Schizotypalism appears to entail the same biology as schizophrenia but in a much milder form.

Schizotypalism is characterized by having somewhat loose associations (not nearly so severe as in schizophrenia), social withdrawal / detachment (may gravitate to solitary occupations such as lighthouse keeper, fire tower watcher, movie projectionist, etc.), metamagical thinking (believing in

strange things like science fiction and fantasy or getting really involved in some new age ideas or beliefs in mental telepathy or UFOs) with a concrete level of interpretation (tending to extremely fundamentalist concrete interpretations of religious events).

In the 1930s (or 40s) anthropologist Paul Radin observed that in traditional human societies there is a category of people who are "half crazy" and are more commonly known as shamans, witch doctors, or medicine men and women. Radin emphasized the metamagical thinking of these important members of traditional human societies. They converse with the dead, speak in tongues, their role in the society is to be an important religious leader with their metamagical thinking.

Erwin Ackerknecht referred to shamans as being "heeled madmen" in the 1940s.

Alfred Kroeber, who started the Anthro department at Berkeley, wrote "The shaman displays his possession by a spirit by publically reenacting his specific personal experience: that of a man suffering from a particular mental affliction, his projections, his hallucinations, his journey through space and time thus became a dramatic ritual and served as the prototype for all future concepts of the religious road of perfection." Kroeber emphasized that they are not schizophrenics: they are hearing voices at the right time.

Shamanism is a very controlled, socially contextual version of metamagical schizotypalism. Shamans, unlike schizophrenics, are not peripheralized, they are some of the most powerful members of their societies. Most of them are not celibate (though they are in some societies) and as respected members of their society are often able to pass on their genes. That is, shamanism appears to be an adaptive trait. Shamanism is sanctioned in these societies (Kroeber's word) which is related to the word sanctuary. When sanctioned these metamagical traits are revered. But the exact same traits would be psychologically suspect in today's American culture.

Kroeber was told by an informant of the Winnebago (Hocąk) Indians (did Sapolsky mispeak? Wikipedia suggests that Radin studied the Winnebago not Kroeber) that although it is great that they have this fantastic shaman speaking in tongues and all that it is good that we don't have two of them. So there's a recognition that the metamagical schizotypal personality trait needs to be rare in human societies. All human societies tend to have a few metamagical schizotypal people around, but not too many. So it could be that human societies need a few schizotypals and they can accept having a few second cousins with maladaptive schizophrenia. That may explain the 1-2% occurrence of schizophrenia in every culture studied.

Paul Devereux referred to "primitive" religion as organized schizophrenia (1940s).

Ackerknecht wrote an article emphasizing that "modern" religions are equally irrational as the so called "primitive" ones. Ackerknecht observed that "our culture is unique in outlawing the irrational". But a Gallup poll reveals that 25% of Americans believe in ghosts, 36% believe in mental telepathy, 47% believe in UFOs, more than 50% believe that the devil influences daily activity. Sapolsky: "We live in a world that is highly irrational." Schizotypalism runs through all religious contexts including our own. We also value those who invent the appropriate irrationalities at the center of our religious cultures. Sapolsky observes that it is normally considered worrying to hear voices coming out of burning bushes or that you had a conversation with someone who is dead and has risen from the grave.

"Schizotypalism runs through all of human history."

Those who invent religious traditions tend to have a thread of metamagical thinking that falls into the domain of schizotypalism. But the context is very important. When it isn't done "right" it can

lead to abortive cults and the like (e.g., David Koresh in Waco, TX, Jim Jones in Jonestown, Guyana, Charles Manson may have been metamagical thinkers who didn't get it "right").

What distinguishes metamagical thinking that forms the basis of religions from the model thinking that characterizes science? Think about the theory of electricity and magnetism and the theory of gravity. Wouldn't the creative act of conceptualizing such "action at a distance" theories require the basic imaginative power that Sapolsky calls metamagical thinking? Does that suggest that science like religion is built on metamagical thinking?

The basic religious tenets are the structural steel of religion (which is associated with this metamagical thinking). Another component of religion (the modern secular view) is the social community it brings you, encouragement for doing good works, counseling the troubled, helping newlyweds understand what they are getting into, comforting the bereaved, etc. But throughout most of history, religion has always also been about ritual: ritualism is one of the backbones of religion. Religion as the daily practice of ritual.

Sapolsky gives the quote "Religion is daily bread not cake for special occasions (or not just cake for Sundays)" which he attributes to Henry Ward Beecher, but I could not corroborate that with an Internet search. Moreover, Henry was Harriet Beecher Stowe's brother not her father. Maybe the quote is from her father Lyman Beecher who was also a famous preacher? I cannot find any attribution for the quote.

What is the biology of ritual? We all fall into little obsessive rituals during times of anxiety. Intrusive unhelpful thoughts such as Teletubbies jingles get into our heads and ruin our productivity at times. Obsessive rituals and intrusive thoughts tend to happen more often during periods of anxiety.

Obsessive-Compulsive Disorder (OCD) is when the obsessive and intrusive thoughts become so predominant in our lives that it paralyzes our ability to function. The most common form is an obsessive focus on hygiene such as washing one's hands for six hours a day. OCD is an anxiety disorder: a pathological attempt to impose order and structure, predictability and control in a world where everything provokes a sense of dis-ease. It is a fixed action pattern (ethology term discussed in these notes: https://plus.google.com/104222466367230914966/posts/YUnRBsoio66) of compulsive thoughts. People suffering OCD do not fall into learned helplessness (depression) because they are motivated to fix their lives through their rituals. In the right context, the behaviors of OCD are also exactly the ones we need to study hard to get into elite universities like Stanford. OCD occurs in between 1 and 10 percent of the population.

Religious leaders are often the most fervent in the carrying out of rituals. In orthodox Hinduism, a Brahmin will spend six hours per day in cleansing rituals as well as numerous other ritual practices. In orthodox Judaism, Kosher laws, magic numbers, and others provide more examples of the prevalence of rituals. In the middle ages the view developed that there are 365 prohibitions and 248 (representing the number of bones in the body according medieval medicine) obligations for a total of 613 rituals. But no one ever listed them: the numerology was more important than the details. Orthodox Islam also has numerous ritual obligations (magic numbers: 7, 10, 70 and 100). In Christianity, there are rosaries, the number 3, and many rules for prayer, etc.

Religious rituals tend to involve cleansing of the body, food preparation, entering and leaving significant places, and numerology which is the exact same list we find with OCD. About 100 years ago Freud described OCD as "an individual religiosity and religion as a universal obsessional compulsion."

Religious leaders have also reached this conclusion. Saint Ignatius of Loyola defined scrupulosity "as someone going through religious ritual for its own sake, someone who is not thinking of the content but was just doing the ritual". Ignatius warned about the dangers of those who are not paying attention to the content. In the Talmud, there is also an injunction to think about the words, the content, and to not read it in a rote way. Mohammad in Islam argued that if you say prayers without thinking of the content, then they don't count. For millennia, religious leaders have been warning that the power of ritual can potentially become more important than the thought behind it.

The exact same behaviors which in a secular context can earn a diagnosis of OCD, in the right religious content, the behavior may be honored and rewarded. By sharing the dread of the ritual behavior with a community, we share the anxiety of the compulsive behavior.

Franz Kafka's "The Hunger Artist" was about someone performing starving (yikes!). Historical records document this practice in the Middle Ages (double yikes!).

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Martin Luther, a 16th century Augustinian monk in Germany, left extensive records of evidence of his OCD behavior: fear of performing priestly rituals incorrectly and needing to stop and start over, five hours a day in confession. Quote: "the more I wash, the dirtier I get." He became the founder of Protestantism. Sapolsky summarizes: "Talk about somebody who takes his personal affliction and turns it into perhaps the most influential event of the last thousand years in Western European history."

If you are OCD, religion can provide a sanctuary. If you are OCD in the right setting, you can make a living by being religiously ritualistic. OCD people probably invented most of religion's rituals.

The most common manifestations of OCD and religious ritual are 1) cleansing of the body, 2) food preparation, 3) entering or leaving significant religious places, and 4) numerology. Why should that be? It may be evolutionary convergence: selection for health via cleansing and safe food preparation. Sapolsky suggests that some of it may be that in the right place and time, people with OCD may offer their obsessions as rituals to help people at a major religious transition point (he claims this small speculation is the only part of this lecture that is his own contribution to our understanding of religiosity).

Is what people do with diligence and attention to detail distinguishable from OCD behavior? What distinguishes a psychological affliction from strength of character?

Parallelism of schizotypalism and the metamagical backbone of theology, OCD and the ritualism of mainstream orthodox religion.

Key point: The exact same behaviors that in one context destroys your life and peripheralizes you,

when performed in the right context can be honored and rewarded.

One of the healthiest things you can do for your life is to be religious. Religiosity is a very strong protector against major depression. Religious belief extends your life expectancy after you control for risk factors like excessive drinking. No one knows if personal religiosity or a community religiosity offer the same health benefits or not. It is straightforward to see how religiosity increases one's ability to avoid depression by providing elements of control, predictability, explanation, and outlet.

If so much evidence supports the value of religiosity, how can scientists like Sapolsky claim to be rational as atheists? Is this an example of scientific-minded atheists engaging in metamagical thinking where despite all the evidence they continue to dogmatically attend to their atheism?

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Rats with hippocampal damage have more difficulty making causal connections: "they are more vulnerable to superstitious conditioning". We all differ in the number of hippocampal neurons, variations in enzymes, degree of myelination, etc., so individual differences can affect our susceptibility to superstitious belief.

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Sapolsky calls it Temporal Lobe Personality. It is a cluster of personality traits that are far more common in people with frontal lobe epilepsy. It involves being extremely serious and humorless, neophobia (averse to new things, small circle of friends, always do the same sorts of things), hypergraphia (obsessive writing), obsessive interest in religious and philosophical subjects (not necessarily becoming religious, just an interest in these topics). Saint Paul may have had frontal lobe epilepsy. What does it mean that a burst of action potentials for 30 seconds in the temporal lobe once every six months means that one somewhat deterministically gets more interested in religious subjects?

Sapolsky describes the fascinating novel "Lying Awake" by Mark Salzman where a dispirited nun starts having religious visions which makes her an honored and respected member of the convent. The cause was a temporal lobe tumor but raises wrenching questions about what it means that the visions were "caused" by a disease? Should we want such a disease cured?

Sapolsky clarifies a few points: He is not saying that you gotta be crazy to be religious; He is not saying that most religious people are psychiatrically suspect. He is saying that "it is absolutely fascinating that the same exact traits which in a secular context are life destroying, separate you from the community, and in the right setting are at the very core of what is protected, what is sanctioned, what is rewarded, what is valued in a religious setting; so often there could be an underlying biology to all of this. And what do we do with this?"

He confesses that he broke with his extremely orthodox upbringing at age 14 and has no spirituality

and is utterly atheist but that that is one of his biggest regrets, yet he seems completely unable to change his atheism.

What do we make of the fact that each of our decisions to be religious or irreligious is one of the most defining decisions of our lives yet it may be due to a neurotransmitter hiccup or genetic influence. It is just as interesting to ask biologically why do some of us lose faith whereas others find faith?

What are we to make of who we are if even one defining moment of history is due to some biological abnormality?

What does it mean that biology appears to be the cause of each of our individual differences?

CJ Fearnley's post

CJ Fearnley

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The Biological Underpinnings of Religiosity a 1h 22m lecture by Robert Sapolsky for his 2002 Human Behavioral Biology course.
Sapolsky's profound conclusion is a question: If biological "abnormalities" are responsible for many of the significant developments in religious history, what does that say about being human?
http://www.youtube.com/watch?v=4WwAQqWUkpI
What is the adaptive value of mental illnesses such as schizophrenia? Creativity may be enhanced in manic depression. But Sapolsky claims that there is no adaptive benefit to schizophrenia, it is maladaptive.
This lecture builds on a previous one on schizophrenia. My notes for that lecture from the 2010 edition of "Human Behavioral Biology" are at https://plus.google.com/104222466367230.../posts/2cnR76GTzFF (Note: the 2010 edition does not include a lecture on religiosity, fortunately this 2002 lecture is available).
Review of a basic medical genetics concept: "some of the time a genetic trait which is god-awful in one setting may have some advantages elsewhere." The gene for sickle cell anemia is awful in one setting, but can protect from malaria in another setting. "There is no such thing as a bad gene, there is only a bad gene-environment interaction."
In full-blown Tay–Sachs disease (predominately found in Ashkenazi Jews) there is complete cortical failure resulting in death by around age 4; in a partial expression of the gene there is resistance to tuberculosis. This genetic trait may explain the belief that Jews were safe from tuberculosis and poisoning the Christian wells resulting in numerous pogroms in the medieval period.

Cystic fibrosis is the most common genetic disorder that is life threatening: the lungs fill with fluid often resulting in death by age 20. But in the partial expression version, the exact mechanism of fluid in the lungs protects you from dehydration from cholera.
The point pertinent to evolutionary theory is that if there are enough people with the partial expression protecting them, it can be evolutionarily advantageous to have the occasional full-blown pathological case in a cousin.
My notes on Seymour Kety's adoption studies in Denmark from previous Sapolsky lectures:
https://plus.google.com/104222466367230.../posts/1vyQNjwemgX
https://plus.google.com/104222466367230.../posts/2cnR76GTzFF
Kety and his team of psychiatrists in their decade of research in Denmark interviewed families of adoptees in Denmark looking for schizophrenics. From their extensive data about the family members of schizophrenics, they were able to conclude that schizoptypal personality occurs at a higher than expected rate in the family members of schizophrenics. Schizotypalism is a mild genetic version of schizophrenia. Modern studies suggest that the genetics of schizotypalism are associated with mild tendencies toward the dopamine role in schizophrenia. Of course, there is a range of degrees of schizophrenia and schizotypalism: they live on a broad continuum. Schizotypalism appears to entail the same biology as schizophrenia but in a much milder form.
Schizotypalism is characterized by having somewhat loose associations (not nearly so severe as in schizophrenia), social withdrawal / detachment (may gravitate to solitary occupations such as lighthouse keeper, fire tower watcher, movie projectionist, etc.), metamagical thinking (believing in strange things like science fiction and fantasy or getting really involved in some new age ideas or beliefs in mental telepathy or UFOs) with a concrete level of interpretation (tending to extremely fundamentalist concrete interpretations of religious events).
In the 1930s (or 40s) anthropologist Paul Radin observed that in traditional human societies there is a category of people who are "half crazy" and are more commonly known as shamans, witch doctors, or medicine men and women. Radin emphasized the metamagical thinking of these important members of traditional human societies. They converse with the dead, speak in tongues, their role in the society is to be an important religious leader with their metamagical thinking. Erwin Ackerknecht referred to shamans as being "heeled madmen" in the 1940s.
Alfred Kroeber, who started the Anthro department at Berkeley, wrote "The shaman displays his possession by a spirit by publically reenacting his specific personal experience: that of a man suffering from a particular mental affliction, his projections, his hallucinations, his journey through space and time thus became a dramatic ritual and served as the prototype for all future concepts of the religious road of perfection." Kroeber emphasized that they are not schizophrenics: they are hearing voices at the right time.
Shamanism is a very controlled, socially contextual version of metamagical schizotypalism. Shamans, unlike schizophrenics, are not peripheralized, they are some of the most powerful members of their societies. Most of them are not celebrate (though they are in some societies) and as respected members of their society are often able to pass on their genes. That is, shamanism appears to be an adaptive trait. Shamanism is sanctioned in these societies (Kroeber's word) which is related to the word sanctuary. When sanctioned these metamagical traits are revered. But the exact same traits would be psychologically suspect in today's American culture.
Kroeber was told by an informant of the Winnebago (Hocąk) Indians (did Sapolsky mispeak? Wikipedia suggests that Radin studied the Winnebago not Kroeber) that although it is great that they have this fantastic shaman speaking in tongues and all that it is good that we don't have two of them. So there's a recognition that the metamagical schizotypal personality trait needs to be rare in

human societies. All human societies tend to have a few metamagical schizotypal people around, but not too many. So it could be that human societies need a few schizotypals and they can accept having a few second cousins with maladaptive schizophrenia. That may explain the 1-2% occurrence of schizophrenia in every culture studied.
Paul Devereux referred to "primitive" religion as organized schizophrenia (1940s).
Ackerknecht wrote an article emphasizing that "modern" religions are equally irrational as the so called "primitive" ones. Ackerknecht observed that "our culture is unique in outlawing the irrational". But a Gallup poll reveals that 25% of Americans believe in ghosts, 36% believe in mental telepathy, 47% believe in UFOs, more than 50% believe that the devil influences daily activity. Sapolsky: "We live in a world that is highly irrational." Schizotypalism runs through all religious contexts including our own. We also value those who invent the appropriate irrationalities at the center of our religious cultures. Sapolsky observes that it is normally considered worrying to hear voices coming out of burning bushes or that you had a conversation with someone who is dead and has risen from the grave.
"Schizotypalism runs through all of human history."
Those who invent religious traditions tend to have a thread of metamagical thinking that falls into the domain of schizotypalism. But the context is very important. When it isn't done "right" it can lead to abortive cults and the like (e.g., David Koresh in Waco, TX, Jim Jones in Jonestown, Guyana, Charles Manson may have been metamagical thinkers who didn't get it "right").
What distinguishes metamagical thinking that forms the basis of religions from the model thinking that characterizes science? Think about the theory of electricity and magnetism and the theory of gravity. Wouldn't the creative act of conceptualizing such "action at a distance" theories require the basic imaginative power that Sapolsky calls metamagical thinking? Does that suggest that science like religion is built on metamagical thinking?
The basic religious tenets are the structural steel of religion (which is associated with this metamagical thinking). Another component of religion (the modern secular view) is the social community it brings you, encouragement for doing good works, counseling the troubled, helping newlyweds understand what they are getting into, comforting the bereaved, etc. But throughout most of history, religion has always also been about ritual: ritualism is one of the backbones of religion. Religion as the daily practice of ritual.
Sapolsky gives the quote "Religion is daily bread not cake for special occasions (or not just cake for Sundays)" which he attributes to Henry Ward Beecher, but I could not corroborate that with an Internet search. Moreover, Henry was Harriet Beecher Stowe's brother not her father. Maybe the quote is from her father Lyman Beecher who was also a famous preacher? I cannot find any attribution for the quote.
What is the biology of ritual? We all fall into little obsessive rituals during times of anxiety. Intrusive unhelpful thoughts such as Teletubbies jingles get into our heads and ruin our productivity at times. Obsessive rituals and intrusive thoughts tend to happen more often during periods of anxiety.
Obsessive-Compulsive Disorder (OCD) is when the obsessive and intrusive thoughts become so predominant in our lives that it paralyzes our ability to function. The most common form is an obsessive focus on hygiene such as washing one's hands for six hours a day. OCD is an anxiety disorder: a pathological attempt to impose order and structure, predictability and control in a world where everything provokes a sense of dis-ease. It is a fixed action pattern (ethology term discussed in these notes: https://plus.google.com/104222466367230.../posts/YUnRBsoio66) of compulsive thoughts. People suffering OCD do not fall into learned helplessness (depression) because they are

motivated to fix their lives through their rituals. In the right context, the behaviors of OCD are also exactly the ones we need to study hard to get into elite universities like Stanford. OCD occurs in between 1 and 10 percent of the population.

Religious leaders are often the most fervent in the carrying out of rituals. In orthodox Hinduism, a Brahmin will spend six hours per day in cleansing rituals as well as numerous other ritual practices. In orthodox Judaism, Kosher laws, magic numbers, and others provide more examples of the prevalence of rituals. In the middle ages the view developed that there are 365 prohibitions and 248 (representing the number of bones in the body according medieval medicine) obligations for a total of 613 rituals. But no one ever listed them: the numerology was more important than the details. Orthodox Islam also has numerous ritual obligations (magic numbers: 7, 10, 70 and 100). In Christianity, there are rosaries, the number 3, and many rules for prayer, etc.

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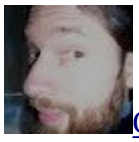 CJ Fearnley - 2016-03-11 13:26:29-0500 - Updated: 2016-07-01 18:20:10-0400
This final lecture in the course "Human Behavioral Biology" with Robert Sapolsky explores how everything learned in the course leads to a profound biological understanding of individuality built on a bedrock of randomness and determinism. What does this mean to us as individuals and to us as a society?

The challenging question about where free will enters the picture is discussed. If each of us is just a few neurons, neurotransmitters, and genetic variations on receptor proteins away from being like one of them (whether you choose to imagine someone whose body or mind is only a little bit different or someone who you love and admire or someone who you cannot stand), what is the real nature of being human?

Is each of us just the output of the biological accidents that make up a human brain and the body with which it is intimately interacting through blood and neuron?

http://www.youtube.com/watch?v=-PpDq1WUtAw

I started posting notes summarizing videos for this course on 18 March 2013 (https://www.facebook.com/cj.fearnley/posts/423274701095489 https://plus.google.com/u/0/104222466367230914966/posts/c4k79s7fCx5). I've written notes for each of the 24 previous videos in the course. This is my last set of notes for this course!

BIO 250, HUMBIO 160: Human Behavioral Biology is the 54th free on-line course I've "completed" since I started taking such courses in 2008. Since I only took about 30 courses for my BA degree from Binghamton University, I am pretty sure I now deserve an edutainment degree for this effort. However, only 18 of those courses (12 offered statements of accomplishment while the other six involved no independent assessment of my efforts), including this one, were pursued with

a significant intensity of effort.

The whole course has been about biologically answering the question why did a given behavior occur? The course has asked the question for both benign human social behaviors and for disastrous ones (violence, mental illness, etc.). Exploring the biological causes of these behaviors raises the troubling question: Whose fault is it when an abnormal behavior occurs?

Sapolsky feels that the whole course in explaining the neurological, hormonal, experience, genetics, and evolutionary bases of human behavior challenges the notion of free will, culpability, and responsibility. Both the questions whose fault is it? and Who is worthy of praise for "good" behaviors? are equally interesting.

"Where are these behaviors coming from?"

500 years ago, epilepsy was well understood: it was certainly caused by demonic possession. Today if an epileptic strikes someone in the midst of a seizure they are no longer held culpable. For the past hundred years or so we've been taught "it's not him, it's his disease". Which is a major change from burning them at the stake. We now understand there is a difference between the individual and the action potential storms that sometimes occur in an epileptic's brain.

Since John Hinkley's attempted 1981 assassination of President Reagan with his paranoid schizophrenia and successful insanity defense, public opinion that "he's getting away with it" has increased. The Insanity Defense Reform Act of 1984 has made it more difficult to use the insanity defense and now America incarcerates many of its mentally ill. Can and should we distinguish between John Hinkley and the dopamine abnormalities in his brain?

Parents, teachers, and guidance counsellors also struggle with these distinctions of culpabilities with the biology of learning disabilities and dyslexias. Are such kids "lazy" or "stupid" or is their biology to blame? "An awful lot of people in positions of power, parents, et. al., have still not gotten very good at drawing the line between the essence of who that person is and the biological constraints that are superimposed on top of it.

At some point in considering the distinction between the biology of "them and their disorders" we must consider the biology of us, the biology of our individual differences, quirks, and idiosyncrasies. Am I who I am because of my biology? Or because it is me? What's the difference?

What are we to make of subtle neuropsychiatric "conditions" which lie in the netherland between normal and abnormal?

Schitzotypalism is not a psychiatric disorder (it is a much milder form of schizophrenia with loose associations and the like but in more appropriate contexts; see these notes for a broader discussion https://www.facebook.com/cj.fearnley/posts/10207550012844056).

What is the difference between us and our quirky behavior and "them" and their psychiatric disorders?

Sapolsky reviews cases of frontal lobe damage causing abnormal behavior (Phineas Gage and other examples are discussed in these notes https://plus.google.com/u/0/104222466367230914966/posts/bQbeD1mgzdj). Each of us has a differently wired frontal cortex which is responsible for our style, quirks, intellectual strengths and weaknesses. Are our disinhibitions and inhibitions the result of too many or too few neurons in the right place? Is it OK when our frontal lobes make mistakes? What is a mistake?

Huntington's disease (more details about Huntington's are in these notes https://plus.google.com/u/0/104222466367230914966/posts/QDRe7itaLsk) is a horrible neurological disorder that gradually results in an uncontrollable writhing. A few years before Huntington's becomes a neurological disorder, it starts as a psychiatric disorder of disinhibition (he gives the examples of a happily married man who suddenly in his 40s punches someone at work, has an affair, gets into a fight in a bar, and embezzles all the funds from work). The damage in the frontal cortex precedes the damage in the motor pathways. Huntington's may have a selective advantage in its promiscuity (onset around age 40) before the neurological devastation begins. It is a genetic disease involving a single gene!

How many non-disease single gene variations does each of us have? Is one of those responsible for this or that behavior in you or your neighbor? Would knowing that your neighbor makes their choices one way because of their brain wiring (either caused by developmental accidents or genetic ones) change the way we think about who we are and how we choose? Does it clarify what we are really free to do? Are we free to behave in a way in which are brains are not wired?

Frontal cortex damage problems remind us that every day we all have thoughts that are boastful or lustful or petulant, etc where we would be horrified if anyone knew what we were thinking. With frontal cortex damage, we say what we are thinking revealing just how tenuous our behavior really is.

Tourette's disease involves aggressive and sexually inappropriate gesturing, facial ticks, animal sounds, extensive cursing. The essence of a Tourette's patient has nothing to do with their behavior. And in milder forms, it is just individual variation. It tends to manifest in adolescence and affects girls more than boys.

PANDAS (pediatric auto-immune neurological disorders associated with streptococcal infections) refers to the 1 in 10,000 kids who recover from a strep infection and then develops symptoms akin to Tourette's. PANDAS can sometimes be "cured" by giving immunosuppresive drugs. Future infection may cause a recurrence of the behavior. Evidently, the immune system pierces the blood-brain barrier and affects us in places where the immune system ought not go resulting in antibodies that attack your own brain. Adult Tourette's patients and those afflicted with obsessive-compulsive disorder have a higher than chance levels of antibodies in their blood against constituents of their nervous systems and a higher than expected rate of childhood diseases.

Are we necessarily the behaviors we exhibit? If our behavior is somehow different from what we would value, does that mean our biology is doing stuff with our bodies that we need not or ought not own? Can and should we separate a person from their behavior? Can and should we sanction or praise someone's behavior if it is beyond their control?

People with temporal lobe personality (see these notes for more details https://www.facebook.com/cj.fearnley/posts/10207550012844056) tend to be more interested in religion. How are we supposed to make sense of the other if they may be the product of a classifiable brain type? How are we supposed to make sense of us if we are the product of our brain types? Are we simply random variations of the type of brain we ended up with? Is that fair? How can we accept that?

OCD (obsessive compulsive disorder which is also discussed in the notes at https://www.facebook.com/cj.fearnley/posts/10207550012844056) has links to strep infections and genetic components. Brain imaging studies suggest that people with OCD have increased metabolic rate in the basal ganglia (see here for more details

https://plus.google.com/104222466367230914966/posts/9U61Mxmj61F) which is a part of the brain involved in movement. Maybe OCD is an itch in the motoric subsystem of the brain? OCD patients treated with SSRIs (selective serotonin reuptake inhibitor, for more details see these notes https://plus.google.com/104222466367230914966/posts/6S7wF6aMejY) respond with reduced activity in their basal ganglia.

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In another case a stroke in a gang member led to becoming obsessed with Polka music.

There are countless ways in which things can go wrong in our brains. As psychiatry categorizes more and more patterns, pretty soon every single one of us will have two or three bone fide psychiatric conditions.

What are we to make of this? Is this a profound window into the nature of our individual differences? Are each of us two or three neurons and 1 or 2 cc of neurotransmitter away from a psychiatric disorder? Do our brains and bodies give us our behavior and we just ascribe the label "our choice" to the result?

What is the difference between a psychiatric disorder and "normal" brain functioning?

Every one of us has elements of some of these psychiatric disorders. Sapolsky admits to some obsessive intrusive thoughts such as counting steps or thinking about the YouTube video "Charlie Bit Me" (http://www.youtube.com/watch?v=HE4FJL2IDEs).

The same biology for schizophrenia (a thought disorder which frequently involves metamagical thinking; for more see these notes https://plus.google.com/104222466367230914966/posts/2cnR76GTzFF) in a milder form results in people interested (but less pathologically so) in metamagical thinking and in the mildest form allows each of us to have a fantasy in the supermarket line.

The same biology that causes "them and their diseases" becomes us and who we are.

Now that biology is revealing the details of the genetics and brain science involved in tastes and religion and risk tolerance and faithfulness and ... everything. As biology "explains" everything about human behavior we become uncomfortable because it challenges the notion that we are

utterly unique, we believe that each of us is a flame of individuality that ought not be reduced to a category of neural firing patterns subject to genetic and chemical constraints.

Arthur C. Clarke's story "One Billions Names of God" has a scene where as we name the names of God a star goes out. This metaphor captures the idea of our fear that as we turn each aspect of our personalities into a biochemical pathway, we lose a bit of our individuality, a bit of what makes us who we are.

Sapolsky argues that even if science understood everything, we could still be moved by the experiences of life. Moreover, every time science answers one question many more new ones get generated which are often more interesting than the first question. It is an infinite fractal of knowledge to discover.

Quote from J. B. S. Haldane "Life is not only stranger than we imagine, life is stranger than we can imagine." The actual quote is "I have no doubt that in reality the future will be vastly more surprising than anything I can imagine. Now my own suspicion is that the Universe is not only queerer than we suppose, but queerer than we can suppose."

Sapolsky adds: "The purpose of science is not to go cure us of the sense of mystery, the purpose of science is to constantly reinvent it."

As biology unravels the nature of human behavior and identifies how we tick and how we can control the way we tick, what should society do?

Sapolsky emphasizes that in all the continua of biology and behavior in the course, "there but for the grace of god and a couple of neurotransmitters and three or four more receptors could go I".

Should our empathy, care, understanding, and protection extend to cover even the most biologically "damaged" among us? Even those who are psychologically damaged? Even our so-called criminals?

Should we empathize with murderers and rapists and terrorists as victims of circumstance and biology?

Does understanding the biology that makes us unique along many continua of mental disorders allow us to better protect and care for each other? Is there a role for judgement of others when we know that it is their biology that is causing their "erroneous" behavior?

Is being a healthy person merely saying "we merely have the same diseases that everybody else does"?

Sapolsky argues that understanding our biology should make us more compassionate. Should it?

Should we celebrate and reward those whose biology gives us joy such as top-level scientists, athletes, entertainers, etc. If their biology gives them those faculties should we treat them as "special" in any way?

How does understanding this biology affect you and me?

The concept of modulation was discussed repeatedly in the course where instead of directly causing some behavior, it just amplifies or dampens some pre-existing tendency. Is the point of the course that causality doesn't exist as everything is modulated by a large set of factors?

Sapolsky advises that although it is a huge amount of work to collect the data to understand some phenomena in human behavior with its litany of contingent clauses, it is necessary to synthesize and intuit its nature in order to work to achieve one's goals.

Our biology gives us the capacity to sin, to do wrong, so likewise, you have the power to attain a state of grace through the work you will do.

Oppenheimer after the successful atomic bomb test said something to the effect that now even physics has sin.

"Even though its complicated, you gotta do something."

In archeology, best practice is to excavate only part of a site so that future generations with better tools can come back and learn more. Metaphorically, Sapolsky suggests that we should be aware that people in the future may be thinking very, very differently.

Sapolsky ends by arguing that you don't have to choose between being compassionate and scientific.

What do you make of our predicament as biological creatures with biological brains whose function clearly proves that everything we do is ultimately a function of biology?

What is the impact of this realization? Does it prove that there is no (or little) free will? Does it provide a perspective to help us better appreciate our humanity? Does paying too much attention to our biology blind us to choices, capabilities and possibilities that we may have that biology doesn't yet imagine that we have?

How might science and biology inhibit us from better understanding our humanity? How might science and biology help us better understand our humanity?

What does it mean to be an individual human being?

CJ Fearnley's post

CJ Fearnley

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This final lecture in the course "Human Behavioral Biology" with Robert Sapolsky explores how everything learned in the course leads to a profound biological understanding of individuality built on a bedrock of randomness and determinism. What does this mean to us as individuals and to us as a society?

The challenging question about where free will enters the picture is discussed. If each of us is just a few neurons, neurotransmitters, and genetic variations on receptor proteins away from being like one of them (whether you choose to imagine someone whose body or mind is only a little bit different or someone who you love and admire or someone who you cannot stand), what is the real nature of being human?
Is each of us just the output of the biological accidents that make up a human brain and the body with which it is intimately interacting through blood and neuron?
http://www.youtube.com/watch?v=-PpDq1WUtAw
I started posting notes summarizing videos for this course on 18 March 2013 (https://www.facebook.com/cj.fearnley/posts/423274701095489 https://plus.google.com/.../10422246636.../posts/c4k79s7fCx5). I've written notes for each of the 24 previous videos in the course. This is my last set of notes for this course!
BIO 250, HUMBIO 160: Human Behavioral Biology is the 54th free on-line course I've "completed" since I started taking such courses in 2008. Since I only took about 30 courses for my BA degree from Binghamton University, I am pretty sure I now deserve an edutainment degree for this effort. However, only 18 of those courses (12 offered statements of accomplishment while the other six involved no independent assessment of my efforts), including this one, were pursued with a significant intensity of effort.
The whole course has been about biologically answering the question why did a given behavior occur? The course has asked the question for both benign human social behaviors and for disastrous ones (violence, mental illness, etc.). Exploring the biological causes of these behaviors raises the troubling question: Whose fault is it when an abnormal behavior occurs?
Sapolsky feels that the whole course in explaining the neurological, hormonal, experience, genetics, and evolutionary bases of human behavior challenges the notion of free will, culpability, and responsibility. Both the questions whose fault is it? and Who is worthy of praise for "good" behaviors? are equally interesting.
"Where are these behaviors coming from?"
500 years ago, epilepsy was well understood: it was certainly caused by demonic possession. Today if an epileptic strikes someone in the midst of a seizure they are no longer held culpable. For the past hundred years or so we've been taught "it's not him, it's his disease". Which is a major change from burning them at the stake. We now understand there is a difference between the individual and the action potential storms that sometimes occur in an epileptic's brain.
Since John Hinkley's attempted 1981 assassination of President Reagan with his paranoid schizophrenia and successful insanity defense, public opinion that "he's getting away with it" has increased. The Insanity Defense Reform Act of 1984 has made it more difficult to use the insanity defense and now America incarcerates many of its mentally ill. Can and should we distinguish between John Hinkley and the dopamine abnormalities in his brain?
Parents, teachers, and guidance counsellors also struggle with these distinctions of culpabilities with the biology of learning disabilities and dyslexias. Are such kids "lazy" or "stupid" or is the biology to blame? "An awful lot of people in positions of power, parents, et. al., have still not gotten very good at drawing the line between the essence of who that person is and the biological constraints that are superimposed on top of it.
At some point in considering the distinction between the biology of "them" and "their disorders" we must consider the biology of us, the biology of our individual differences, quirks, and idiosyncrasies. Am I who I am because of my biology? Or because it is me? What's the difference?

What are we to make of subtle neuropsychiatric "conditions" which lie in the netherland between normal and abnormal?
Schitzotypalism is not a psychiatric disorder (it is a much milder form of schizophrenia with loose associations and the like but in more appropriate contexts; see these notes for a broader discussion https://www.facebook.com/cj.fearnley/posts/10207550012844056).
What is the difference between us and our quirky behavior and "them" and their psychiatric disorders?
Sapolsky reviews cases of frontal lobe damage causing abnormal behavior (Phineas Gage and other examples are discussed in these notes https://plus.google.com/.../10422246636.../posts/bQbeD1mgzdj). Each of us has a differently wired frontal cortex which is responsible for our style, quirks, intellectual strengths and weaknesses. Are our disinhibitions and inhibitions the result of too many or too few neurons in the right place? Is it OK when our frontal lobes make mistakes? What is a mistake?
Huntington's disease (more details about Huntington's are in these notes https://plus.google.com/.../10422246636.../posts/QDRe7itaLsk) is a horrible neurological disorder that gradually results in an uncontrollable writhing. A few years before Huntington's becomes a neurological disorder, it starts as a psychiatric disorder of disinhibition (he gives the examples of a happily married man who suddenly in his 40s punches someone at work, has an affair, gets into a fight in a bar, and embezzles all the funds from work). The damage in the frontal cortex precedes the damage in the motor pathways. Huntington's may have a selective advantage in its promiscuity (onset around age 40) before the neurological devastation begins. It is a genetic disease involving a single gene!
How many non-disease single gene variations does each of us have? Is one of those responsible for this or that behavior in you or your neighbor? Would knowing that your neighbor makes their choices one way because of their brain wiring (either caused by developmental accidents or genetic ones) change the way we think about who we are and how we choose? Does it clarify what we are really free to do? Are we free to behave in a way in which are brains are not wired?
Frontal cortex damage problems remind us that every day we all have thoughts that are boastful or lustful or petulant, etc where we would be horrified if anyone knew what we were thinking. With frontal cortex damage, we say what we are thinking revealing just how tenuous our behavior really is.
Tourette's disease involves aggressive and sexually inappropriate gesturing, facial ticks, animal sounds, extensive cursing. The essence of a Tourette's patient has nothing to do with their behavior. And in milder forms, it is just individual variation. It tends to manifest in adolescence and affects girls more than boys.
PANDAS (pediatric auto-immune neurological disorders associated with streptococcal infections) refers to the 1 in 10,000 kids who recover from a strep infection and then develops symptoms akin to Tourette's. PANDAS can sometimes be "cured" by giving immunosuppresive drugs. Future infection may cause a recurrence of the behavior. Evidently, the immune system pierces the blood-brain barrier and affects us in places where the immune system ought not go resulting in antibodies that attack your own brain. Adult Tourette's patients and those afflicted with obsessive-compulsive disorder have a higher than chance levels of antibodies in their blood against constituents of their nervous systems and a higher than expected rate of childhood diseases.
Are we necessarily the behaviors we exhibit? If our behavior is somehow different from what we would value, does that mean our biology is doing stuff with our bodies that we need not or ought

not own? Can and should we separate a person from their behavior? Can and should we sanction or praise someone's behavior if it is beyond their control?
People with temporal lobe personality (see these notes for more details https://www.facebook.com/cj.fearnley/posts/10207550012844056) tend to be more interested in religion. How are we supposed to make sense of the other if they may be the product of a classifiable brain type? How are we supposed to make sense of us if we are the product of our brain types? Are we simply random variations of the type of brain we ended up with? Is that fair? How can we accept that?
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