Evolution & Mental Health

Course #: 3EV3
Time: Tuesdays, 9:30-10:20am; Fridays, 9:30-11:20am
Location: BSB, Rm 136

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The course website is accessible via Avenue to Learn: http://avenue.mcmaster.ca

Course Description and Objectives

This course involves applying basic principles of evolutionary science to address important questions in the science and practice of mental health. What is a disorder and what distinguishes it from the non-disordered state? Why do disorders exist and how are they maintained in populations? How does an evolutionary approach help inform treatment practices? Registrants are expected to be familiar with the basic principles underlying evolutionary biology. The course will be a seminar with a combination of lecture and readings. Grades will be determined by pop quizzes (6%), two midterms (24% and 30%), and a final exam (40%).

The following is a partial list of learning objectives:

1. Students will become familiar with Jerome Wakefield's "harmful dysfunction" definition of disorder.
2. Relatedly, students will become familiar with the concept of an evolved adaptation, how to recognize adaptations in nature, and how the concept of adaptation is crucial to understanding disorder.
3. Students will become familiar with the mutation-selection balance argument for why mental disorders persist in populations.

Communication Policy

E-mail communications must originate from your designated McMaster e-mail account (i.e. mcmaster.ca account). Should we need to communicate with you about individual matters, the email will be sent to your mcmaster.ca account. You should monitor this account regularly. Email sent from third-party providers (yahoo, hotmail, cogeco, sympatico, etc.) will not be received. We have this policy for three reasons: 1. Reduce the amount of incoming spam to our accounts; 2. Ensure that we know with whom we are communicating; 3. Teach the professional use of e-mail. Please note that instructors cannot return long distance telephone calls. Please consider that email is a formal means of communication. You are expected to address your emails to the instructor formally, use coherent complete sentences, and should be signed with your name and student number.

Grading Policy

Grades will be determined by pop quizzes (6%), two midterms (24% and 30%), and a final exam (40%).

Attendance

Students are expected to attend every class. Pop quizzes are randomly given over the term to provide students with an incentive to attend.

Quizzes

There will be six pop quizzes throughout the semester that will collectively constitute 6% of the grade. Pop quizzes are intended to give students an incentive to attend the class and to do the readings. Consequently, unlike exams (see below), pop quizzes will only be based on the readings for that week. There will be no makeups for missed quizzes. In general, missed quizzes will receive a score of zero. However, at the instructors' discretion (usually based on a Very Good Excuse), the value of the final exam may be increased by the amount of the missed quiz.

Midterms

The midterms will cover all the material discussed in lecture up to that time. Normally, a missed midterm will receive a score of zero. However, at the instructors' discretion (usually based on a Very Good Excuse), the value of the final exam may be increased by the amount of the missed midterm.

Final Exam

The final exam will cover all the material discussed in lecture over the entire term.

Final grade

Students will be assigned a grade from the McMaster University Grading Scale based on an overall assessment by the professor on the work submitted. To pass the course, the student will achieve a passing grade on all graded portions of the course (including attendance). Grades will be computed out of 100 points and converted to a letter grade as follows:

90-100 = A+
85-89 = A
80-84 = A-
77-79 = B+
73-76 = B
70-72 = B-
67-69 = C+
63-66 = C
60-62 = C-
57-59 = D+
53-56 = D
50-52 = D-
< 50 = F

However, the instructors reserve the right to adjust final marks up or down, on an individual basis, in the light of special circumstances and/or the individual total performance in the course. The instructors also reserve the right to adjust the final marks of the entire class based on the overall performance of the class (i.e., we reserve the right to assign grades based on a 'curve').

Missed Work Policy

If you are absent from the university for a temporary medical issue (e.g., the flu), lasting fewer than 5 days, you may report your absence using the McMaster Student Absence Form (MSAF): https://pinjap01.mcmaster.ca/msaf/. Absences for a longer duration or for other reasons must be reported to your Faculty/Program office, with documentation. When using the MSAF, report your absence to pandrews@mcmaster.ca. You must be in contact with the instructor within 5 business days.

Readings

Readings will be posted on Avenue. Readings are designed to introduce or supplement material that will be presented in class. However, they are not a replacement for material presented in class. We will provide you with full references for papers available at the McMaster libraries. You will be expected to obtain these papers from Avenue and read them before the next class.

Other

The professor reserves the right to change any and all course requirements if the need should arise. Any change in the course requirements will be posted on Avenue and the details will be announced in class. Any concerns about announced changes should be addressed with the professor as soon as the changes are announced.

Academic Integrity

Students are expected to be familiar with McMaster's policies on academic integrity as found in the Senate Policy Statements distributed at registration and available in the Senate Office (see http://www.mcmaster.ca/policy/Students-AcademicStudies/AcademicIntegrity.pdf). Any student who infringes one of these resolutions will be treated according to the published policy.

Any instance of plagiarism will be dealt with in the most severe terms allowable by the Senate Policy on such matters.

Policy Regarding Video or Audio Recording the Lectures

You may not record lectures without prior permission from the instructor. You also may not post any recordings of any lecture on the internet.

Course Topics

1. **Scientific Scandals in Psychiatry.** Many students don't have any idea that we need new approaches to mental health. They believe that the conventional wisdom is founded on good science. I often start off the class with a list of scientific scandals in psychiatry, to cast doubt on their certainty and soften them up a bit.

    1.1. Ghost writing
    1.2. Non-disclosure agreements in the DSM-5
    1.3. Failure to disclose financial ties to drug companies
    1.4. Selective publication of RCTs
    1.5. Evidence that the pharmaceutical industry is purposefully trying to convince people that common ailments (e.g., sexual difficulties that some women have) are disorders that are in need of medical intervention. In fact, the pharmaceutical industry really was given a boon with the adoption of the clinical significance criterion in the DSM-III (next topic).

    POSSIBLE READINGS: None. This is usually the first day, and I have prepared slides.

2. **Definitions of Disorder.** Here, I set up two competing definitions of disorder. The one endorsed by mainstream psychiatry, and an evolutionarily based one.

    2.1. The DSM's definition of disorder starts off noting that there must be some dysfunction. However, they have difficulty identifying dysfunction. They operationalize it as clinically significant distress and impairment—aka the clinical significance criterion (CSC).
    2.2. Jerome Wakefield's harmful dysfunction criterion is evolutionarily based and is the most widely cited definition of disorder out there. He ended up converting Robert Spitzer (see Spitzer's introduction to Horwitz & Wakefield's book, The Loss of Sadness). Wakefield's definition has several implications.
        2.2.1. Every true instance of disorder involves a malfunction in an evolved adaptation.
        2.2.2. To truly know that a condition (physical or psychological) is a disorder, one must be able to identify the underlying adaptation that is malfunctioning.
        2.2.3. It therefore is crucially important to be able to accurately detect adaptations.
        2.2.4. We are setting up the idea that ALL the things that psychiatry deems evidence of disorder are actually NOT strong evidence of disorder—distress/pain, psychosocial impairment, anhedonia, wasting away of somatic tissues, even suicide.

    POSSIBLE READINGS:

Wakefield, J. C. (1992). The concept of mental disorder: on the boundary between biological facts and social values. *American Psychologist*, *47*(3), 373. [This is his classic paper.]

Wakefield, J. C. (1999). Evolutionary versus prototype analyses of the concept of disorder. *Journal of abnormal psychology*, *108*(3), 374. [There are parts of Wakefield's classic paper that I like better. But this is an update in which he responds to critics.]

3. **The Concept of Adaptation.** The topic is adaptationism—as a research strategy. The principles help organize the entire class.

    3.1. What are adaptations?
    3.2. Alternative explanations for construction of traits—byproduct, constraint, exaptation
    3.3. How are adaptations recognized? Non-random biological organization sufficient to rule out alternative explanations
    3.4. What is the best way to determine whether a trait is an adaptation or something else?
    3.5. The reverse engineering approach
    3.6. Building conceptual blueprints that are help one understand how a mechanism/trait is constructed so as to perform its function
    3.7. The relevance of building conceptual blueprints for understanding malfunction. E.g., the watch/car repair person has a conceptual blueprint for how watches/cars are constructed so they perform their functions. They use this to diagnose and repair malfunctions.

    POSSIBLE READINGS:

Andrews, P. W., Gangestad, S. W., & Matthews, D. (2002). Adaptationism–how to carry out an exaptationist program. *Behavioral and Brain Sciences*, *25*(04), 489-504.

Andrews et al. (in review, American Psychologist) [Hopefully this will be published or in press by then.]

4. **Energy regulation in response to stress.** Another important principle for the class is the idea that energy is a limited resource. When the organism encounters a substantial stressor, it often must make tradeoffs in how it allocates these limited resources. With this topic, we discuss the stress response, and how it affects energy allocation. We also discuss the evolution of serotonin and the role that it plays in energy regulation. Finally, we look at glycolysis vs. oxidative phosphorylation as two types of metabolism that also have tradeoffs.

    4.1. Chronic activation of the stress response is often considered pathological (see, e.g., Sapolsky), but there are a number of conditions where (as we will see) the stress response is useful (infection, pregnancy)
    4.2. Perhaps better to think of chronic activation of the stress response as part of an adaptive tradeoff, where there are both benefits and costs
    4.3. Insulin resistance as a mechanism for directing energy to needed areas by shutting down areas uptake in areas that are less important
    4.4. Glycolysis is fast, but inefficient; oxidative phosphorylation is efficient, but slow. Interestingly, Pfeiffer's paper demonstrates how selection can favor metabolic processes that lead to smaller population sizes. Implications for group selection.
    4.5. We use these facts in melancholia later on in the class, but they also play a role in sickness behavior and starvation

    POSSIBLE READINGS:

Andrews et al. (2015). Is serotonin an upper or a downer? I may have a much shorter paper that can be used here by the time you teach this class.

Monaghan, P., & Haussmann, M. F. (2015). The positive and negative consequences of stressors during early life. *Early human development*, *91*(11), 643-647.

5. **Selection for Survival.** Here, we want to show how natural selection favors reproductive success, not survival. Survival is important ONLY to the degree that it enhances reproductive success. But there are MANY examples in nature where selection trades off reproduction and survival.

   POSSIBLE READINGS:

Smith, F. A., & Charnov, E. L. (2001). Fitness trade-offs select for semelparous reproduction in an extreme environment. *Evolutionary Ecology Research*, *3*(5), 595-602.

Andrade, M. C. (1996). Sexual Selection for Male Sacrifice in the Australian Redback Spider. *Science*, *271*(5245), 70-72.

6. **Suicidal Behaviour.** This follows up on the previous topic to discuss several adaptationist hypotheses for suicidal behaviour that apply to humans. Again, hammer away at the DSM's concept of disorder.

   6.1. Honest signal of the need for help
   6.2. Leveraging
   6.3. Inclusive fitness

   POSSIBLE READINGS:

Andrews, P. W. (2006). Parent-offspring conflict and cost-benefit analysis in adolescent suicidal behavior. *Human Nature*, *17*(2), 190-211.

Syme, K. L., Garfield, Z. H., & Hagen, E. H. (2016). Testing the bargaining vs. inclusive fitness models of suicidal behavior against the ethnographic record. *Evolution and Human Behavior*, *37*(3), 179-192.

de Catanzaro, D. (1995). Reproductive status, family interactions, and suicidal ideation: Surveys of the general public and high-risk groups. *Ethology and Sociobiology*, *16*(5), 385-394.

7. **Sickness Behaviour & Fever.** The DSM's focus on distress and impairment takes a big hit when we talk about the psychological and physiological responses to infection. These responses cause both distress and impairment, but they are clearly adaptive.

   7.1. Note that sickness behaviour is a depressive phenotype, but it is clearly adaptive

7.2. Symptoms show evidence of design
  7.2.1. Sickness behaviour involves an altered motivational state
    7.2.1.1. Anhedonia is specific—interest in resting, but nothing else
    7.2.1.2. Dietary preferences are specific—preference in carbs, less interest in protein
      7.2.1.2.1. Carbs fuel immune response
      7.2.1.2.2. Loss of interest in protein limits pathogen growth
  7.2.2. Insulin resistance helps reallocate energy to immune response
  7.2.3. Fever coordinates immune response
  7.2.4. Suppression with antipyretic medication disrupts evolved function

POSSIBLE READINGS

Shephard, A. M., Bharwani, A., Durisko, Z., & Andrews, P. W. (2016). Reverse Engineering the Febrile System. *The Quarterly Review of Biology*, *91*(4), 419-457.

Dantzer, R. (2001). Cytokine-induced sickness behavior: where do we stand?. *Brain, behavior, and immunity*, *15*(1), 7-24.

8. Starvation and Anorexia Nervosa. Starvation is another evolutionarily ancient problem that triggers an adaptive depressive phenotype. We continue to hammer away at the DSM's focus on distress and impairment. In starvation, the distress motivates the individual to seek out food, and the impairing symptoms help the body reallocate energy to preserve brain function and promote foraging behavior. Here, we also discuss anorexia nervosa, which simply cannot be understood without understanding the body's adaptations to starvation. Symptomatically, they are identical—or nearly so.

8.1. ***Physiology of starvation***. The body is sacrificed to maintain brain function. The brain is so important to keep going that even the heart is sacrificed!
8.2. Depressive symptoms show evidence of design
  8.2.1. ***Anhedonia is specific***—loss of interest in sex, humor, social companionship, but interest in food is retained
  8.2.2. ***Physical activity is upregulated*** in starving vertebrates and invertebrates; related to foraging behaviour
  8.2.3. ***Dietary preference for protein***; note the difference from sickness behavior. Why the loss of interest in carbs? Carbs trigger insulin, which has the effect of altering electrolyte levels (phosphate, in particular, I think). In starving individuals, the insulin spike caused by carbs causes an electrolyte imbalance that can trigger a heart attack. This phenomenon is called *refeeding syndrome*, and it was discovered in the aftermath of World War II when the allies liberated the concentration camps and tried to nourish the starving survivors back to health. Sadly, their attempts to feed the survivors often caused their deaths. The preference for protein will not trigger the insulin spikes that cause refeeding syndrome, and it will help the body reconstitute the muscle that has wasted.

8.2.4. ***Insulin resistance*** reduces energy expenditure in peripheral tissues, including immune tissues, and helps redirect limited energy resources to the brain.

8.3. ***Most of the symptoms of anorexia are actually adaptive responses to food restriction, including depressive symptoms***. The discussion of anorexia centers around the fascinating article by Bergh et al. (2013). Towards the end of the article, they argue that anorexia nervosa does not have any features that seem to be characteristic of true disorders.

POSSIBLE READINGS:

Bergh, C., Callmar, M., Danemar, S., Hölcke, M., Isberg, S., Leon, M., ... & Palmberg, K. (2013). Effective treatment of eating disorders: Results at multiple sites. *Behavioral neuroscience*, *127*(6), 878.

Zandian, M., Bergh, C., Ioakimidis, I., Esfandiari, M., Shield, J., Lightman, S., ... & Södersten, P. (2015). Control of body weight by eating behavior in children. *Frontiers in pediatrics*, *3*.

Ruiz-Núñez, B., Pruimboom, L., Dijck-Brouwer, D. J., & Muskiet, F. A. (2013). Lifestyle and nutritional imbalances associated with Western diseases: causes and consequences of chronic systemic low-grade inflammation in an evolutionary context. *The Journal of nutritional biochemistry*, *24*(7), 1183-1201.

9. Melancholia

POSSIBLE READINGS:

Andrews, P. W., & Thomson Jr, J. A. (2009). The bright side of being blue: depression as an adaptation for analyzing complex problems. *Psychological review*, *116*(3), 620.

Andrews, P. W., Bharwani, A., Lee, K. R., Fox, M., & Thomson, J. A. (2015). Is serotonin an upper or a downer? The evolution of the serotonergic system and its role in depression and the antidepressant response. *Neuroscience & Biobehavioral Reviews*, *51*, 164-188.

Andrews, P. W., & Durisko, Z. The Evolution of Depressive Phenotypes. In *The Oxford Handbook of Mood Disorders*.

10. Other Adaptationist Hypotheses for Depression

POSSIBLE READINGS:

Hagen, E. H., & Rosenström, T. (2016). Explaining the sex difference in depression with a unified bargaining model of anger and depression. *Evolution, medicine, and public health*, *2016*(1), 117-132.

Allen, N. B., & Badcock, P. B. (2003). The social risk hypothesis of depressed mood: evolutionary, psychosocial, and neurobiological perspectives. *Psychological bulletin*, *129*(6), 887.

11. <u>Anxiety.</u> I don't have any ideal readings for this topic yet. But these are not bad.

<u>POSSIBLE READINGS:</u>

Nesse, R. M. (2005). Natural selection and the regulation of defenses: A signal detection analysis of the smoke detector principle. *Evolution and Human Behavior*, *26*(1), 88-105.

Stein, D. J., & Nesse, R. M. (2011). Threat detection, precautionary responses, and anxiety disorders. *Neuroscience & Biobehavioral Reviews*, *35*(4), 1075-1079.

12. Post-Traumatic Stress. I purposefully drop the word "disorder" from here, which may seem weird. PTSD flows off of the tongue so easily because that is what we always say. But to use the word "disorder" to refer to an adaptive phenotype is an oxymoron. Moreover, it reifies everything that the class is attempting to question. For this reason, I usually discuss this early on in the class. "Don't use the word 'disorder' unless you really mean to argue that the trait involves a brain malfunction. And then you better be able to back up your assertion!" That may seem a little heavy handed, but I usually say it with a smile. Anyway, many trauma researchers are willing to entertain PTS as ancestrally adaptive, though not adaptive in modern environments.

<u>POSSIBLE READINGS</u>:

Sipahi, L., Uddin, M., Hou, Z. C., Aiello, A. E., Koenen, K. C., Galea, S., & Wildman, D. E. (2014). Ancient evolutionary origins of epigenetic regulation associated with posttraumatic stress disorder. *Frontiers in human neuroscience*, *8*.

13. Schizophrenia and the Maintenance of Disorders in Populations. Some mental traits are undeniably disorders, and schizophrenia is a good example. Why doesn't selection completely eliminate them from populations?

<u>POSSIBLE READINGS</u>:

Keller, M. C., & Miller, G. (2006). Resolving the paradox of common, harmful, heritable mental disorders: which evolutionary genetic models work best?. *Behavioral and Brain Sciences*, *29*(04), 385-404.

[I also have an unpublished chapter by Matt Keller and Steve Gangestad on schizophrenia that might be of interest to you.]

14. **Alzheimer's Disease.** A great example of a mental disorder, because we have some understanding of the adaptations that are malfunctioning. I usually take a whole bunch of material that I've collated and present it here. Someday, when I have copious time, I will write it up for publication. Sigh….

15. **Psychopathy.** This trait is a good example of the problem with group selection arguments. Psychopathy is primarily viewed to be pathological on the basis of how it affects society. But there is little evidence of malfunction in psychopathy. In fact, psychopaths have above average reproductive success!

    POSSIBLE READINGS:

Krupp, D. B., Sewall, L. A., Lalumière, M. L., Sheriff, C., & Harris, G. T. (2012). Nepotistic patterns of violent psychopathy: evidence for adaptation?. *Frontiers in psychology*, *3*, 305.

16. **Addictive Drugs.** Here I think we could utilize the section we wrote for the RDoC paper on addictive drugs. Additionally, there is some amazing work done by Ed Hagen on our body's evolved physiology for dealing with many addictive drugs, most of which evolved in plants to deter herbivores.

    POSSIBLE READINGS:

Roulette, C. J., Mann, H., Kemp, B. M., Remiker, M., Roulette, J. W., Hewlett, B. S., ... & Hagen, E. H. (2014). Tobacco use vs. helminths in Congo basin hunter-gatherers: self-medication in humans?. *Evolution and Human Behavior*, *35*(5), 397-407.

Roulette, C. J., Kazanji, M., Breurec, S., & Hagen, E. H. (2016). High prevalence of cannabis use among Aka foragers of the Congo Basin and its possible relationship to helminthiasis. *American Journal of Human Biology*, *28*(1), 5-15.

Sullivan, R. J., & Hagen, E. H. (2015). Passive vulnerability or active agency? An ecological and evolutionary perspective on human drug use. *The Impact of Addictive Substances and Behaviours on Individual and Societal Well-being*, 13. [This is a book chapter. I can probably get it for you from the authors, if you want it.]

17. **Treatments.**