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Guest Editorial

Why Has Natural Selection Left Us So Vulnerable to Anxiety and Mood Disorders?

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What can evolutionary biology offer to our understanding of anxiety and depression? According to 2 articles in this issue, a lot.[1,2] Both include details and debates that could easily obscure their shared crucial main point—the capacities for anxiety and mood were shaped by natural selection because they have been useful. Like sweating, pain, and cough, emotions are only useful in certain situations, so natural selection shaped them in tight conjunction with regulation mechanisms that express them when they are likely to be useful.[3] High body temperature arouses sweating, tissue damage arouses pain, and foreign material in the respiratory tract arouses cough. People who lack these response capacities are likely to die young. So are people who express them too readily, too intensely, or too long. Regulation mechanisms have been finely tuned by millions of years of selection.

However, spending a day in the clinic suggests that the designer of the systems that regulate anxiety and mood must have been having a very bad day. Any engineer responsible for such apparently slipshod design would certainly be subject to legal action!

So, what is the problem? Is natural selection too weak to do better? Evolutionary medicine suggests that this is only 1 of 6 reasons why selection has left us vulnerable to diseases.[4,5] Two others, emphasized in these articles,[1,2] are that some conditions that seem like diseases are actually defences, and that every trait is subject to trade-offs; making it better in one respect will make it worse in others.

Dr Melissa Bateson, Dr Ben Brilot, and Dr Daniel Nettle[1] provide a sophisticated analysis of the trade-offs involved in regulating anxiety. They begin by explaining that evolutionary and mechanistic explanations are equal partners in any complete biological explanation. This foundation from basic behavioural biology should be familiar to readers; if not, the first chapter of any animal behaviour textbook will explain. The authors proceed to argue that anxiety can be useful. This is not controversial, and is already the foundation for much work on anxiety disorders,[6–8] although research documenting the benefits of normal anxiety is overwhelmed by studies showing the costs of anxiety disorders.

They next apply signal detection theory to calculate the optimal signal threshold for expressing an anxiety response. The mathematical foundation they provide is essential, but a simple example illustrates the smoke detector principle. You are a hunter-gatherer at a watering hole. You hear an animal behind a small hill. The noise could have been made by a lion, or by a monkey. Should you flee? Noises made by lions are generally louder, so it depends on how loud the noise is, the relative prevalence of monkeys and lions in the area, and the cost of fleeing versus the cost of not fleeing if a lion is really there. If the cost of fleeing—that is, a panic attack—is about 200 calories, and the average cost of not fleeing in the presence of a lion is about 200 000 calories, then you should flee if the sound is loud enough to make the probability of a lion's presence greater than 1/1000. This means that 999 times out of 1000 your panic response will be a false alarm, but nonetheless normal.

When I first did this calculation, I could hardly believe it. Ever since, I have found it valuable in the clinic. I had previously explained to patients that their panic symptoms were not caused by heart problems but by a disease, panic disorder; most listened politely, and then went for more cardiac tests. Now I tell patients that a panic attack is a normal response that is useful in the face of life-threatening danger, and panic disorder results from false alarms in that system, some of which are to be expected, just as they are in smoke detectors. I also explain that experiencing panic attacks makes the world seem more dangerous, lowering the anxiety threshold, thus causing a vicious circle. Most

patients deeply appreciate this evolutionary explanation; for some, it is a sufficient intervention.

Dr Bateson and colleagues[1] then advance our understanding with an analysis of how selection would have shaped a second-order mechanism that adjusts the threshold as a function of experience. They conclude that when threats are common, or coping ability is limited, the system should lower the threshold. This is what we see in the clinic, and the authors review supporting epidemiologic evidence. The authors also suggest that their analysis should encourage clinicians to help patients to change their beliefs about the probability that a danger is present, and their ability to cope with it. These are, of course, already staples of cognitive-behavioural therapy. What is curious is how weak such interventions often are, compared with simple exposure therapy. I once wondered if patients with anxiety disorders may simply view life's risks more accurately than other people; however, the data showed that their objective assessments of risk were identical to those of control subjects; the problem is not in logical cognition, it is in the intensity of the anxiety response.[9]

The clinical utility of the capacity for low mood is harder to see than that of anxiety. From a clinician's point of view, it can be easy to dismiss the whole idea that anything about depression could be useful. Depression traps people in their current circumstances, keeps them from doing things that would improve their lives, causes untold misery, and is far too often fatal. For these reasons, I avoid writing about the utility of depression, per se; instead, I emphasize the need to understand the capacity for normal low mood as the missing foundation for understanding mood disorders. Dr Edward H Hagen's article[2] nicely summarizes the main proposals about how low mood can be useful. I wish he had put more emphasis on the importance of the core question of why the capacity for mood exists, instead of jumping right into explaining depression, and even major depressive disorder. Natural selection does not shape diseases, it shapes traits that make us vulnerable to disease; keeping this distinction straight is crucial.[10] Dr Hagen[2] is aware of this; his thesis is that the symptoms of major depression could have adaptive functions, even suicidality. I think the extreme version of this thesis is unlikely, but it should not be dismissed out of hand. Reading his work has made me think more deeply about why it is that seriously depressed people so often become preoccupied with their worthlessness, and so often convinced that the world would be better off without them.

Some readers will wonder which evolutionary explanation is correct, or even: Why can't these scientists make up their minds? Work in this area is difficult. First, it can be hard to combine various methods to arrive at a strong test. Second, multiple answers can be correct; emotions have many functions, each of which can offer a selective advantage. Third, it is not clear that depression is all one thing. Different causes can be responsible in different cases: demoralization, loss, distorted thinking, manipulation, being manipulated, being devalued by a social group, inflammation, drugs, and primary brain abnormalities. Worse, in many cases, a full explanation requires analyzing how these factors interact with each other over time to explain an individual's symptoms. Instead of being a problem, this complexity helps to explain why it is so important for clinicians to do what many do well already—investigating all the details of an individual's life, psychology, brain, and symptoms to understand why this person may be especially vulnerable to depression, and what accounts for this episode.

As for the need to avoid a brave new world, Dr Hagen's point that drugs can interfere with adaptive responses is important and correct, but not clinically informed.[2] Much of what general medicine does is to use medications to block normal defensive responses, such as pain, cough, and fever. We can do this safely most of the time because the body has redundant defences that are regulated by mechanisms shaped according to the smoke detector principle. Dr Hagen is correct that we should, like other physicians, investigate what may be arousing symptoms before jumping to the conclusion that they come from abnormal mechanisms.[2] Some have argued that medication treatment for mental disorders is justified because they are caused by brain abnormalities. This unnecessarily limits the use of treatments in psychiatry that can, as in the rest of medicine, relieve enormous suffering by blocking normal aversive responses that may not be necessary for this individual in the current situation.

References

1. Bateson M, Brilot B, Nettle D. Anxiety: an evolutionary approach. Can J Psychiatry. 2011;56(12):707–715.
2. Hagen EH. Evolutionary theories of depression: a critical review. Can J Psychiatry. 2011;56(12):716–726.
3. Nesse RM, Ellsworth PC. Evolution, emotions, and emotional disorders. Am Psychol. 2009;64(2):129–139.
4. Nesse RM, Williams GC. Why we get sick—the new science of Darwinian medicine. New York (NY): Times Books; 1994.
5. Nesse RM, Bergstrom CT, Ellison PT, et al. Making evolutionary biology a basic science for medicine. Proc Natl Acad Sci U S A. 2010;107(Suppl 1):1800–1807.
6. Barlow DH. Anxiety and its disorders. New York (NY): Guilford Press; 1988.
7. Marks IM. Fears, phobias, and rituals. New York (NY): Oxford University Press; 1987.
8. Marks IM, Nesse RM. Fear and fitness: an evolutionary analysis of anxiety disorders. Ethol Sociobiol. 1994;15(5–6):247–261.
9. Nesse RM, Klaas R. Risk perception by patients with anxiety disorder. J Nerv Ment Dis. 1994;182:465–470.
10. Nesse RM. Ten questions for evolutionary studies of disease vulnerability. Evol Appl. 2011;4(2):264–277.

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