SPECIAL ARTICLE

Evolutionary biology: a basic science for psychiatry

RANDOLPH M. NESSE

Departments of Psychiatry and Psychology, and Institute for Social Research Evolution and Human Adaptation Program, University of Michigan, 426 Thompson St., Ann Arbor, MI 58104, USA

One of the most basic advances in biology during the past twenty years is the new clear recognition that two kinds of explanation are needed for all biological traits:

a) a proximate explanation of how the trait works,

b) an evolutionary explanation of what the trait is for (1).

These are not alternatives; both are necessary to a full understanding. For instance, to explain why polar bears have white fur, we need to know both the proximate reason why the fur is white (absence of genes for fur pigment), and the selective advantage of white fur (polar bears with dark fur catch fewer seals).

Most medical research has focused on how the body works and on the proximate factors that explain why some people get a disease and others do not. Darwinian medicine asks a different, evolutionary, question. It asks why we all have bodies that are vulnerable to disease (2,3). Why do we have an appendix and wisdom teeth? Why are our coronary arteries so narrow? Why do we have eyes designed inside out so that the nerves and arteries run between the light and the retina? Why is breast cancer so common now? Why do so many people have anxiety and depression?

At first it seems that the answer is simple. Natural selection is a random process, so it can't bring any trait to ultimate perfection. This is correct and does explain some disease. However, recent more careful consideration has highlighted several other evolutionary reasons why our bodies remain vulnerable to disease: novel environmental factors that our bodies are not designed for, design trade-offs that make us more vulnerable to disease but nonetheless give a net benefit, pathogens that evolve faster than we do, and defenses like pain and cough that seem like diseases but are actually protective mechanisms shaped by natural selection. There is space here for only a few examples of how an evolutionary approach provides a foundation for understanding mental disorders.

Some psychiatric disorders persist because natural selection is not strong enough to eliminate the genes that cause them. Huntington's chorea is the classic example. Because this autosomal dominant gene does not usually cause symptoms until after the age of childbearing, it is not strongly selected against and it spreads in certain family lines. Schizophrenia also results from genetic factors and thus seems superficially similar, but an evolutionary approach calls attention to the relatively uniform prevalence of about 1% worldwide, and the substantially decreased reproduction of individuals with schizophrenia in developed countries. How can we explain the uniform distribution of schizophrenia, and the persistence of genes that decrease fitness? It may be that vulnerability to schizophrenia results from many genes with small effects that make them resistant to elimination by selection. It may also be, however, that these genes also offer benefits, perhaps not to people with schizophrenia, but to relatives who are not ill. These might be mental benefits or they might be something as remote as ability to mount a strong immune response to cholera or plague. A more speculative evolutionary explanation for the persistence of schizophrenia is the possibility that very rapid selection for language and cognitive ability over the past 100,000 years has pushed some aspect of brain development close to a threshold which, if exceeded, causes psychosis in a few unfortunate people.

Some medical disorders result from living in a modern environment that is poorly suited for bodies designed for life foraging on the African savannah. For instance, the current epidemic of atherosclerotic heart disease seems to result from such a mismatch between design and environment. In psychiatry, eating disorders are a good example. Obesity has proximate explanations in brain mechanisms that regulate eating, but to explain why half the people in some developed countries are now overweight, an evolutionary approach is needed. The general answer seems to be that selection to ensure adequate food consumption has always been strong, but selection for mechanisms to prevent excessive intake has been much weaker. When young people decide to lose weight by dieting, the body knows only that too few calories are being taken in to sustain life. The normal and adaptive response to a life-threatening famine is to eat whatever food is available, quickly, in private. This is just what bulimics do. The experience of lack of control causes additional fear of obesity, which motivates more stren-

uous dieting, in a positive feedback cycle that culminates in severe eating disorders. There are, of course, many individual differences in genetics, brain chemistry, past life experiences, and personality that make some people much more vulnerable to such disorders than others. These are all proximate explanations. An evolutionary approach cannot explain these individual differences, but it can help us to understand why the syndrome exists at all, and why it is common now.

Perhaps the most useful contribution of an evolutionary approach to mental disorders is emphasis on the distinction between defects and defensive responses. Most problems that bring patients to doctors are defenses. Cough, pain, fever, vomiting and diarrhea are defenses shaped by natural selection to protect us in certain situations. They are hidden until they are needed. A respiratory infection stimulates regulation mechanisms that arouse cough and fever. As most doctors know, blocking cough can make an ordinary infection fatal because secretions are not cleared from the lungs. Fever is also useful because bacteria cannot grow as well at higher temperatures. Vomiting and diarrhea clear toxins and pathogens from the gastrointestinal tract. Note that all of these experiences are aversive. People intensely dislike them and this is what brings them in for treatment. Treatment often consists of using drugs to block the defense. We use codeine to block cough, aspirin to block fever and analgesics to block pain.

This brings up a big question. If natural selection has shaped the mechanisms that regulate these defenses, then why are they so often expressed excessively? Much of the general practice of medicine consists of blocking these unpleasant defenses, and most of the time this does not harm people. How can this be? Here again, an evolutionary approach reveals the hidden sophistication of the body. The regulation of defenses is governed by what has been called 'the smoke detector principle' (4). We accept smoke detectors that go off when the toast burns because we want to be absolutely sure of a warning if there is a real fire. Similarly, the cost of vomiting is small compared to the cost of a severe intestinal infection, so natural selection has shaped a regulation mechanism that sets off the defense whenever there is any real chance that an intestinal infection is present. This has a profound implication: most human suffering is unnecessary in the specific instances, even though it arises as part of nearly optimal regulation of a normal defense.

Our capacities for emotional suffering are also products of natural selection. It is not always obvious how they are useful, but anxiety is a good place to start. A person who lacked all anxiety would quickly become a meal for a tiger. The capacity for anxiety is useful (5), but many of our patients experience excessive anxiety. Much of this excess can be attributed to the design of the regulation mechanism according to the smoke detector principle. Specific brain mechanisms cause anxiety in all of us, and differences in these mechanisms make some people inordinately anxious in situations that don't bother most people at all. These are proximate explanations; we also need evolutionary explanations for why anxiety exists at all and why it is regulated in the way that it is.

Practical implications come quickly from this perspective. Many people with panic disorder, for instance, believe that their symptoms mean that they have heart disease. Telling them that the symptoms are caused by panic is helpful, but I have found it much more effective to explain that these symptoms would be perfectly normal and useful if a tiger was coming. The syndrome of panic is just a fight-flight reaction that is going off at the wrong time. It is a false alarm. Furthermore, in dangerous environments, the body adjusts the anxiety threshold downwards, just as it should to increase the level of protection. Unfortunately, this system seems to be unable to distinguish between a real life-threatening danger, and a useless panic attack. This is the evolutionary reason why having one panic attack often leads to escalating cycles of panic. In a dangerous environment it is also adaptive to stay close to camp. This is, of course, what agoraphobics do and offers an evolutionary explanation for the comorbidity of panic and agoraphobia. Our lives are so safe now, that the whole system seems unnecessary. But, for our ancestors, the ability to flee at the least hint of danger was essential, and a system to regulate the threshold for flight as a function of the safety of the environment would be crucial. This can help to explain how medications can offer lasting relief from panic. By stopping the cycle of panic attacks, the person gradually begins to experience the environment as safer, and the anxiety threshold again increases. This explanation often helps patients to understand how a medication is doing something more than 'covering up the symptoms'.

Depression offers a more challenging problem. At first glance, it seems impossible that there could be any benefit from lacking energy, being fearful, and withdrawing from social life, to say nothing of the problems caused by not eating or sleeping. Any discussion of depression must start by acknowledging that it often is a pathological condition with no adaptive value. However, what about more mild variation in states of motivation? Are there some situations in which energy, enthusiasm and risk taking would be valuable? Are there some situations in which lack of initiative, pessimism and fearfulness would be useful? While much research is needed to explore these hypotheses in detail, it seems likely that in propitious situations, where a small investment of effort will likely bring large payoffs, a burst of effort and energy will bring big rewards (6). In unpropitious situations, where efforts will be wasted, the best thing to do may be nothing at all. This seems hard to imagine for modern people who always have adequate food and shelter. But imagine a deer waiting in deep snow for spring to come. If it is starving, what should it do? An optimistic deer that wanders off in search of nonexistent food will die much sooner than the one who just waits and waits. High and low states of motivation are each useful, but only in certain situations.

For people now, of course, the availability of food is not a major influence on mood. The resources that make the most difference to us are social. When we experience our efforts as efficacious and bringing us friends and recognition, mood goes up. When all efforts seem to be wasted or to bring danger, mood goes down.

One group of researchers has argued that depression is a state of 'involuntary yielding' that protects against attack after a loss of social position (7). Others see some depressions as states of withdrawal in which the individual regroups to emerge with alternative strategies (8). A major area of psychological research on goal pursuit is very relevant, but relatively unknown in psychiatry. The core idea is that most human action is organized by pursuit of large goals and that there must be a mechanism to disengage effort from unreachable goals (9,10). If people persist in the pursuit of an unreachable goal, ordinary normal low mood is likely to escalate into full-blown depression (11). Much clinical evidence supports this, including the frequent remission of depression when someone finds a new strategy or truly gives up a goal. Preliminary results from our epidemiological study confirm this finding in a community sample. The next step is to find more efficient ways to measure and record information about goal pursuit in humans, and to look for the psychological and brain mechanisms that normally regulate motivation and mood. With this information in hand, it should be easier to find the genes that influence vulnerability to depression.

There is a strong human tendency to seek unitary explanations for diseases, and to think of multiple explanations as competing. This mistake has left most investigations of mental disorders seeking only one half of a full biological explanation. The remedy is to carefully pursue both evolutionary and proximate explanations for each disease. Our bodies are amazingly well designed in many respects, but they also have flaws that leave us vulnerable, flaws that make sense in an evolutionary perspective. There is every reason to think that the synergy between evolutionary and proximate approaches will soon bring major advances in our understanding of mental disorders (12).

References

1. Mayr E. The growth of biological thought: diversity, evolution, and inheritance. Cambridge: Harvard University Press, 1982.
2. Williams GW, Nesse RM. The dawn of Darwinian medicine. Q Rev Biol 1991;66:1-22.
3. Nesse RM, Williams GC. Why we get sick: the new science of Darwinian medicine. New York: Vintage, 1994.
4. Nesse RM. The smoke detector principle: natural selection and the regulation of defenses. In: The unity of knowledge. New York: New York Academy of Sciences; 2001;935:75-85.
5. Marks IM, Nesse RM. Fear and fitness: an evolutionary analysis of anxiety disorders. Ethol Sociobiol 1994;15:247-61.
6. Nesse RM. Is depression an adaptation? Arch Gen Psychiatry 2000;57:14-20.
7. Sloman L, Price J, Gilbert P et al. Adaptive function of depression: psychotherapeutic implications. Am J Psychother 1994;48:1-16.
8. Gut E. Productive and unproductive depression. New York: Basic Books, 1989.
9. Klinger E. Consequences of commitment to and disengagement from incentives. Psychol Rev 1975;82:1-25.
10. Emmons RA. Striving and feeling: personal goals and subjective well-being. In: Gollwitzer PM (ed). The psychology of action: linking cognition and motivation to behavior. New York: Guilford Press, 1996:313-37.
11. Carver CS, Scheier MF. Origins and functions of positive and negative affect: a control-process view. Psychol Rev 1990;97:19-35.
12. Nesse RM. Proximate and evolutionary studies of anxiety, stress, and depression: synergy at the interface. Neurosci Biobehav Rev 1999;23:895-903.