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WHAT DARWINIAN MEDICINE OFFERS PSYCHIATRY

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Since the late 1970s psychiatry has accumulated an enormous base of knowledge and remarkably effective new treatments. Despite this new knowledge, however, fundamental disagreements persist about the very nature of psychopathology. While the need for a "bio-psycho-social model" of mental disorders is widely acknowledged, psychiatry remains split into factions, some that emphasize genetic/physiological factors and others that focus on life events and psychological mechanisms. Some emphasize that mental disorders are "diseases like all others" while others decry this "medical model" and insist that many mental disorders can only be understood in their social context.

An evolutionary approach to medicine poses equally thorny conceptual issues. These difficulties include confusion about the distinction between proximate and evolutionary explanations, the need to recognize vulnerabilities instead of diseases as the object of evolutionary explanation, and the difficulties of finding adequate tests for many evolutionary hypotheses. In addition, a wrenching change in perspective is required to look for possible benefits of traits or genes that also cause disease and to begin to ask why all members of a species are susceptible to a disease, instead of why some individuals get sick and others do not. Given difficulties faced by Darwinian medicine, its application to psychiatry might seem to be a case of the blind leading the befuddled. But in fact, psychopathology has been a major early target for systematic and somewhat successful evolutionary approaches to disease. Why? The explanations are partly historical and partly a matter of the obvious need to address some long-standing problems.

While medicine has long had a foundation in the functional understanding provided by physiology, psychiatry has no comparable understanding of normal human behavior. The field of ethology has successfully traversed this

path, and psychiatry is now where ethology was in the 1960s: trying to compensate for the lack of a theoretical foundation by striving for scientific rigor in detailed observations and measurements about carefully defined categories. As soon as ethology found its bedrock in the principles of evolutionary biology, it took off in a still-growing burst of scientific advances (Alcock, 1993; Krebs and Davies, 1991). The hope that similar progress may be possible for understanding human behavior explains the early interest of many psychiatrists in an evolutionary approach. In addition to hopes fostered by history, evolutionary approaches are also spurred by the need for a framework that can accommodate the complexity of psychiatric disorders. The human tendency to oversimplify causation has resulted in separate foci on genetic and environmental causes of mental disorder, leaving chasms where there should be studies of interaction effects. Distinguishing carefully between proximate and evolutionary causation and between defenses and defects may help to bridge this gap.

This chapter will not attempt to summarize all the work in evolutionary psychology or even all of evolutionary psychiatry. Excellent overviews of work in evolutionary approaches to human behavior are available (Barkow, 1989; Barkow et al., 1992; Betzig, 1997; Eibl-Eibesfeldt, 1983; Konner, 1983, 1990; Ridley, 1993; Smith and Winterhalder, 1992; Wright, 1994). Work specifically about psychopathology has been summarized in several review articles (McGuire, 1977; McGuire et al. 1992; Nesse, 1984, 1991a), textbook chapters (Gardner, 1995; Konner, 1995) and books on the topic (McGuire and Fairbanks, 1977; McGuire and Troisi, 1998; Stevens and Price, 1996; Wenegrat, 1984, 1990).

Instead of reviewing all the available material, this chapter will use the categories that proved appropriate for Darwinian medicine (Nesse and Williams, 1998; Williams and Nesse, 1991) to see how useful they can be in the field of psychiatry. Evolutionary explanations for the vulnerabilities that make us susceptible to disease fall into just a few categories. First, there are novel environmental factors that change faster than our bodies can evolve. Many of these novel factors are aspects of our human-constructed environment, but novelty also emerges constantly from arms races between competing organisms, either pathogens and hosts, predators and prey, or members of the same species. Second, there are design trade-offs that offer an advantage overall, but that leave us vulnerable to disease. Some such trade-offs are genes that cause disease but give a net fitness advantage. Others are traits that remind us that every aspect of the body is a compromise. Third, there are constraints, that is, limits on what natural selection can do because of its stochastic nature and the impossibility of "fresh starts" because every generation must compete and survive. Finally, there are accidents and mishaps that cause disease that is unrelated to the evolutionary process, except insofar as the body can or cannot prevent or repair the damage (see table 14.1).

Of particular importance for psychiatry is another category, not a true cause of susceptibility to disease, but one that is often confused with diseases-namely, defenses. Examples include the capacities for pain, fever, nausea,

Table 14.1 Evolutionary explanations for disease

1. Novelty
   a. From pathogens or competitors
   b. From aspects of the modern environment
2. Trade-offs
   a. Genes with costs as well as benefits
   b. All traits have positive and negative trade-offs
3. Constraints
4. Accidents and mishaps too rare to shape defenses

(5. Defenses that are often confused with diseases.)

vomiting, cough, diarrhea, and fatigue. Aversive emotions like anxiety and sadness almost certainly have similar origins and functions. We turn first to these defenses.

Defenses, Normality, and Emotions

Much that seems abnormal about the functioning of the brain and the body is not abnormal at all. Many manifestations of illness are not defects in the body's mechanisms, but sophisticated adaptations. They are observed only when aroused by cues that indicate a situation where they may be helpful. For instance, pain is aroused by cues that indicate tissue damage. It is not an abnormality itself, but a useful defense. People born without the capacity for pain are usually dead by their early 30s (Melzack, 1973).

Some manifestations of disease, such as seizures, jaundice, coma, and paralysis, arise from defects in the organism. But many other manifestations of disease are defenses. Coughing clears foreign matter from the respiratory tract. Vomiting eliminates toxins from the stomach; diarrhea clears the colon. The capacity for fatigue protects against tissue damage. The low iron levels seen in chronic infection limit the growth of pathogens (Weinberg, 1984). Fever is a particularly subtle and well-regulated defense against infection (Kluger, 1979), but it sometimes goes so high that seizures result. This is a fine example of a cost that is maintained by the trade-off with its associated benefit. An evolutionary approach encourages sharp attention to distinction between manifestations of disease that are defects versus those that are defenses, and it forces us to acknowledge that much suffering can be adaptive.

Most of the problems people bring to psychiatrists involve aversive emotions. Patients complain of sadness, anxiety, jealousy, anger, or boredom. To understand these complaints thoroughly, we need a comprehensive theory of emotions, something that is just now being formulated. Emotions researchers have reached a consensus that the fundamental emotional capacities have been shaped by natural selection because they give a selective advantage (Ekman, 1992; Frank, 1988; Frijda, 1986; Nesse, 1990). Plutchik, in particular, has advanced this line of reasoning to understand psychiatric disorders (Plutchik,

1980; Plutchik and Kellerman, 1989). Aversive emotions like anxiety and sadness are almost certainly examples of defenses (Barlow, 1991; Morris, 1992; Tooby and Cosmides, 1990a,b). Recognizing the utility of aversive emotions has implications for the whole problem of distinguishing normality versus pathology in general, a long-standing conundrum for psychiatry. It is to this large topic that we first turn.

It is easy to imagine how natural selection could have shaped the coherent and consistent patterns of physiology, behavior, cognition, and subjective experience that we recognize as emotions. If our ancestors repeatedly encountered certain situations that posed consistent adaptive challenges, then individuals whose bodies adjusted so they could cope especially well with those challenges would have a selective advantage (Nesse, 1990).

Take, for instance, male sexual jealousy. Daly and Wilson, among others, have argued that this capacity is a mate-guarding tactic which, if absent, is likely to result in a man having fewer children because his wife may sometimes be pregnant with the children of other men and because he will likely invest much parental effort in children fathered by other men (Daly, Wilson, and Weghorst, 1982; Symons, 1979). Although jealousy is an uncomfortable, undesirable state that can give rise to antisocial acts, it nonetheless is likely to increase reproductive success and therefore be maintained by natural selection. Consistent with this hypothesis, sexual jealousy is more intense in males than in females across a wide variety of cultures, despite the enormous cultural differences in the situations that arouse jealousy and the intensity and nature of its expression (Daly et al., 1982). Furthermore, there are sex differences in the cues that arouse jealousy. For men, cues to infidelity are more potent, while for women, losses of relationships and resources are stronger (Buss et al., 1992).

Such knowledge about the functions of normal jealousy is useful in understanding pathological jealousy. Although psychiatrists often interpret pathological jealousy as reflecting repressed and projected sexual desires, an evolutionary approach encourages the clinician to investigate whether the jealous partner has reason to believe that the spouse may well prefer someone else. Thus, during a period of loss of status or resources, or low self-esteem caused by depression, a man is especially likely to begin acting more jealous. This often precipitates a withdrawal of affection by the spouse, which is interpreted as confirmation of suspicions of infidelity, and the escalating feedback spiral can easily end a marriage or even lead to tragic violence.

Compared with jealousy, the utility of anxiety is more readily recognized, but exactly how it increases fitness is not so easy to describe. More than 60 years ago, Cannon (1929) documented the many benefits of the "fight-flight reaction" in the face of serious danger. He noted that everything about fear-from the cooling sweat, to the trembling that indicates tense muscles, to the increased clotting of blood and shortness of breath and rapid-pounding heartbeat-were all useful when one needed to flee or fight. Today we recognize this same pattern of responses as a panic attack. We now know much more about the proximate mechanisms that mediate these responses, but our knowl-

edge base about its functions and evolutionary origins remains quite crude. Even a basic understanding can be useful, however. In the case of panic, it can account for the syndrome of shortness of breath, a wish to gee, pounding heart, sweating, and thinking of nothing but escape. Agoraphobia, fear of leaving the home, is almost always a complication of panic attacks. What is the connection? There are various proximate explanations, but from an evolutionary point of view, if you have just experienced life-threatening danger, it is wise indeed to stay close to home or to go out only with a companion.

Marks and I have argued that the different subtypes of anxiety disorder and their behavioral characteristics demonstrate a remarkable correspondence to the different kinds of danger that humans are likely to have experienced during the course of evolution (Marks and Nesse, 1994). Thus, fear of heights is manifested by freezing. The sight of blood is the only fear that causes fainting, a useful response if you are bleeding (Marks, 1988). A looming predator causes flight. Social threats cause embarrassment and social anxiety. This approach to the subtypes of anxiety obviates many of the difficulties posed by a system of discrete anxiety disorders. Enormous effort has gone into trying to determine whether the anxiety disorders arise from one underlying disorder or if they are variety of distinct diseases. An evolutionary approach suggests that the various manifestations of anxiety have been partially differentiated by natural selection into incompletely differentiated subtypes, each designed to cope with a particular kind of threat. If this is correct, it becomes less urgent to ask, for instance, whether social phobia and generalized anxiety disorder are essentially different or essentially the same. Both are overlapping response patterns to somewhat related dangers.

An evolutionary view also assists in dismantling the false dichotomy of instinct and learning. Research has begun to reveal the specialized learning mechanisms that mediate fear (Marks, 1987; Marks and Tobena, 1990; Mineka et al., 1980; Ohman and Dimberg, 1984). It has long been apparent that people are more likely to be afraid of heights, spiders, or the dark than they are of leaves, butterflies, or sunshine. For that matter, they are more likely to be afraid of a garter snake, than some dangerous things like guns, knives, and greasy hamburgers. In earlier work on preparedness, certain kinds of cues were found to be more readily paired with certain kinds of responses (Garcia and Ervin, 1968; Seligman, 1970). For instance, it is easy to condition a nausea response to a taste cue, and easy to condition a limb withdrawal response to a pain, but difficult to switch the cues and responses around. Fundamental research on learning is based increasingly on its evolutionary origins and functions, with attempts at integration making good progress (Staddon, 1983).

Mood and Its Disorders

An evolutionary explanation for the capacity for high and low mood has been a major, albeit unachieved, goal of evolutionary psychology. So far, even defining the terms remains problematic. The distinctions among grief, sadness,

and depression remain fuzzy, and we do not even know if these intuitive concepts reflect functionally distinct subsets of a single experience, separate subcategories, or variations on the same response.

One major school of thought has emphasized the role of status changes in the regulation and functions of mood. British psychiatrist John Price, after recognizing a connection between pecking orders in birds and primate status hierarchies, began work on the behaviors that follow a rise or fall in status (Price, 1967; Price and Sloman, 1987). This work has been expanded and extended (Gardner, 1982; Gilbert et al., 1995; Price and Sloman, 1987), into a theory that emphasizes the role of depression in creating "involuntary yielding" and the benefits of voluntary yielding-that is, giving up a status competition-in relieving depression (Gilbert, 1992).

The epidemiological studies of Brown and colleagues show the profound effects of losses in precipitating depression, and the role of threats in precipitating anxiety disorders (Brown and Harris, 1978; Finlay-Jones and Brown, 1981). In recent work from an evolutionary vantage point, Brown has reanalyzed these data and found that much of the variance in the loss events is related to experiences of humiliation or entrapment (Brown et al., 1995).

Raleigh et al. (1983,1991) have studied related mechanisms in vervet monkeys. They found that whole-blood serotonin levels are high in the dominant male, but fall precipitously with loss of rank. Monkeys that receive drugs that increase serotonin levels (versus a placebo) reliably take over a group that has no alpha male, while monkeys that receive drugs that block the effects of serotonin are consistently displaced by those receiving a placebo. I know of no other studies that so clearly demonstrate the two-way street between social circumstances and brain chemistry.

Another tradition of work on depression goes back at least to the psychoanalyst Bibring. He observed that depression often arises when a person is pursuing an unattainable life goal, and often remits when that goal is achieved or given up (Bibring, 1953). Gut (1989) has reinterpreted and extended this work in an evolutionary context. She emphasizes the adaptive significance of stopping current activity after a loss, conserving energy, and thinking hard about the current situation and the available alternatives.

I have emphasized similar functions for mood as a regulator of patterns of resource investment among a variety of enterprises with differing patterns of risk and payoff (Nesse, 1990, 1991b). High mood tends to increase fitness in situations of opportunity, whereas low mood tends to stop investment in hopeless endeavors and facilitates consideration of alternative strategies or enterprises. In short, low mood is a coordinated pattern of responses that are useful in unpropitious situations, whether the loss is discrete or continuing. Momentary changes in positive and negative states may help to fine-tune the tactics required for coping with everyday social life.

The relationship between sadness and depression remains problematic. Sadness is almost certainly adaptive, but depression may arise from dysregulated sadness or from an entirely separate mechanism. Also unanswered is the question of why 15% of the population is especially susceptible to anxiety

and mood disorders (Kessler et al., 1994). Vulnerability to the severe mood disorders is mediated substantially by genetic factors. Why the responsible genes persist in the gene pool is another unanswered question. In the case of manic-depressive disorder, it has been proposed that increased reproductive success during periods of high mood may counterbalance the periods of low reproductive success during low mood (Wilson, 1992).

Attachment and Relationships

Human emotions usually arise, of course, in the context of relationships. This is not the place to review the complete history of work in this field, but Rene Spitz's studies of the high death rates in orphanages inspired Harlow (1974) to carry out his well-known studies that showed infant monkeys prefer milk-less terry-cloth "mothers" to wire-mesh "mothers" with a milk source. These studies showed that both behaviorists and the psychoanalysts were wrong in their supposition that attachment arises out of drive satisfaction. Influenced by these studies, and by contact with ethologist Konrad Lorenz, Bowlby (1969) arrived at a view derived from psychoanalysis but informed by ethology. Bowlby's emphasis on the adaptive functions of attachment led to studies by Ainsworth and others (1978) demonstrating strong correlations between parental behavior and children's attachment styles and between a child's attachment pattern early in life and in later years. The strong implication through most of these studies has been that the attachment style is shaped by the mother's behavior early in life. It is now clear, however, with recent studies by Kagan et al. (1987) that these patterns are strongly influenced by genetic factors. Furthermore, the suggestion has been made by that the "abnormal" styles of attachment, specifically anxious attachment and ambivalent attachment, may in fact be adaptive in certain situations (Chisholm, 1996). If the mother is disinclined to invest in a child, ordinary secure attachment may be less beneficial than anxious or avoidant attachment.

In work at the interface between attachment and depression, Engel and Schmale (1972) have argued that the protest phase of primate infant separation alerts the mother, while the despair phase of quiet huddling conceals the infant from predators and conserves energy in much the same way as hibernation. They suggest that the similarities among depression, hibernation, and the despair phase of separation may arise from this mechanism.

Trivers's theory of parent-offspring conflict (Trivers, 1974) has been remarkably underutilized and even unrecognized in psychiatry. While many clinicians continue to imagine that the interests of the mother and her infant are identical, Trivers has shown that there are many situations in which a mother can maximize her reproductive success by investing less in an offspring than is in the offspring's interest. Because offspring share only half of the mother's genes, the fitness of the mother's genes depends on correctly allocating effort between having more offspring of her own or investing in the offspring she already has. The specific conflicts depend on the stage of life and

the available alternatives. Although Trivers noted the significance of conflicts during adolescence, he used the weaning conflict as an exemplar. At some stage of development it is to the infant's advantage to continue to nurse, but the mother's genetic interests would be better served by starting to invest in another offspring. Fierce battles arise that testify to the lack of concordance. As Trivers (1985:155) notes wryly, "An offspring cannot fling its mother to the ground and nurse at will. . . . Given this competitive disadvantage, the offspring is expected to employ psychological tactics. It should attempt to ***induce*** more investment than the parent is selected to give." In particular, Trivers suggests, the child may pretend to be younger and more helpless than it really is to try to deceive the mother into thinking it is still in her interest to provide more help than she would otherwise. This is, of course, a fine description of regression, and may explain not only the phenomenon of regression but also the annoyance we experience when we think we are being manipulated by people who act more helpless than they are.

Insight into the conflicts between offspring and parents has provided a foundation for a reinterpretation of some core psychoanalytic ideas. Slavin (1992) notes that it is in the interest of parents to suppress conflicts between siblings, and it is also in the parents' interests to manipulate their children to cooperate more than is in the children's best (fitness) interests (Slavin, 1992). Slavin (1992) proposes that if this excess cooperation persists into adulthood, the result is neurosis, or worse. Several other authors, notably Badcock (1988) and Rancour-Laferriere (1985), have gone much further and tried to understand the possible adaptive significance of specific psychoanalytic phenomena such as the Oedipus complex and castration anxiety. Daly and Wilson (1992) however, note the ignorance of most psychoanalysts about fundamental evolutionary principles. They have proposed alternative evolutionary explanations for what have been described previously as Oedipal phenomena.

Trivers (1976) and Alexander (1974) have each proposed that a mechanism for limiting self-knowledge might allow individuals to unconsciously, and thus more successfully, pursue strategies of deception in reciprocity exchanges (Lockard and Paulhus, 1988). This explanation must, however, be incomplete, given that we also repress the prods of conscience as well those of the drives. Lloyd and I (Nesse and Lloyd, 1992) have attempted to understand the origins of the psychodynamic defenses, and more specifically to understand ways in which a lack of knowledge about one's own motives might give a selective advantage. Much work remains to integrate psychodynamics with a modern approach to evolution, but a start has been made (Badcock, 1988; Stevens, 1982; Wenegrat, 1990).

The principle of kin selection has proved particularly powerful in explaining cooperation and lack of cooperation in the family (Essock-Vitale and Fairbanks, 1979; Lancaster et al., 1987). Perhaps the single most dramatic finding in the field so far has come been made by the behavioral ecologists Daly and Wilson. On considering the phenomenon of fatal child abuse, they wondered how this apparent anomaly could be reconciled with the evolutionary expectation that parents will go to great lengths to protect their offspring. They

hypothesized that perhaps the children who were killed were not, in fact, the biological offspring of both parents in the home. When they did the study to test this hypothesis, they found that the rates of child abuse in homes with at least one step-parent were at least 70 times higher compared to homes with both biological parents present (Daly and Wilson, 1981, 1989). This finding could not be accounted for by confounding factors such as alcoholism, poverty, or mental disorders. The discovery of this extraordinarily powerful effect is highlighted, in contrast by decades of studies by child protection specialists that had uncovered factors that are weak by comparison.

Marriage and Other Relationships

Understanding of the origins of conflicts over mates and mating strategies (Hrdy, 1981) has given significant new insights into marriage and its vicissitudes. Buss (1992) has documented, in a study of 37 human cultures, that in choosing a mate, appearance is consistently more important to men, and wealth is consistency more important to women. Given that variance in reproductive success is quite limited in women, but can be large in men, the two sexes can and do pursue different and often conflicting mating strategies (Daly and Wilson, 1983). These distinct strategies help to account for patterns of cooperation and conflict and are proving useful in understanding associated marital pathology (Kerber, 1994).

Based on these principles, Fisher (1992) has summarized data from many societies that shows an average duration of marriage of less than 5 years. She argues that this is sufficient time to provide initial nurturing to human offspring, and that a mechanism ("planned obsolescence of the pair-bond") may have evolved to end marriages after this period of time. Although the data are clear enough, it remains unclear why a specific mechanism would evolve to end pair-bonds when spouses are certainly capable of discerning their individual interests and pursuing them in a more flexible strategy of staying or leaving (Betzig et al., 1988; Hrdy, 1981). Of course, social pressures on mating patterns are profound, so it should be no surprise that an individual's decisions are often severely constrained.

Reciprocity has long been recognized as the engine motivating nonkin relationships, even before Trivers's (1971) seminal paper, but its special significance to psychology results from two major discoveries. The first is that kin selection now explains much that was previously attributed to group selection (Hamilton, 1964; Williams, 1966). Much altruistic behavior benefits genes identical to those of the altruist that are in another (related) individual. With this perspective, the special relationships between blood relatives are expected. Although opportunities for conflict remain, much self-sacrificing behavior is expected as a result of kin selection.

The second discovery is that tit-for-tat is a robust strategy for maximizing the payoff in reciprocity interactions (Axelrod, 1984; Axelrod and Hamilton, 1981). When people trade favors, an exchange that can be seen as the foun-

dation of human social life, there is always the risk that the other will not reciprocate. If the maximum long-term benefit comes from repeated cooperation, but the maximum short-term benefit comes from defecting, a problem of strategy arises. When Axelrod invited scientists to submit programs for how to deal with this dilemma, the winner was "tit-for-tat," that is, cooperate on the first move of the game, and for every subsequent move, do what the other person did last time. The strategy takes advantage of cooperation when that is possible but avoids exploitation.

Cosmides and Tooby (1989) have provided data supporting the existence of specific mental algorithms to detect cheaters, a finding that could have importance for states of paranoia. Others have argued that sociopathy is a specialized evolved strategy for taking advantage of human dependence on reciprocity exchange (Harpending and Sobus, 1987; Mealey, 1995).

If the social emotions-friendship, anger, suspicion, anxiety, and guilt-are viewed as adaptations designed to deal with different outcomes in a reciprocity exchange, then this begins to explain some of the complexity and randomness of the expression these emotions (Frank, 1988; Nesse, 1990) and their variable development and association with various kinds of psychopathology. In particular, specialized emotions of friendship and affection may arise from trustworthy exchanges. When you think that the other may defect, suspicion is aroused, while if the defection actually takes place, anger signals that the defection is unacceptable and must be remedied if the relationship is to continue. Conversely, when we are tempted to defect, anxiety often inhibits our actions after a defection, most of us feel guilty, which induces states of self-punishment that are hard to understand from other perspectives but that make sense as a way to reestablish a reciprocity relationship after a defection (table 14.2).

This chapter has so far addressed various aspects of human behavior that are often thought to be abnormal but in fact can be recognized either as defenses or as behavior patterns that tend to increase reproductive success. What kinds of research projects will be needed to advance work in this area? It is essential to keep separate several questions about such defenses.

First, there is the question of the function of the basic capacity for a defense, such as anxiety or mood. Recast, this question asks why individuals with this capacity have a selective advantage over those who do not. To address the question, one uses the same approaches used by physiologists in ascertaining

Table 14.2 The social emotions and the Prisoner's Dilemma

	Other cooperates	Other defects
Self cooperates	Friendship, trust	***Before:*** Suspicion ***After:*** Anger
Self defects	***Before:*** Anxiety ***After:*** Guilt	Hatred, mistrust, rejection

the functions of an organ: take it out, block its effects, or look at what happens to people who have innately high or low levels. Such studies face special difficulties. People who report few experiences of the negative emotion may lack the capacity, but they also may simply be living fortunate lives. Then, also, there is the expectation that natural selection will set the regulation of inexpensive defenses to a hair-trigger (the "smoke detector principle"), so that many expressions of the defense will be unnecessary. We don't even have solid documentation of the value of fever in many everyday infections, so it may be some time before we have evidence for the value of low mood.

Second is the question of how a defense is regulated. This involves multiple levels, from the psychological to brain chemicals. Interestingly, it appears that we know more about the brain mechanisms than we do about the psychological mechanisms that regulate mood. The range of factors that can explain within-individual variation is daunting: diet, exercise, sunlight, changes in social status, sexual experiences, martial changes, family conflicts, work stress, social support, community structure changes, substance use and abuse, health, sleep patterns, cognitive patterns, media exposure, trauma, opportunity, and myriad other factors. Studies of the same individuals with frequent sampling over days and months may begin to give answers to these questions.

Third is the question of why people differ in their tendencies to certain emotions like low mood and anxiety. Unpacked, this question includes many facets, such as baseline levels of mood and anxiety, overall emotionality, responsiveness to positive events, responsiveness to negative events, duration of responses, cognitive patterns that mediate responses, and responsiveness to social cues. There is now strong evidence that genetic factors have strong effects on baseline mood levels and tendencies to mood disorders, but we do not yet know whether these effects are direct or mediated by genetic effects on cognition or stress responses.

Fourth, there are questions about differences in these reactions between groups. Rates of major depression vary lo-fold between different countries (Weissman et al., 1996). Although this variation may be explained by the above factors, it may also require systematic assessment of cultural and family structures to see how they interact with the regulation mechanisms.

The research agenda to study emotions and other defenses is crowded. Even as it is just getting started, an evolutionary approach helps to clarify the questions and suggest possible answers.

Novelty

Arms Races

In general medicine, evolutionary arms races are most clearly manifest in pathogen-host competitions. We are still susceptible to infection after all these eons of exposure because pathogens evolve much faster than we do and so can outflank nearly any defense our body evolves (Ewald, 1993). Some psychiatric

illnesses may turn out to result from infection with agents that have evolved to evade our defenses. In particular, some cases of schizophrenia may result from in utero exposure to viruses (Kendell and Kemp, 1989). Also, there are some recent data suggesting that some cases of obsessive-compulsive disorder may arise after streptococcal-infection-precipitated autoimmune damage to the basal ganglia, including the caudate nucleus which is strongly implicated in the pathophysiology of obsessive-compulsive disorder (Allen et al., 1995) These findings are by no means conclusive, but would tie together many confusing aspects of obsessive-compulsive disorder and would help to explain the pattern of onset, the association with Sydenham's chorea, the pattern of blood antibodies observed in patients and controls, and the genetic predisposition to obsessive-compulsive disorder.

Another kind of arms race has already been alluded to, that between members of the same species. A number of authors have emphasized the possibility that intense human-human competition has shaped the rapid increase in human brain size and intelligence and cognitive complexity (Alexander, 1979; Dawkins, 1976; Humphrey, 1976). The results of such a competition could also explain, as some of the previous authors of works on evolution and psychodynamics have shown, some of the inordinate complexity of human psychology. If our empathy is a tool to help us manipulate others and avoid being manipulated, then layer on layer of complexity may have been laid down by the process of natural selection (Krebs and Dawkins, 1984).

Novel Aspects of the Physical and Social Environment

A large proportion of disease now results from novel aspects of our physical environment. For instance, atherosclerosis, the complications of which will prove fatal for more than a third of us, is due mainly to our evolved preference for fats and the current ready availability of kinds of fats that were less common in the ancestral environment (Eaton et al., 1988). Substance abuse, auto accidents, many infections, cancer, Alzheimer's disease, and other diseases of old age are also experienced almost exclusively in the modern environment. It is enough to make one long for life in the Stone Age. Until, that is, one thinks for a moment about the even greater burden posed by other diseases in the Paleolithic. The same goes for psychiatric disorders. While it is undoubtedly true that some proportion of current mental disorders arise because of our modern environment (Glantz and Pearce, 1989), it is by no means correct that this implies we would be mentally better off in an earlier environment. It is possible that mood and anxiety disorders are less frequent in more traditional cultures, but we do not yet know.

Substance abuse is the most significant mental disorder that is a product of our novel environment. While people in all cultures and times have used drugs, ready availability of a steady supply of potent agents, in conjunction with novel means of administration such as hypodermic needles and crack pipes, has made substance abuse an international plague. An evolutionary view makes one hesitant to seek simplistic solutions (Nesse, 1994). Organisms

are designed to repeat behaviors that stimulate reinforcing mechanisms in the brain. When these mechanisms are stimulated directly by drugs instead of by natural experiences, drug-seeking behavior can take over the organism's behavioral control mechanisms (Nesse and Berridge, 1998; Pomerleau, 1997). Although it is not surprising that people vary in their genetic susceptibility to addiction, it is also not surprising that untoward circumstances also make certain people especially vulnerable. The genes that predispose to substance abuse are sometimes called defects, but given that they probably imposed no harm in the Paleolithic, it would be much more appropriate to call them "quirks."

Obesity and eating disorders also arise from our novel environment. In the Paleolithic, high-calorie foods were apparently rarely available without substantial effort, or else natural selection would have shaped more powerful mechanisms to restrict food intake. Given the selective power of famine, it is also unsurprising that many people have a tendency to add more pounds than are necessary or desirable in a modern environment. Severe eating disorders seem to be peculiarly modern phenomena, arising mainly in the past few score years in technological societies (Kurth et al., 1995). Some combination of modern media, food availability, and cultural factors causes many young people to diet, which then leads to behavior appropriate to starvation, namely, seeking out and devouring any available high-calorie food. This loss of control causes increased fear of obesity and emotional upheaval that initiates a vicious cycle of attempts at self-control by dieting, binge eating, followed by guilt and helplessness, more dieting and more binge eating. A small proportion of people who begin this cycle are able to more and more strictly control their intake to the point it is manifest as anorexia nervosa. The absence of menstrual periods associated with anorexia may well be adaptive in a natural environment, where it would be wasteful to become pregnant when food supplies are inadequate (Condit, 1990; Surbey, 1987; Voland and Voland, 1989).

Some aspects of modern life are so ubiquitous that we rarely notice how unusual they are. For instance, spending time is classrooms learning to read is completely novel. It is hard to see what dyslexia and attention deficit disorder would have been like in the Paleolithic. For another example, the simple invention of electric lights seems to have changed people's sleep patterns profoundly. When people enter a sleep lab with lighting that matches that of the sun, they typically make up a cumulative sleep deficit of 15 hours before settling in to a pattern of sleeping approximately nine hours per night with nightly periods of restful wakefulness.

Even conflicts between the sexes may be viewed as a product of novel environments. When it was not possible for one person to amass significant resources, no one man could control more than a few women. When agriculture became organized, however, men promptly used surpluses to control other men to control large number of women in harems (Betzig, 1986). Smuts (1995) has looked at patterns of social organization and suggested that the relative absence of female-female bonds in some cultures makes women far more vulnerable to male aggression.

One of the more worrisome recent epidemiologic findings suggests that an extraordinary epidemic of depression may be upon us. In nine separate epidemiological studies, the rates of depression in young people are far higher than those over a lifetime for older people in the same country (Cross-National Collaborative Group, 1992). Although these studies deserve criticism on methodological grounds, it also seems plausible that changing social patterns and technological changes may indeed be changing rates of depression. If so, we have an even more urgent need to understand the functions and regulation of normal mood.

Genes

In psychiatry, as in the rest of medicine, genes that predispose to common disorders may often also offer benefits that account for their continued frequency, or they may cause disease only in interaction with aspects of the modern environment. Early hopes that we would be able to identify one or two specific genes to account for schizophrenia or manic depressive illness have foundered, but even if multigenic effects are responsible, an evolutionary explanation for their prevalence can be helpful.

For instance, schizophrenia appears to be quite constant at a prevalence of about 1% in diverse human groups. In less modern societies, the course of the disease may be somewhat more benign, but the prevalence is nonetheless similar (Gottesman and Shields, 1982). Given evidence for strong genetic factors that predispose to schizophrenia, one must ask why these genes have persisted given the reproductive disadvantage they cause (Slater et al., 1971). Many authors have suggested possible pleiotropic benefits to the genes that cause schizophrenia. Early, probably erroneous, observations that people with schizophrenia could withstand cold and disease better than other patients were once thought to account for the finding (Jarvik and Chadwick, 1972). More recently, a variety of proposals for alternative explanations have been put forth, ranging from ability to discern the unseemly motives of others (Allen and Sarich, 1988), to sexual selection (Crow, 1993), and circadian regulation (Feierman, 1994). While one of these ideas could eventually prove correct, it seems equally plausible that the relationship is far less direct, such as the possibility that people with a certain antigenic makeup are protected against certain infections but they are vulnerable to autoimmune or other factors that interfere with brain development.

Depression and anxiety are not defects like schizophrenia, but defenses whose regulation has gone awry. Thus, the genetic trade-offs that cause differences in susceptibility to mood and anxiety disorders are directly related to the higher level trade-off of the regulation of the normal emotion. One cannot help but wonder why there is such between-individual variability in regulation of these emotions. The answer may be that different response patterns are optimally adaptive in different environments. This would be a special case of the proposal that differences in personality are adaptations for different social niches (Buss, 1991; Tooby and Cosmides, 1990a)

Alzheimer's disease may be an example of a manifestation of a pleiotropic gene with benefits early in life and a cost later in life (Albin, 1994). The justification for this speculation is the extraordinary frequency of Alzheimer's disease, affecting more than half of the people over the age of 90. So far, however, no one has identified benefits earlier in life for people who carry the tendencies to Alzheimer's disease, although factors associated with apolipoprotein variants could provide an explanation.

Trait Trade-offs

Every trait in the body is a trade-off between multiple benefits and costs. Heavier bones would break less easily, but they would be more expensive to create and maintain. Lower blood pressure or blood glucose would cause less damage to tissues, but only at the cost of decreased ability to respond to situations that require sudden exertion.

Such trade-offs probably account for many aspects of psychiatric illness. As noted above, too little anxiety may be even worse than too much. On the level of life strategy, there are disadvantages to investing too little effort in parenting, and disadvantages to investing too little in mating. The patterns of modern life are quite peculiar along these lines because the environment does not require us to each allocate effort proportionately among the tasks of growth, development, protection, mating, and parenting. Instead, an individual can devote essentially all his or her resources to one area, neglecting all the rest. Furthermore, thanks to caffeine, electric lights, and grocery stores, an individual can, for instance, spend nearly all of his or her waking life simply reading and writing, a thoroughly abnormal pattern of activity that necessitates neglect of other tasks. Studies that relate overall patterns of allocation of life effort to patterns of symptoms have yet to be done.

Historical Constraints

The body offers many illustrations of natural selection's inability to start fresh and create a sensible design instead the jury-rigged bodies we have. Our eyes are inside out, causing blind spots. Our respiratory passages intersect within the esophagus and food can thus choke us. The awkward design of the spine causes most of us pain at one time or another. There are undoubtedly similar examples in our mental mechanisms, but because the connection between the structure and the function is so much more difficult to ascertain and because of the difficulties of carrying out comparative or historical studies, this is a nebulous area. Our patterns of reasoning may, however, provide an important example. We all routinely make substantial errors in logic that reveal underlying biases and rules of thumb (Nisbett and Ross, 1980; Tversky and Kahneman, 1974) Rules of thumb substitute for logic perhaps because the first cognitive mechanisms were based on these crude rules, and there has never been a selective force that could supplant them or make the leap to primary de-

pendence on Bayesian thinking. The mystery is that we have a capacity for logical thought at all. It seems quite possible that this is either an epiphenomenon or a product of sexual selection (Miller, in press). Our emotional reactions may likewise reflect the continuity with our predecessors. Studies of primate cognition may eventually intersect with the studies of human cognitive and social psychology to give us more knowledge about these possibilities.

Conclusions

Given that there is not yet an evolutionary psychology that can provide psychiatry with knowledge comparable to what physiology provides for the rest of medicine, can an evolutionary approach still be useful to psychiatry? As documented in the preceding pages, the answer is yes. The danger now is in taking this preliminary knowledge too far. Several caveats may help. First of all, I would argue that Darwinian psychiatry is not, and should not be, a field of medical practice, but only a basic science that offers us insight and good questions to ask. A Darwinian approach to human nature and human psychopathology is essential to our understanding, but it gives no direct treatment recommendations whatsoever. As is the case with the rest of medicine, clinical recommendations must come from clinical studies. In the area of psychiatry, one must also reinforce the warning that behavior and ethical precepts cannot be derived from biological knowledge. For instance, just because males have more of a tendency than females to abandon their families tells us nothing whatsoever about the moral significance of such acts. Such warnings about the naturalistic fallacy are common in the literature on evolution and psychology, but this is for the good reason that many people, despite warnings, readily draw norms from facts. Furthermore, our understandings of the origins and functional significance of aspects of culture, and the biological underpinnings that make it possible for individuals to absorb culture, remind us that our understanding is rudimentary and is not to be trusted as a guide for action.

With these caveats in mind, however, an evolutionary view already offers an extraordinarily useful guide for understanding our patients. Even our crude state of knowledge takes us a giant leap from the idiosyncratic theories without biological foundation that have characterized much of the history of psychology. Furthermore, the age-old problem of "normality" makes much more sense in an evolutionary perspective. From a perspective of reproductive success, many emotions, cognitions, and behaviors patterns that have been thought of as pathological can be recognized as adaptive, even if often distressing or socially prohibited. And for some of these, like anxiety, it is clinically useful to explain their functions to patients. When people who experience panic attacks learn that their symptoms are the normal fight-flight response that is going off at the wrong time, this often helps them give up their fears that the symptoms are caused by heart or brain disease.

These principles are likely to have early application as a foundation for a revised perspective on psychiatric diagnosis. The successive ***Diagnostic and***

Statistical manuals published by the American Psychiatric Association have been essential in allowing researchers to study comparable groups, but they have also been widely criticized because they lack a foundation in theory and they encourage simplistic thinking of psychiatric syndromes as discrete diseases (Kendell, 1984). An evolutionary view, by contrast, requires us to use what we know about the emotions and their regulation and thus to distinguish several categories of mental disorders. First, there are conditions that are painful and perhaps undesirable, but normal and evolutionarily useful, like much jealousy, anxiety and sadness. Second, there are conditions that arise from an initially normal brain that has been exposed to learning or trauma that leads to emotional pathology or maladaptive behavior. Third, there are conditions, like most depression and panic disorder, that arise from dysregulation caused by genetic vulnerability interacting with environmental factors. Fourth, there are conditions that arise from primary abnormalities of brain tissue, like obsessive-compulsive disorder, schizophrenia, and Alzheimer's disease. In contrast to current diagnostic systems that encourage separation of a patient's difficulties into many "co-morbid" conditions, an evolutionary approach fosters exploration of all the factors that interact to account for a person's emotional and behavioral difficulties.

Finally, the greatest current value of a Darwinian perspective on psychiatry is its heuristic utility. Without an evolutionary perspective, Daly and Wilson never would have asked about the frequency of child abuse in families with a step-parent present. Without an evolutionary perspective, it is far harder to even imagine the possible benefits of anxiety, sadness, and grief. Without an evolutionary perspective, it is hard to ask the right questions about the functional significance of various aspects of human nature, adaptive and maladaptive.

References

Ainsworth, M. D., Blehar, M. C., Waters, E., and Wall, S. (1978). ***Patterns of Attachment: A Psychological Study of the Strange Situation.*** Hillsdale, NJ: Lawrence Erlbaum.

Albin, R. L. (1994). Antagonistic pleiotropy, mutation accumulation, and human genetic disease. In M. R. Rose and C. E. Finch, eds., ***Genetics and the Evolution of Aging.*** Amsterdam: Kluwer.

Alcock, J. ***(1993). Animal Behavior,*** 5th ed. Sunderland, MA: Sinauer Associates.

Alexander, R. D. (1974). The evolution of social behavior. ***Annual Review of Systematics,*** 5, 325-383.

Alexander, R. D. ***(1979). Darwinism and Human Affairs.*** Seattle: University of Washington Press.

Allen, A. J., Leonard, H. L., and Swedo, S. E. (1995). Case study: A new infection-triggered, autoimmune subtype of pediatric OCD and Tourette's syndrome. ***Journal of the American Academy of Child and Adolescent Psychiatry 34(3), 307-311.***

Allen, J. S., and Sarich, V. M. (1988). Schizophrenia in an evolutionary perspective. ***Perspectives in Biology and Medicine 32(l), 132-153.***

Axelrod, R. (1984). The ***Evolution of Cooperation.*** New York: Basic Books, Inc.

Axelrod, R., and Hamilton, W. D. (1981). The evolution of cooperation. ***Science,*** **211,** 1390-1396.

Badcock, C. (1988). ***Essential Freud.*** Oxford: Basil Blackwell.

Barkow, J. H. (1989). ***Darwin, Sex, and Status: Biological Approaches to Mind and Culture.*** Toronto: University of Toronto Press.

Barkow, J., Cosmides, L., and Tooby, J., (eds.) ***(1992). The Adapted Mind.*** New York: Oxford University Press.

Barlow, D. H. (1991). Disorders of emotions: Clarification, elaboration, and future directions. Psychological ***Inquiry*** **2(l),** 97-105.

Betzig, L. L. (1986). ***Despotism and Differential Reproduction: A Darwinian View of History. New*** York: Aldine Publishing Company.

Betzig, L., ed., (1997). ***Human Nature: A Critical Reader.*** New York: Oxford University Press.

Betzig, L., Mulder, MB., and Turke, P. (eds.). (1988). ***Human Reproductive Behavior: A Darwinian Perspective.*** Cambridge: Cambridge University Press.

Bibring, E. (1953). The mechanisms of depression. In P. Greenacre, ed., ***Affective Disorders*** (pp. **1348).** New York: International Universities Press.

Bowlby, J. (1969). ***Attachment. New*** York: Basic.

Brown, G. W., and Harris, T. (1978). ***Social Origins of Depression. New*** York: The Free Press.

Brown, G. W., Harris, T. O., and Hepworth, C. (1995). Loss, humiliation and entrapment among women developing depression: A patient and non-patient comparison. ***Psychological Medicine*** **25(l), 7-21.**

Buss, D. (1991). Evolutionary personality psychology. ***Annual Review of Psychology,*** **42,459491.**

Buss, D. (1992). Mate preference mechanisms: Consequences for partner choice and intersexual competition. In J. Barkow, L. Cosmides, and J. Tooby, eds., ***The Adapted Mind.*** New York: Oxford, pp. 249-266.

Buss, D. M., Larsen, R. J., Westen, D., and Semmelroth, J. (1992). Sex differences in jealousy: Evolution, physiology, and psychology. ***Psychological Science,*** **3, 251-253.**

Cannon, W. B. (1929). Bodily Changes ***in Pain, Hunger, Fear, and Rage. Researches into the Function*** **of** ***Emo***tional Excitement. New York: Harper and Row.

Chisholm, J. S. (1996). The evolutionary ecology of human attachment organization. ***Human Nature*** **7(l),** l-**38.**

Condit, V. K. (1990). Anorexia nervosa: Levels of causation. ***Human Nature*** **l(4),** 391414.

Cosmides, L., and Tooby, J. (1989). Evolutionary psychology and the generation of culture, part II: Case study: A computational theory of social exchange. ***Ethology and Sociobiol***-**ogylO(l-3),** 51-98.

Cross-National Collaborative Group. (1992). The changing rate of major depression. Cross-national comparisons. ***Journal of the American Medical Association,*** **268(21),** 3098-105.

Crow T.J. (1993). Sexual selection, Machiavellian intelligence, and the origins of psychosis. ***Lancet*** **342(8871),594-598.**

Daly, M., and Wilson, M. I. (1981). Abuse and neglect of children in evolutionary perspective. In R. D. Alexander and D. W. Tinkle, eds., ***Natural Selection and Social Behavior.*** New York: Chiron Press, pp. **405-416.**

Daly, M., and Wilson, M. (1983). Sex, ***Evolution, and Behavior,*** 2nd ed. Boston: Willard Grant Press.

Daly, M., and Wilson, M. (1989). ***Homicide. New*** York: Aldine.

Daly, M., and Wilson, M. (1992). Is parent-offspring conflict sex-linked? Freudian and Darwinian models. *Journal of Personality 58(1), 163-189.*

Daly, M., Wilson, M. I., and Weghorst, S. J. (1982). Male sexual jealousy. *Ethology and Sociobiology 3, 11-27.*

Dawkins, R. (1976). *The Selfish Gene.* Oxford: Oxford University Press.

Eaton, S. B., Konner, M., and Shostak, M. (1988). Stone agers in the fast lane: chronic degenerative diseases in evolutionary perspective. *The American Journal of Medicine 84(4), 739-749.*

Eibl-Eibesfeldt, I. *(1983). Human Ethology.* New York: Aldine de Gruyter.

Ekman, P. (1992). An argument for basic emotions. *Cognition and Emotion 6(3/4), 169-200.*

Engel, G., and Schmale, A. (1972). Conservation-withdrawal: A primary regulatory process for organismic homeostasis. In R. Porter and J. Night, eds., *Physiology, Emotion, and Psychosomatic Illness,* vol. 8 (pp. 57-85). Amsterdam: CIBA.

Essock-Vitale, S. M., and Fairbanks, L. A. (1979). Sociobiological theories of kin selection and reciprocal altruism and their relevance for psychiatry. *Journal of Nervous and Mental Diseases 167(l), 23-28.*

Ewald, P. *(1993). Evolution of Infectious Disease. New* York: Oxford University Press.

Feierman, J. R. (1994). A testable hypothesis about schizophrenia generated by evolutionary theory. *Ethology and Sociobiology 15, 263-282.*

Finlay-Jones, R., and Brown, G. (1981). Types of stressful life event and the onset of anxiety and depressive disorders. *Psychological Medicine 10, 445-454.*

Fisher, H. E. *(1992). Anatomy of Love.* New York: W.W. Norton.

Frank, R. H. (1988). *Passions Within Reason: The Strategic Role of the Emotions.* New York: W.W. Norton.

Frijda, *N.* H. *(1986). The Emotions.* Cambridge: Cambridge University Press.

Garcia, J., and Ervin, F. (1968). Gustatory-visceral and telereceptor-cutaneous conditioning: Adaptation in internal and external milieus. *Communications in Behavioral Biology A 1,389-414.*

Gardner, R. Jr. (1982). Mechanisms in manic-depressive disorder. *Archives of General Psychiatry 39, 1436-1441.*

Gardner R. Jr. (1995). Sociobiology and its applications to psychiatry. In H. I. Kaplan and B. J. Saddock, eds., *Comprehensive Textbook of Psychiatry/VI* (pp. 365-376). Baltimore, MD: Williams and Wilkins,

Gilbert, P. *(1992). Depression: The Evolution of Powerlessness. New* York: Guilford.

Gilbert, P., Price, J., and Allen, S. (1995). Social comparison, social attractiveness and evolution: How might they be related? New *Ideas in Psychology 13(2), 149-165.*

Glantz, K., and Pearce, J. *(1989). Exiles from Eden. New* York: W.W. Norton.

Gottesman, I., and Shields, J. (1982). *Schizophrenia: The Epigenetic Puzzle.* New York: Cambridge University Press.

Gut, E. *(1989). Productive and Unproductive Depression. New* York: Basic Books.

Hamilton, W. D. (1964). The evolution of social behavior I and II. *Journal of Theoretical Biology 7, l-52.*

Harlow, H. F. (1974). *Learning to Love.* New York: J. Aronson.

Harpending, H. C., and Sobus, J. (1987). Sociopathy as an adaptation, *Ethology and Sociobiology 8(3S),63s-72s.*

Hrdy, *S.* B. (1981). *The Woman That Never Evolved.* Cambridge: Harvard University Press.

Humphrey, N. K. (1976). The social function of intellect. In P. Bateson and R. Hinde, eds., *Growing Points in* Ethology (pp. 303–318). London: Cambridge University Press.

Jarvik, L. F., and Chadwick, S. B. (1972). Schizophrenia and survival. In M. Hammer, K. Salzinger, and S. Sutton, eds., *Psychopathology. New* York: John Wiley, pp. 57-74.

Kagan, J., Resnick, R. S., and Snidman, N. (1987). Biological bases of childhood shyness. *Science* 240, 189–208.

Kendell, R. E. (1984). Reflections on psychiatric classification. *Integrative Psychiatry* 2, 43–47W.

Kendell, R. E., and Kemp, I. W. (1989). Maternal influenza in the etiology of schizophrenia. *Archives of General Psychiatry* 46, 878-882.

Kerber, K. (1994). The marital balance of power and the quid pro quo: An evolutionary perspective. *Ethology and Sociobiology* 15(5–6), 283-298.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshelman, S., Willchen, H. U., and Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the national comorbidity survey. *Archives of General Psychiatry 5 1, 8-19.*

Kluger, M. J., ed. (1979). Fever, *Its Biology, Evolution, and Function.* Princeton, NJ: Princeton University Press.

Konner, M. (1983). *The Tangled Wing: Biological Constraints on the Human Spirit.* New York: Harper Colophon Books.

Konner, M. (1990). *Why the Reckless Survive.* London: Penguin,

Konner, M. (1995). Anthropology and psychiatry. In H. I. Haplan and B. J. Saddock, eds., *Comprehensive Textbook of Psychiatry,* vol. VI (pp. 337-355). Baltimore, MD: Williams and Wilkins.

Krebs, J., and Davies, *N. (1991). Behavioral Ecology: An Evolutionary Approach,* 3rd ed. Oxford: Blackwell.

Krebs, J. R., and Dawkins, R. D. (1984). Animal signals: mind-reading and manipulation. In J. R. Krebs and N. B. Davies, eds., *Behavioral Ecology: An Evolutionary Approach* (pp. 380-402). Sunderland, MA: Sinauer Associates.

Kurth, C., Krahn, D., Nairn, K., and Drewnowski, A. (1995). The severity of dieting and bingeing behaviors in college women: Interview validation of survey data. *Journal of Psychiatric Research* 29(3), *211-225.*

Lancaster, J. D., Altmann, J., Rossi, S. A., and Sherrod, L. R., eds. (1987). *Parenting Across the Life Span. New* York: Aldine de Gruyter.

Lockard, J., and Paulhus, D. L., eds. *(1988). Self-deception: An Adaptive Mechanism?* Engelwood Cliffs, NJ: Prentice Hall.

Marks, I. M. *(1987). Fears, Phobias, and Rituals.* New York: Oxford University Press.

Marks, I. M. (1988). Blood-injury phobia: A review. *American Journal of Psychiatry* 145(10), *1207-1213.*

Marks, I. M., and Nesse, R. M. (1994). Fear and fitness: An evolutionary analysis of anxiety disorders. *Ethology and Sociobiology* 15(5–6), *247-261.*

Marks, I. M., and Tobena, A. (1990). Learning and unlearning fear: A clinical and evolutionary perspective. *Neuroscience and Biobehavioral Reviews 14, 365-384.*

McGuire, M. T. and Fairbanks, L.A. (eds.) *(1977). Ethological Psychiatry: Psychopathology in the Context of Evolutionary Biology. New* York: Grune and Stratton.

McGuire, M., Marks, I., Nesse, R., and Troisi, A. (1992). Evolutionary biology: A basic science for psychiatry.

Acta Psychiatrica Scandinavica **86, 89-96.**

McGuire, M. T., and Troisi, A. (1998). ***Darwinian Psychiatry. New*** York: Oxford University Press.

Mealey, L. **(1995).** The sociobiology of sociopathy: an integrated evolutionary model. ***Behavioral and Brain Sciences.*** 18:523–541.

Melzack, R. **(1973).** ***The Puzzle of Pain.*** New York: Basic Books.

Miller, G.F. (In press). ***Evolution of the*** **Human Brain** ***through*** Runaway Sex***ual Selection.*** **Boston: MIT Press/** Bradford Books.

Mineka, S., Keir, R., and Price, V. **(1980).** Fear of snakes in wild- and laboratory-reared rhesus monkeys (*Macaca mulatta*). ***Animal Learning*** and Behavior 8(4), **653-663.**

Morris, W. N. **(1992).** A functional analysis of the role of mood in affective systems. In M. S. Clark, ed., ***Emotion.*** Newbury Park, CA: Sage.

Nesse, R. M. **(1984).** An evolutionary perspective on psychiatry. ***Comparative Psychiatry,*** 25(6), **575-580.**

Nesse, R. M. **(1990).** Evolutionary explanations of emotions. ***Human Nature,*** 1(3), **261-289.**

Nesse, R. M. (1991a). Psychiatry and sociobiology. In M. Maxwell, ed., ***The Sociobiological Imagination.*** Albany: SUNY Press, pp. **23-40.**

Nesse, R. M. (1991b). What is mood for? *Psycholoquy* 2(9.2).

Nesse, R. M. **(1994).** An evolutionary perspective on substance abuse. ***Ethology and Sociobiology*** 15(5–6), **339-348.**

Nesse, R. M., and Lloyd, A. T. **(1992).** The evolution of psychodynamic mechanisms. In J. Barkow, L. Cosmides, and J. Tooby, eds., The Adapted ***Mind: Evolu tionary Psychology and the Generation of Culture. New*** York: Oxford University Press, pp. **601-624.**

Nesse, R., and Williams, G. (1994). ***Why*** We ***Get Sick: The New Science of Darwinian Medicine. New York:*** Times Books.

Nesse, R.M., and Berridge, K.C. (1997). Psychoactive drug use in evolutionary perspective. ***Science,*** **278, 63-66.**

Nisbett, R., and Ross, L. **(1980).** ***Human Inference: Strategies and Shortcomings of Social Judgment.*** Englewood Cliffs, NJ: Prentice-Hall.

Öhman, A., and Dimberg, U. **(1984).** An evolutionary perspective on human social behavior. In W. M. Waid, ed., *Sociophysiology. New* York: Springer, pp. **47-86.**

Plutchik, R. **(1980).** ***Emotion: A Psychoevolutionary Synthesis. New*** York: Harper and Row.

Plutchik, R., and Kellerman, H., eds. **(1989).** ***Emotion: Theory, Research, and Experience. New*** York: Academic Press.

Pomerleau, C. **(1997).** Cofactors for smoking and evolutionary psychobiology. ***Addiction*** **92, 397-408.**

Price, J. **(1967).** The dominance hierarchy and the evolution of mental illness. *Lancet 2,* **243-248.**

Price, J. S., and Sloman, L. **(1987).** Depression as yielding behavior: An animal model based on Schjelderup-Ebbe's pecking order. ***Ethology and Sociobiology*** **8, 85s–98s.**

Raleigh, M., McGuire, M., Brammer, G., Pollack, D., and Yuwiler, A. (1991). Serotonergic mechanisms promote dominance acquisition in adult male vervet monkeys. ***Brain Research*** 559(2), 181–190.

Raleigh, M. J., McGuire, M. T., Brammer, G. L., and Yuwiler, A. **(1983).** Social and environmental influences on blood serotonin concentrations in monkeys. Archives of General Psy*chiatry* 41, 405–410.

Rancour-Laferriere, D. **(1985).** ***Signs of the Flesh.*** New York: Mouton de Gruyter.

Ridley, M. (1993). The *Red Queen.* New York: Macmillan.

Seligman, M. (1970). On the generality of the laws of learning. Psychological *Reviews* 77, 406–418.

Slater, E., Hare, E. H., and Price, J. S. (1971). Marriage and fertility of psychiatric patients compared to national data. *Social Biology 18* (suppl.), s60–s73.

Slavin, M. *(1992). The Adaptive Design of the Human Psyche : Psychoanalysis, Evolutionary Biology, and the Therapeutic Process. New* York: Guilford Press.

Slavin, M. 0. K., Daniel. (1990). Toward a new paradigm for psychoanalysis: An evolutionary biological perspective on the classical-relational dialectic. *Psychoanalytic Psychology, 7* (suppl.), 5-31.

Slavin, M., and Kriegman, D. (1992). *The Adaptive Design of the Human Psyche: Psychoanalysis, Evolutionary Biology, and the Therapeutic Process.* New York: Guilford Press.

Smith, E. A., and Winterhalder, B., eds. *(1992). Evolutionary Ecology and Human Behavior. New* York: Aldine de Gruyter.

Smuts, B. (1995). The evolutionary origins of patriarchy. *Human Nature* 6(1), 1-32.

Staddon, J. E. R. (1983). *Adaptive Behavior and Learning.* Cambridge: Cambridge University Press.

Stevens, A. (1982). *Archetype: A Natural History of the Self.* London: Routledge.

Stevens, A., and Price, J. (1996). *Evolutionary Psychiatry: A New Beginning.* London: Routledge.

Surbey, M. K. (1987). Anorexia nervosa, amenorrhea, and adaptation. *Ethology and Sociobiology* 8(3), 47-61.

Symons, D. *(1979). The Evolution of Human Sexuality.* New York: Oxford University Press.

Tooby, J., and Cosmides, L. (1990a). On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation. *Journal of Personality 58, 17-67.*

Tooby, J., and Cosmides, L. (1990b). The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology,* 11(4/5), *375424.*

Trivers, R. L. (1971). The evolution of reciprocal altruism. *Quarterly Review of Biology46, 35-57.*

Trivers, R. L. (1974). Parent-offspring conflict. *American Zoologist* 14, 249–264.

Trivers, R. L. (1976). Foreword to *The Selfish Gene* by R. Dawkins. New York: Oxford University Press, pp. v–vii.

Trivers, R. (1985). *Social Evolution.* Menlo Park, CA: Benjamin/Cummings.

Tversky, A., and Kahneman, D. (1974). Heuristics and biases. *Science 185,* 1124-1131.

Voland, E., and Voland, R. (1989). Evolutionary biology and psychiatry: The case of anorexia nervosa. *Ethology and Sociobiology 10, 223-*240.

Weinberg, E. D. (1984). Iron withholding: a defense against infection and neoplasia. *Physiology Reviews* 64, 65–102.

Weissman, M. M., Bland, R. C., and Canino, G. J. (1996). Cross-national epidemiology of major depression and bipolar disorder. *Journal of the American Medical Association, 276, 293-296.*

Wenegrat, B. (1984). *Sociobiology and Mental Disorder: A New View.* Menlo Park, CA: Addison-Wesley.

Wenegrat, B. *(1990). Sociobiological Psychiatry: Normal Behavior and Psychopathology.* Lexington, MA: Lexington.

Williams, *G. C. (1966). Adaptation and Natural Selection: A Critique of Some Current Evolutionary Thought.*

Princeton, NJ: Princeton University Press.

Williams, G. W., and Nesse, R. M. (1991). The dawn of Darwinian medicine. *Quarterly Review of Biology* 66(1), 1-22.

Wilson, D. R. (1992). Evolutionary epidemiology. *Acta* Biotheretica 40:187–189.

Wright, R. (1994). ***The Moral Animal.*** New York: Vintage.