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Toward an Evolutionary Taxonomy of Treatable Conditions

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The definition of disorder as a harmful dysfunction (J. C. Wakefield, 1999) is a useful concept, anchored in the recognition that the evolved human architecture consists of a collection of functional mechanisms that may potentially be impaired and whose impairment may be harmful. Because natural selection organized each mechanism to solve a distinct adaptive problem under ancestral conditions, the criteria for whether a mechanism is dysfunctional are supplied by whether the mechanism has become impaired in performing its *ancestral* function. Because evolutionary function and dysfunction diverge markedly from normal human standards of value, many dysfunctions are beneficial, whereas various mechanisms that are performing their evolved function may cause disturbing outcomes. For this reason, many conditions in addition to disorders may require treatment, and the authors attempt to sketch an evolutionary taxonomy of treatable conditions.

Abnormal psychology has two distinct but related identities: (a) as an essentially medical discipline concerned with the study, diagnosis, and treatment of psychological conditions that may invite treatment, and (b) as the scientific study of psychological phenomena that fall outside the range of normal mental functioning, including but not limited to those that ensue when the psychological or neural architecture is damaged in some respect. At present both parent disciplines, psychology and medicine, are being gradually transformed through their emerging integration with modern evolutionary biology (Nesse & Williams, 1994; Tooby & Cosmides, 1992), and it seems likely that abnormal psychology will eventually follow the same course (see, e.g., Baron-Cohen, 1995, 1997).

Although the transformation is already well underway in many subfields of psychology, medicine is beginning to catch up, by using cutting-edge theories from evolutionary biology and evolutionary psychology to understand diseases, disorders, syndromes, and, more generally, conditions that cause pain and discomfort to those that have them (Ewald, 1993; Nesse & Williams, 1994; Toft, Aeschlimann, & Bolis, 1991; Williams & Nesse, 1991). This approach, called Darwinian medicine, is changing not only how conditions are conceptualized, investigated, and classified, but also how they are treated. For example, in the past, iron supplements were routinely given to those diagnosed with anemia. But new evidence indicates that many types of infectious bacteria are rate-limited by their access to bioavailable iron, and that as a result, humans and other animals have complex systems designed to sequester or withhold iron when the body is infected with strains of bacteria that thrive on it. That is, iron-withholding (manifesting itself as one form of anemia) appears to be an evolved defense against infection, not a dysfunction, and well-meaning physicians, believing that deviations from normal levels of iron were the problem, have inadvertently exacerbated infections by prescribing iron supplements (Weinberg, 1984).

Similarly, fever, once thought of as a disorder of temperature regulation, is now recognized as another of the body's evolved defenses against infection (Kluger, 1997). Knowing these facts and distinctions expands one's capacity for informed choice: Depending on your plans and values, you can choose either to relieve the fever and prolong the illness by a few days or to endure it and recover faster. More significantly, evolutionarily sophisticated physicians can avoid damaging their patients by learning to distinguish an evolved defense from a dysfunction, for example by distinguishing anemia caused by insufficient iron in the diet from anemia as a defense against iron-limited infections. Through the understanding of function, they can distinguish functional variation from dysfunctional variation as well as the functional response (e.g., pain) to an assault from the assault itself.

Moreover, understanding function also involves the unwelcome yet necessary recognition that function is relative to a specific agent, and so may involve conflicts of interest and forced choices among the rival values of different agents when such values are mutually inconsistent. Of course, although the infectious agent's health is the host's illness, few health practitioners are troubled by, or need be troubled by, a concern for the welfare of disease organisms. However, other adaptations—such as those involved in pregnancy, parenting, mating, or in regulating other forms of social interaction—do entangle multiple humans, and so in such cases the issue of rival value systems can acquire urgency. For example, mild cases of gestational diabetes, rather that being a dysfunction, may reflect the fact that fetuses can increase their growth rate by secreting hormones that bring more glucose-laden blood to the placenta; severe cases may reflect conflicting adaptations in the

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mother and fetus pursuing inconsistent functional agendas over allocation of maternal resources. In this case, the present fetus's adaptations are designed to "value" the present fetus over future fetuses more than are the mother's adaptations (Haig, 1993), leading to maternal–fetal conflict. Of course, it is critical to distinguish the weightings or "valuations" built into such adaptations from the valuations present in the minds of the participants. The mother's mind (treating it, for the moment, as a single unit) may value the fetus more (or less) than do her physiologically based reproductive adaptations.

The call for a more evolutionarily well-informed medical community is based on the striking advantages conferred by understanding the functional organization of human design, which is vital for recognizing, illuminating, and treating dysfunctions and other harmful conditions, as well as for recognizing and promoting health. However, although it is clear that an evolutionary perspective can shed light on "physical" disorders, can it shed light on the often questioned construct of "mental" disorder? We believe that it has equal applicability, although the shift from the "physical" to the "mental" raises some important issues, including several that are usually neglected in the case of physical disorders.

We use the terms *mental* and *mind* in the standard cognitive sense. According to this view, the mind is an information-processing description of that subset of the physical activity of the brain that implements organized computational activity (Jackendoff, 1987; see Cosmides & Tooby, 1987, for an evolutionary extension). The reason an information-processing view is particularly appropriate and useful for psychological science (as opposed to cardiology or osteology) is that the brain is the organ that specifically evolved to carry out the function of information-processing or computation.

Evolutionary psychology is the research program that attempts to supplement and integrate existing psychological and neuroscience techniques and approaches with the new knowledge provided by modern evolutionary biology and biological anthropology (see, e.g., Cosmides & Tooby, 1987; Tooby & Cosmides, 1992). One of the central insights now available to psychologists, psychiatrists, and neuroscientists from this integration is that the human psychological architecture consists of a constellation of adaptations or devices, each of which evolved among our foraging ancestors to perform specific computational functions necessary or useful in such ancestral conditions. The problem-solving organization of each device (or mechanism, module, mental organ, circuit, program, design feature, etc.) can be directly related to the functional requirements and demand characteristics of the particular adaptive problem that selected for the creation or elaboration of that device among our ancestors.

Thus, the cost-effective avoidance of bites from venomous snakes is the function of the computational circuits underlying snake phobias (Marks, 1987); disinvestment of effort and hedonic attachment to unprofitable activities or relationships is the hypothesized function of the computational systems underlying depression (Nesse & Williams, 1994; Tooby & Cosmides, 1990b); a countervailing distaste for sexual contact with close genetic relatives who are otherwise available and attractive potential sex partners is the function of the Westermarck incest-avoidance mechanism (Wolf & Huang, 1980); and the ability to make computations about the interactions of physical objects is the function of a specific module sometimes called *ToBy* (i.e., for "theory of bodies"; Leslie, 1994; Spelke, 1990).

Dysfunctions, Disorders, and Treatable Conditions

Obviously, if brains are sets of functional devices, these constituent devices can either fail to develop or lose their functional organization. Because selection was the source of the functional organization present in the design of each mechanism, evolutionary analyses provide the defining "objective" standards for functional performance for psychological mechanisms: Is the mechanism operating as it was designed to (Tooby & Cosmides, 1992, in press)? Hence, to begin with, an evolutionary perspective sheds light on the concept of a mental disorder by providing the criteria for assessing functional impairment (as opposed to mere variation) for each implicated mechanism. To integrate this with Wakefield's (1999) definition, a psychological disorder is a condition in which one or more psychological mechanisms are not computing according to the criteria that constitute their evolved function (perhaps because of a physical disorder) and in a way that is considered harmful.

Defining the concept of *disorder* in a way that links it to the underlying scientific reality is more than quibbling over semantics. Wakefield's (1999) definition is powerful because it recognizes that the brain as well as the body is composed of an interlocking set of functional units or evolved adaptations. Categorization schemes that recognize this allow individuals to see more clearly whether treating a condition will benefit them or be counterproductive, as the examples of fever and anemia show.

In fact, Wakefield (1999) does an admirable job of showing that the "harmful dysfunction" definition captures how people intuitively categorize in this domain far more effectively than does a Roschian approach (for recent research questioning the generality of Roschian concepts, see Bloom, 1996; Hirschfeld & Gelman, 1994; Keil, 1989; Markman, 1989). To us, however, the central point is to make progress toward a reflective consensus about how the medical profession ought to usefully define *disorder,* whether or not this is presently the way they think. Given this goal, a key problem with a Roschian analysis of disorder is that it lacks scientific power, as its application only leads to symptom clusters. In contrast, most biomedical scientists strive for a causal account of diseases and disorders; they classify a disorder by symptom clusters only when they lack knowledge of its etiology and cause, as an interim measure. This is, in part, because the best hope for finding new and more effective ways of relieving human suffering is by understanding the causes and nature of the condition generating the distress. As we discuss, the condition may be an evolved defense, normal variation within a universal design, a properly functioning adaptive system in an evolutionarily novel environment, an accommodation to the needs of a different organism (such as a fetus), a dysfunction in an adaptation, or many other things. It matters which: Both research agendas and treatment decisions may differ depending on the answer.

For these reasons, in our view, Wakefield's (1999) analysis of disorder is illuminating and scientifically valuable. It is rare—even in the biological sciences—to find a scholar with Wakefield's nuanced understanding of adaptation and natural selection. We do not comment on the challenges to his conceptual analysis because Wakefield's own responses are, in our view, on target.

Instead, what we discuss is a larger category of interest to health professionals: treatable conditions. As Wakefield (1999) himself points out (pp. 374, 391), disorders (harmful dysfunctions) are not the only conditions that individuals, physicians, and mental health professionals consider worthy of treatment. Indeed, many conditions that are not evolutionary dysfunctions are judged to be quite harmful to the people that exhibit them, just as many conditions that are not harmful or are even desirable are evolutionary dysfunctions.

We suspect that some of the reluctance to accept Wakefield's (1999) proposed definition of disorder stems from an implicit belief that the concept of disorder must serve as the sole rationale underlying treatment, and the resultant wish for a simple one-to-one mapping between dysfunction, disorder, and treatable condition. When a full taxonomy of treatable conditions is developed, and the concept of disorder is contextualized within it, this source of resistance should disappear. In fact, a full application of modern evolutionary biology and evolutionary psychology suggests there may be many types of conditions that may be viewed as worthy of treatment that fall outside of both the harmful dysfunction categorization scheme and the other taxonomic groups to which Wakefield alludes.

Evolved Reasoning Specializations Shape Scientific and Medical Intuitions

Wakefield (1999) makes a compelling case that disorder is a classical concept implicitly meaning "harmful dysfunction" to most members of the language community who use the term. Indeed, Wakefield's analysis does capture the intuitions of medical professionals to a remarkable degree. Why this should be so could be regarded as something of a puzzle. After all, not only is evolutionary biology not a standard feature of medical curricula, but it is often almost unknown. In consequence, the intuitions of most physicians and other health professionals were not educated by knowledge of the causal processes that designed the systems they repair. So why does a scientific definition of disorder, grounded in modern theories of how natural selection produces functional organization in phenotypes, classify conditions as disorders in a way that so closely resembles the folk theories of health professionals?

We suggest that the scientific concepts of function and design drawn from evolutionary biology and applied to living systems are similar in many respects to analogous intuitive concepts that lay people routinely apply to human-made inanimate artifacts. These intuitive concepts, as well as many others, appear to be embedded in evolved reasoning specializations that reliably develop in all normal humans in all cultures (Hirschfeld & Gelman, 1994; Tooby & Cosmides, 1992), in Paris no less than at our Amazonian field site. These concepts come to mind spontaneously in certain eliciting contexts, making certain ideas and inferences seem obvious, natural, and transparent to the individuals involved. Recent work in cognitive development and cognitive neuroscience suggests that the human cognitive architecture is permeated with such evolved, content-specific inference engines (Hirshfeld & Gelman, 1994; Sperber, 1994; Tooby & Cosmides, 1992). Many of these appear to resemble expert systems developed in artificial intelligence, and, like expert systems, they often appear to come equipped with "innate concepts" and "innate" operations: inference procedures and assumptions that embody knowledge specific to the given problem domain they evolved to reason about.

For example, there is a growing body of evidence for the existence of functionally (and, in some cases, neurally) dissociable inference systems that are specialized for reasoning about objects, physical causality, artifacts, number, the biological world, the beliefs and motivations of other individuals, cheating, precautions, and threats (Baron-Cohen, 1995; Cosmides, 1989; Cosmides & Tooby, 1992, 1997; Hirschfeld & Gelman, 1994; Leslie, 1987; Stone, Cosmides, & Tooby, 1996). Specifically relevant to the issue of how people spontaneously think about disorders, it appears as if humans have a number of evolved inference engines that interact to shape how we reason about mechanical causality, artifacts, teleology, and function. These include a module for reasoning about rigid object mechanics (Leslie, 1994; Spelke, 1990), sometimes called ToBy. This provides conceptual primitives about nonteleological mechanical forward causality—a set of evolved concepts that form the basis for much of scientific thinking. Another inference engine represents events in teleological terms: How are movements, objects, or events organized to bring about a goal state? Humans also appear to have an inference engine for reasoning about artifacts, which supports linking inferences between the physical structure of a tool (ToBy), and the goal state or function it is intended or designed to serve (teleology, and/or ToMM, the theory of mind module; Brown, 1990). Although these recent challenges to the tabula rasa view strike many as exotic and implausible, they are supported by neuropsychological evidence demonstrating selective impairments in each of these competences (Caramazza & Shelton, 1998; Farah, Meyer, & McMullen, 1996; Warrington & McCarthy, 1983; Warrington & Shallice, 1984). Even 12-month-olds ignore surface features in favor of functional ones in reasoning about tool use (Brown, 1990).

If these mental programs are part of the common mental equipment of our species, then one would expect to detect traces of their operation in the history of science and in the organization of medical practices. Conceptual frameworks would not be driven simply by observations, but by their mesh with elaborations of evolved conceptual primitives and their evocations in commonly experienced contexts. For example, the views of Democritus and other early atomists such as the Stoics are plausibly based less on "observations" of atoms or their effects than on the appeal of applying to as many phenomena as possible the mental operations drawn from the module that reasons about rigid object mechanics. In the rigid object mechanics module, there is no action at a distance (despite the reality of such forces in nature), causation moves forward in time (leaving little room for teleology), and events are explained by physical contact and the shapes of objects (rather than by the mental, spiritual, vitalistic, or intentional). The rise of Renaissance experimental science was strongly informed by these mechanistic primitives, persuading many that even Newton's law of universal gravitation was unscientific and occult, because it proposed action at a distance. If humans have a large menu of alternative inference engines that can be deployed at any time, then the same situation may be interpreted very differently depending on which engine or set of engines is activated and applied to construct an interpretation: Is the earth an aggregation of chemical processes or a mother? Are the heavens a clock or an abode of animate entities? Different scientific and cultural movements may

be driven by the differential evocation of distinct inference engines.

Medical Intuitions and Artifact Reasoning: Bodies as Collections of Functional Mechanisms

> Now, as each of the parts of the body, like every other instrument, is for the sake of some purpose.
>
> —Aristotle

The history of medicine and its rival schools seems to have been shaped by the rival intuitions rooted in alternative inference engines. Although medicine began with a vitalistic framework (that continues today, e.g., in holistic medicine and psychiatry), the alternative tradition of conceptualizing the body as a machine built of component machines became more prominent once Harvey demonstrated that the circulatory system could be conceptualized as a system of pipes governed by valves. Several centuries of subsequent progress in anatomy and physiology rested on this functionalist and mechanistic framework (Mayr, 1983) before Darwin and Wallace emerged to give it a scientific justification. An artifact, or a machine, is a physical system whose physical arrangements cause valuable outcomes by design (usually meaning by the intent of the creator of the machine). Obviously, if one applies the concepts and inferences appropriate to artifacts to the body, then it becomes natural to think of organs as machines designed to achieve functions and, as a corollary, natural to think of this functional organization as susceptible to breakdown—that, like other machines, organs can become *dis*ordered, thereby becoming *dys*functional, and fail to achieve their designed outcomes.

This parallelism between biological systems and human-made machines is a powerful one, having some basis in reality. Selection does indeed build functional organization into organisms, creating structures that are analogous to artifacts in that they are physically organized to cause specific and useful outcomes. Alternative designs are "chosen" (i.e., spread) on the basis of how well they function to cause propagative outcomes, so a design feature that solves an adaptive problem can be outcompeted by a new design feature that solves it better. This process has produced exquisitely engineered biological machines—the vertebrate eye, the immune system, photosynthetic pigments, echolocation—whose performance is unrivaled by any machine yet designed by humans. Insofar as physicians interpret bodies to be collections of functional systems, and natural selection builds functional systems, the intuitions of physicians will parallel reality and ground Wakefield's (1999) harmful dysfunction analysis.

Where the analogy to human-made artifacts holds, the harmful dysfunction analysis of disorder captures much of what is needed to reason about physical and psychological disorders. Where the analogy breaks down, other concepts are needed. What are some of the ways in which the analogy breaks down? Organisms differ from human-made machines in a number of ways: For example, (a) an organism assembles itself; (b) a single organism usually morphs through several different designs during its life (e.g., fetus, infant, child, adolescent, and adult in humans); (c) the biological definition of function is grounded in the logic of replication, not in human values of convenience, suffering, or individual or mutual well-being, so a condition may be evolutionarily dysfunctional but welcome and valuable, or functional but catastrophic; (d) a single organism does not have a unitary "purpose"—the fitness of different complements of genes is promoted by mutually incompatible adaptations (resulting in intragenomic conflict, see below); (e) many organisms behave (whereas few machines do), and they are designed to achieve goals that are sometimes in conflict with those of other organisms; (f) an organism is designed to tailor itself to local conditions, a betting process that is always incompletely successful, and hence open to help. At points where the parallelism between artifacts and evolved adaptations breaks down, an array of medically interesting conditions can be identified that are not disorders but that might be viewed by the people who have them (and others) as worthy of treatment or intervention.

Treatable Conditions, Health, Disorders, and the Medicalization of Values

Before moving ahead, however, it is necessary to dissect the role that values play in the concept of *treatable condition* and the relationship (or lack thereof) between legitimizing systems of values and an evolutionary perspective. To be a treatable condition, there must be (a) a characterizable condition in a person; (b) a person or social decision-making unit whose values and decisions will govern the actions taken with respect to the condition; (c) a valuation by that person or unit that the condition is negative and that it ought to be changed (that is, that the persistence of the condition is "harmful," "undesirable," or "unhealthy"); and (d) knowledge of a method for changing the condition in the desired direction. What counts as undesirable, and who gets to choose? Often the person being treated gets to act on his or her own values, but sometimes others may be empowered to do so, either benignly as is often the case with parents of young children, or less so, as with Soviet psychiatrists drugging political dissidents. As Wakefield (1999) correctly emphasizes about disorder, the concept of treatable condition clearly involves the intersection of scientific issues (e.g., characterizations of the condition and its possible treatment) with the independent, contentious, and nonobjective world of values.

Indeed, it is a commonplace of evolutionary psychology and biology that selection has shaped the design of motivational systems so that each individual will be at the center of a unique webwork of valuations that is unlikely to duplicate the valuations of anyone else (Hamilton, 1964; Trivers, 1974; Williams, 1966). During evolution, courses of action that benefited the gene sets situated in one individual often came at the expense of the gene sets situated in other individuals, or in other parts of the same individual's genome. Because of this, selection retained and made species-typical genes that built motivational systems that reflected such differences in fitness "interests" in their computed choices. As a result, what is judged harmful or beneficial undergoes frame shifts as the individual (or decision-making unit) making the judgment shifts. More colloquially, conflicts of interest between individuals are endemic to the human condition, and so issues of what is desirable or harmful are rarely matters of harmonious consensus or intersubjective agreement, much less matters of fact. Fierce conflicts over these matters permeate human life.

Moreover, for better or worse, no one has yet solved Hume's question of how to derive an *ought* from an *is*, and so, in our view, it is important to be vigilant in keeping questions of values clearly distinguished from questions of fact. We wish to emphasize that an

evolutionary standard of functionality is a scientific and not a moral concept and does not provide any privileged platform from which to establish the primacy of some values over others. Unfortunately, in various disguised forms (often associated, e.g., with the terms *health* and *natural*), it is often used in just this way (e.g., in debates about sexual orientation or sex roles). It is not our purpose here to propagandize for our particular values. We only wish to point out that in understanding issues of mental disorder, health, and treatable conditions, the choice to act on some set of values as opposed to others is always necessarily a part of the process; that this choice ought not to be disguised with the pretense that only questions of fact are at issue; that the questions of whose values are prevailing and what are the nature of those values ought always to be made explicit; that a scientific account of the underlying psychological or biological situation ought to be kept separate from and undeformed by such questions of value; and that there will be an endemic and motivated temptation to confuse exactly these issues in order to spuriously "win" moral disputes under the guise that they are factual disputes. Although these points are obvious and widely accepted, whenever new scientific ideas emerge there is always an attempt, during periods of conceptual fluidity, to co-opt them into various agendas. So, however far the naturalization or medicalization of morals has proceeded, we hope that the increasing spread of evolutionary psychology will retard it.

Most physical disorders, such as liver failure, tend to impact only one person directly, usually eliciting a single standard of valuation (the good of the individual with the dysfunction, as assessed by that individual). In contrast, behaviors often affect many people at once, creating the possibility of conflicting assessments of value by each affected person. Thus, for psychological or behavioral conditions, there may be no harmony of values among the individual generating the behavior, family members, law enforcement officials, friends, victims, therapists, and so on. Moreover, psychiatric disorders may involve the (real or claimed) impairment of motivational or reasoning systems that individuals use (or would normally use) to make choices about their treatment. This provides an attractive rationale to others for superseding otherwise legally protected individual autonomy and choice.

Because people care about and often object to (or wish to excuse) the conduct of others, the concept of health (the absence of disorder) together with its opposites (illness, sickness, pathology, disorder, etc.) and companion terms (cure, treatment) have become widely used as supposedly objective concepts that nevertheless serve covertly moralizing or exculpatory functions. However, as Wakefield (1999) outlines in the case of disorders, health (as the absence of disorder), is not a value-free scientific concept, but depends on the intersection between the scientific concept of dysfunction with the value-based issue about what constitutes harm. All organisms vary in indefinitely many respects from central tendencies, but only when "harm" is judged to be occurring is sickness or an absence of health attributed.

The Bizarre Nature of Biological Functionality: Dysfunctional and Harmful Are Not the Same Thing

When it comes to mind and behavior, as opposed to physical disorders, there is widespread disagreement about whether any given phenomenon (e.g., free climbing motivated by sensation-seeking; violent sexual jealousy; genital piercing; too much interest in arcane academic questions) is properly viewed as a choice, a disorder, a normal reaction to environmental circumstances, a cultural difference, idiotypic variation within a normal range, and so on. Nevertheless, by embedding the analysis of mental dysfunctions within an evolutionary framework, the distinction between values and choices on the one hand and genuine evolutionary dysfunction on the other becomes more straightforward. Although an evolutionary perspective cannot provide any objective basis for validating some values over others, it does provide an objective basis for analyzing function and dysfunction.

Thus, the question, Is the mechanism in question operating in a way that produces the functional output it was designed to (i.e., was selected to)? is an answerable scientific question. Is the visual system recognizing objects? Are the incest avoidance mechanisms making the prospect of sex with family members distasteful? Can the person recognize that they have been cheated? To answer questions of functional integrity or impairment, one needs at a minimum to have correctly (a) individuated the mechanism from others, (b) identified and characterized its function (that is, the ancestral adaptive problem its design features were selected to solve), and (c) characterized its problem-solving design features and how they interact to produce the target set of functional outputs that would have increased fitness in ancestral environments (regardless of whether they spread their genetic bases in the modern world; Symons, 1992; Tooby & Cosmides, 1990b). Evolutionary psychologists and allied researchers have made a small but genuine measure of progress toward inventorying mechanisms and characterizing their evolved functions, although the field is in its infancy, and only a few out of the multitude of evolved mechanisms have been investigated.

It is critical to recognize, however, that biological standards of functionality are as distant from ordinary folk concepts of functionality as quantum mechanics is from ordinary physical concepts. Despite widespread belief to the contrary, selection does not necessarily favor, for example, the survival of the individual or the group, the maintenance of organs, the happiness of the individual, or the "welfare" of offspring, mates, or other members of the group (although these may sometimes or usually be pursued by individuals as proximate goal states). Indeed, cognitive or physiological adaptations exist that are designed to sacrifice these things under various conditions to serve other ends. Selection favored design features, ancestrally, that caused increases in the probability of the replication of the genes that underlie them (genic fitness), whatever the cost to other gene sets in the same individual or in other individuals (Cosmides & Tooby, 1981). This is an amoral process that does not map in any systematic fashion onto any known moral philosophy or sane set of human values (see Table 1).

For example, few people know or care about the replication of their mitochondrial genes, but mitochondrial genes in females in various species may contain elements that are designed to create adaptations to prematurely kill male offspring while still in the womb (Cosmides & Tooby, 1981). This promotes the fitness of the mitochondria (which are only passed on through daughters), but undermines the fitness of the nuclear genes in the woman, and may cause heartache to her and her husband. This is one instance of a common phenomenon, intragenomic conflict, in which different gene sets within the same individual have evolved adaptations that attempt to impose reproductive outcomes that benefit the gene set

Table 1
Value and Functionality Combinations for Treatable Conditions

How condition is valued	Mechanism is evolutionarily functional	Mechanism is evolutionarily dysfunctional
Person positively values condition (beneficial to self)	Sexual desire	Sensation-seeking, risk-taking
Person negatively values condition (harmful to self)	Appetite for sweets, fear, pain, mild depression	Obsessive–compulsive disorder, impotence, insomnia
Others positively value person's condition (beneficial to others)	Motivation to share in situations of extreme need	Being a scholar, adopting a child
Others negatively value person's condition (harmful to others)	Jealousy, lack of empathy	Schizophrenia
Person positively values condition, others negatively value condition (beneficial to self, harmful to others)	Intact retaliation motivation, competitiveness	Mania, paranoia, incest-seeking, sadism
Person negatively values condition, others positively value condition (harmful to self, beneficial to others)	Subordination to powerful	Gullibility
Some positively value condition, others negatively value condition	In-group favoritism	Use of psychotropic drugs

Note. Whether a condition is a dysfunction is independent of whether it is harmful to self or others as judged by the values of those involved. See text for full explanation.

at the expense of other gene sets in the organism (see Cosmides & Tooby, 1981, for derivation of theoretical principles and examples). Because of this, the dysfunctions that appear in some adaptations may be caused by the functional operation of other adaptations (making evolutionary function a mechanism- or gene-focal concept, rather than one that can be applied to individuals as a whole).

Equally strange to human value systems, genes for male sexual jealousy spread throughout the human species, becoming species-typical, because genes that built computational adaptations that motivated their bearers to discourage their mates from conceiving by other men spread at the expense of genes that motivated indifference to infidelity. Jealousy mechanisms often cause the males that bear them enormous suffering, and often motivate coercive, violent, or even deadly actions toward women (Buss 1994; Daly & Wilson, 1988)—actions or practices that are endorsed in some cultures, condemned in others. Yet jealousy is solely for the "benefit" or fitness-enhancement of the genes underlying the jealousy mechanism, not the individual who bears them, and its function is to cause patterned behaviors that spread those genes and retard the spread of competitive alleles. The evolutionarily correct functioning of those mechanisms is to deliver this output within the parameters that would have been adaptive ancestrally, which in the modern Western world might sometimes even destroy the lives of the man, his mate, and their children. Using intuitive notions of well-being as the standard, many therapists regard jealousy as a pathology (by which they mean it is a disvalued and potentially treatable condition), but to call this a disorder is to confuse the values of the patients involved (or psychiatrists) with the functional integrity of the cognitive adaptations that generate jealousy. If one was to equate "health" with an absence of evolutionary dysfunction, then it is possible than many abusive husbands and stalking ex-husbands are perfectly "healthy."

Reciprocally, accidents of development, culture, or genetic variation may generate individuals who are more empathic than would have been ancestrally functional, or more willing to care for adopted children, less interested in having children, more interested in pursuing scientific ideas, less jealous, more forgiving, less prone to violence, more tolerant of outgroup members, and so on, than would have been ancestrally adaptive. Few would want to treat people exhibiting these evolutionary dysfunctions in order to "cure" them and return them to "health." Similarly, the development of sexual orientation and gender identity appears to involve a series of modules or mechanisms whose function under ancestral conditions was to produce adults with all of the well-calibrated components of a heterosexual orientation and gender identity. Yet few homosexuals would choose to be "cured" of what is almost certainly an evolutionary dysfunction in their gender modularity systems. A large number of other dysfunctions (e.g., a decrement in the ability to forage) may cause no harm to self or others, and if they are not often useful or activated in the modern world, their loss may be scarcely noted or studied, or even be invisible.

This is not to say that evolutionary standards of function invert normal human values—merely that they do not map onto values in any systematic fashion. The functional or "natural" (as it actually is, as opposed to how it is imagined or idealized) is not equatable with the good, by most widely accepted value systems, and the dysfunctional or unnatural is not equatable with the harmful. The functional integrity of many mechanisms (e.g., coalitionalism, violent rage) may systematically cause harm, and so their dysfunction may be welcomed as beneficial. If by health one means only that there is an absence of evolutionary dysfunction, then health is not necessarily valuable, and it would be a bizarre medical or psychiatric system that aimed to return everyone to mental health as defined by evolutionary standards. Indeed, many cultural contrivances (such as legal systems) may have been developed to specifically disable or to minimize the expression of intact adaptations that cause harm to others. Thus, an absence of dysfunction in many normal mechanisms may qualify as a treatable condition if it is capable of being treated and the persons involved wish it to

be treated. Equally, the dysfunction of other mechanisms may be harmful, and so treating the dysfunction may be the choice made.

Factors Governing the Development of Functional Design

Traditional researchers often think of phylogenetic or evolutionary forces as exerting their influence solely through genes (nature) and, hence, tend to think of the environment as a force that works independently of or in opposition to evolutionary organization (nurture). According to this framework, these constitute independent and opposed explanations for phenomena. Even sophisticated researchers who realize that all traits are equally the product of an interaction between genes and environment often accept some aspects of this line of thinking. We have elsewhere argued that this is an ill-formed way of conceptualizing the relationship between development and evolved design (Tooby & Cosmides, 1990a, 1992). Organisms reliably develop their species-typical designs (lungs, eyes, skeleton, etc.) because of the joint and interlocking presence of two parallel inheritances: (a) the species-specific complement of genes and (b) a species-specific set of environmental regularities (which may include everything from gravity, maternal smiles, the uterus, and a certain distribution of shapes presented to the visual system, to the presence of a language community). Together, these interact to produce the species-typical design visible in normal members of a species.

Genes are important not because they proximately cause, independently of the environment, biological structure—this is an impossibility. Instead, genes are important because they are the variable set of control elements that natural selection changes or tunes over evolutionary time so that, in developmental time, the resulting interaction of the organism's genes and its environmental regularities causes the development of biologically functional structure. Selection, by choosing some genes over others, renders some parts of the environment relevant to development, and others irrelevant—so, selection, acting over the long run selects both parallel inheritances: the species-specific complement of genes, and the species-specific set of environmental regularities. So, although nothing is "genetically determined," nevertheless, an immensely intricate architecture full of evolved, species-typical functional machinery, designed by natural selection, reliably develops in all normal members of a species. (Throughout this article we may speak of species-typical design as a shorthand for species-typical design, plus all frequency-dependent equilibria, plus any recent local selection that has endured long enough to forge complex adaptations to local circumstances; Tooby & Cosmides, 1990a).

Thus, the first adaptive problem an organism faces is the task of assembling itself correctly (a problem not faced by human-made artifacts: It is the problem of the creator, not the artifact, to fashion the artifact correctly). To solve this adaptive problem, the organism requires a special class of adaptations: developmental adaptations. The function of developmental mechanisms is to (a) successfully construct the species-typical functional design and (b) calibrate the physical and especially the psychological architecture (which is far more adjustable) so that it is adaptively tailored to the local conditions it will face. Learning mechanisms are a subset of these calibrational developmental adaptations. Such adaptations are designed to resist or buffer the disordering effects of normal genetic and environmental variation (which are substantial; see, e.g., Tooby, 1982; Tooby & Cosmides, 1990a) through canalization or feedback-driven compensation to perturbations and assaults. Thus, it is useful to distinguish impairment in a realized implementational adaptation (e.g., something designed to carry out the operational business of an organism, like an eye) from impairment to a developmental adaptation, the role of which is to build implementational adaptations. Developmental adaptations have goal-states (what we have called *adaptive targets*) built into them, and they are designed to impose these on the organism. Therefore, damage to a developmental adaptation may be far more difficult to correct than damage to a implementational adaptation. Damaged adaptive targets, misspecifying key elements of brain chemistry, may be a major underlying factor in addiction, making it so difficult to treat.

The gene–environment inheritance system is designed to handle the range of genetic and environmental variation that was standard during the species' evolution, but not variation outside that range. If there are two inheritances, genetic and environmental, then "mutations" (unprecedented changes) in either or both may cause implementational adaptations to develop into nonfunctional forms. Virtually every individual carries many genetic defects, and, equally, each organism's developmental environment will have some environmental mutations that render it different from the long run composite set of statistical averages of environmental conditions that defines a species' environment of evolutionary adaptedness. For humans, of course, our modern world diverges enormously in thousands of key respects from the world of our foraging ancestors, and so modern humans can be expected to be riddled with scores of dysfunctions caused by these environmental differences. Of course, the essential question is the magnitude of the suffering the dysfunction causes and the possibilities (including economic) of treatment: Everyone notices schizophrenia or bipolar disorder because of their severity and abnormality, but most dysfunctions fade into the background level of human difficulties, at least until rising incomes motivate the provisioning of real or placebo treatments.

For these reasons, it is important to keep in mind that human design was engineered in ancestral environments. Appreciating the invariant (or statistically recurrent) features of ancestral environments is crucial to any evolutionary analysis. For any given species, an *adaptive problem* is defined as a problem (e.g., finding food, avoiding predators) that recurred over many generations in the environments in which that species evolved, and whose solution tended to promote the reproduction of the genes underlying the trait in those environments. Natural selection favors genes that achieve adaptive outcomes in the environments in which a lineage evolved. This series of environments is sometimes called the *environment of evolutionary adaptedness* (EEA). The EEA is not a specific place or time; rather, it is better conceptualized as the statistical composite of selection pressures and environmental properties that drove the alleles underlying the adaptation to equilibrium or fixation.

Toward an Evolutionary Taxonomy of Treatable Conditions

The general principle is that an individual may seek treatment (to change his or her condition) whenever there is a discrepancy between that individual's calibrationally individuated mechanism-

states (i.e., traits) and that individual's values or aspirations for himself or herself. Because naturally selected mechanisms are not necessarily designed to make us happy and because human standards of value do not correspond to evolutionary standards of function, an individual may suffer either from a harmful dysfunction, or from a harmful function. Hence, individuals may wish to repair or compensate for a harmful dysfunction, or to impair or counteract a harmful function. Moreover, the individual may seek to bring about a personally useful dysfunction of an adaptation (e.g., using antidepressants to make oneself happy with making reproductive sacrifices for career advancement, thereby disabling evolved motivational adaptations; or using steroids to improve athletic performance at the cost of sterility or deferred cardiac problems).

In addition to adaptations, organisms manifest indefinitely many by-products of adaptations as well as idiotypic variation caused by functionally neutral genetic variation or environmental mutations (noise). An appreciation for music is probably an evolutionary by-product (Pinker, 1997), yet people suffering from amusia (the neural loss of the ability to appreciate music) might seek treatment for their condition. Many aspects of appearance are not adaptations but are by-products, yet people obviously seek to restore their appearance after damage, or enhance their beauty even in the absence of damage. Furthermore, many of the traits that make individuals unique (e.g., distinctive tastes, interests, talents, appearances) and that cause those individuals or others to experience them as having unique and valued identities arise from genetic noise, environmental noise, or both (Tooby & Cosmides, 1996). For example, an unusual musical talent, being idiotypic, cannot be an evolved adaptation, yet its impairment might well cause someone to seek treatment. In general, then, individuals can be expected to seek to increase the valued effects of useful functions, dysfunctions, by-products, and idiotypic genetic or environmental variation and to decrease the disvalued effects of harmful functions, dysfunctions, by-products, and idiotypic variation. To the suffering person, it does not matter whether the condition is an adaptively designed outcome, damage to an adaptation, an unwanted side-effect, or simply an entropic accident (see Table 2).

Harmful Dysfunctions

Despite the many sources of value–condition divergence, harmful evolutionary dysfunctions remain the most important category. This is because most of human action depends on the realized functional organization of complex adaptations. These include the abilities to see and hear; to recognize objects, faces, voices, and emotional expressions; to connect individuals with identities and episodes; to understand speech; to walk, swallow, select foods, avoid dangers; to understand social conditionals; and so on. Sources of dysfunctions in adaptations include injury, infectious disease, breakdowns in other adaptations, deleterious genes and gene combinations, malnutrition, environmental assaults, environmental mutations, and novel gene–environment interactions, (see Nesse & Williams, 1994, for a discussion of many of these categories). Subjectively painful evolved defenses may often be mistaken for dysfunctions, but they are functions designed to guide the organism adaptively (e.g., normal depression; Nesse & Williams, 1994; Tooby & Cosmides, 1990b).

Development–Environment Mismatches

Environmental and developmental factors may be the most important sources of harmful dysfunctions and other value–

Table 2
Evolutionary Taxonomy of Treatable Conditions (or Value–Condition Divergences)

1. Disorders in implementational adaptations: design failures (harmful dysfunctions, caused by infection, injury, malnutrition, environmental assault, social assault, deleterious genes, etc.).
2. Disorders in developmental adaptations: developmental failures (caused by misspecification of adaptive targets, environmental mutation, non-species-standard gene set, infection, injury, malnutrition, environmental assault, social assault, rare gene–environment combinations, etc.).
3. Evolutionary average–individual case mismatch (instance failure).
4. Unwanted designed products of adaptations, such as evolved defenses (unpleasant successful function).
5. Unwanted by-product of adaptations.
6. Unwanted consequence of neutral idiotypic genetic or environmental variation.
7. Calibrational mechanisms interacting with ancestrally normal range of environmental variation.
8. Environmental mutation interacting with a normal adult design.
9. Environmental mutation interacting with a normal calibrational mechanism during development.
10. Environmental mutation interacting with previously unexpressed genetic variation.
11. Environmental mutation interacting with adaptive targets during development.
12. Insufficiently successful tailoring to individual or local conditions.
13. Time–integration mismatch.
14. Unnecessary or obsolete design compromise.
15. Motivational adaptations to serve goals without modern payoffs.
16. Ancestrally reliable cue becomes unreliable in modern conditions.
17. Aesthetics.
18. Senescence-generated deterioration.
19. Parent–offspring conflict.
20. Social conflict (including manipulation by others).
21. Promotion of desirable idiotypic trait (e.g., intelligence, musical talent).
22. Intragenomic conflict.

Note. Categorization by causes, etiology, selection pressures, and adaptive function. See text for full explanation.

condition discrepancies. For example, psychopathologies can be the result of environmental conditions that are too different from what is evolutionarily expected, from genetic mutations or novel combinations that are too disruptive, or from an interaction of the two. As a result, psychopathologies are expected to have a heritable component but facilitated by abnormal environmental milieus. "Abnormal" is different from uncustomary: Abnormality is defined against an evolutionary rather than a cultural background, so that what is common now may be abnormal evolutionarily. Moreover, as Waddington's (1957) experiments indicated, as the environment is progressively changed away from the EEA, aberrant heritable differences, formerly unexpressed and undetectable as a result of canalization, will increasingly express themselves (Tooby & Cosmides, 1990a). In more evolutionarily normal environments, systems of developmental canalization using adaptive targets act to compensate for perturbations caused by genetic variation. However, as one reaches the edge of evolutionarily normal environments, the systems of canalization are less able to compensate for genetic perturbations, and more heritable pathologies will appear. Of course, variability in alleles and environment have always been present during evolution, and so developmental adaptations have been designed to withstand their effects to the extent that they commonly occurred during evolution. Combinations of alleles, developmental conditions, and adaptation states should have been debugged by selection in proportion to how frequently they occurred during our evolution. Reciprocally, the evolutionarily rarer the configuration, the more likely dysfunction will ensue. Institutional environments are likely to be more therapeutic the more they incorporate elements that resemble ancestral conditions (e.g., natural settings; small, stable social groups, natural light cycles, etc.).

Arguably, the most damaging kind of disruption that manifests itself at the "psychological" level is a perturbation in the specification of adaptive targets (e.g., in the proper ratio of seratonin receptors to seratonin levels). Because it is the goal state in a feedback-driven system, an important adaptive target that is misspecified will resist or defeat interventions. As a result, steps that may be successful in the short run will be neutralized in the long run (a common phenomenon with drug interventions). In contrast, as anyone who has witnessed the grace of a three-legged cat or a human amputee can attest to, individuals can recover from major damage to implementational mechanisms.

A very common kind of problem results from an environmental mutation interacting with a normal species-typical architecture designed to function in the ancestral world. To take a trivial example, modern humans live in an environment filled with fast food. Given properly functioning adaptations that make humans crave fat, salt, and sugar—hard to come by in ancestral environments—and efficient foraging algorithms (Smith & Winterhalder, 1992), 20th-century Americans eat more fat and sugar than our bodies were designed to handle. The resulting heart disease and tooth decay are diseases of civilization that are virtually unknown in populations that hunt and gather (Eaton, Shostak, & Konner, 1988). Treatable conditions of this kind are legion: Everything from alcoholism, test anxiety, and fear of flying, to gambling problems and incest may fall into this category. Perhaps the commonest form of this type of mismatch is the change, from ancestral conditions to modern conditions, in the validity of cues our psychological architectures evolved to use. Sweetness is no longer a signal of nutritional value and opiates in the brain are no longer a cue that one is engaging in an adaptive activity.

In addition to building species-typical design, developmental adaptations are designed to tailor or calibrate the phenotype to the specifics of the prospective environment to be encountered (often the adult local environment, but it could be a sequence of environments). To accomplish this, the adaptations necessarily use environmental variables sampled earlier to predict later conditions. If the structure of environments has changed and earlier sampling in the environment of ontogeny no longer predicts later conditions as they once did in the EEA, then the organism will be miscalibrated. For example, evolved psychological mechanisms may monitor early environmental cues that have proven reliable over evolutionary time in predicting the nature of the social world the child will be maturing into. Such cues may be used to calibrate the strength or threshold of activation of modules. To take one phenomenon, violent treatment in childhood increases the likelihood that a person has been born into a social environment where violence is an important avenue of social instrumentality. Therefore, the threshold of activation of one's mental organs should be lowered, so one is prepared to act in and cope with such a world. The observation that abused children are disproportionately aggressive when they become adults may be accounted for by a mechanism of this kind (Garbarino, 1986; McCord, 1979, 1983). In *Culture of Honor*, Nisbett and Cohen (1996) described how men raised in pastoralist societies are quicker to take offense, quicker to escalate a conflict, and manifest higher levels of aggression than men raised in agricultural societies, and they documented how such patterns persist culturally for generations beyond their conditions of origin. For individuals no longer living in a context where this kind of reaction is either effective or appropriate, one can imagine situations in which a person might wish to change this setting and seek treatment to do so. Fears of the effects of media violence on children reflect this same concern.

A related category results from the discrepancy between evolutionary averages and individual cases. Adaptations have been engineered to perform well within ancestral statistical distributions, although any individual case may fall outside the range where the designed effect is beneficial. Yet the decision to treat the effect lies with individuals. Indeed, most adaptations operate through a form of adaptive betting. As in all bets, one can lose, even though the betting strategy used may be good or even optimal. Losers in fights and competitions, sufferers from unrequited love, and individuals who take risks that do not pay off may all suffer from negative outcomes, but not necessarily from dysfunctions. So, in addition to design failure and developmental failure, is what one might call instance-failure: The adaptation failed to produce a useful outcome in a particular case, because the world is stochastic.

Time–Integration Mismatches

Adaptations are designed to make allocational trade-offs between short-term and long-term outcomes as a function of alternative temporal event structures. For example, if there is a threat to survival, processes that are beneficial over the long-term (digestion, immune competence) may be suspended in favor of increasing the probability of short-term survival. This is why stressful events (interpreted by organisms as predicting short-term threats to

survival) if continually renewed, compromise the health of the individual (Sapolsky, 1992). Similarly, deferring gratification and controlling impulses only makes sense if the payoff remains to be harvested at the end of the deferral. Calibrating the neural basis of optimal impulse control depends on the long-term structure of the environment: Safe, stable environments ought to lead to calibration of greater impulse control, whereas high variance, capricious social environments ought to encourage nondeferred gratification (e.g., Wilson & Daly, 1997). If the adaptations are designed to integrate probabilities of future conditions over time in a way that no longer corresponds to the temporal structure of the world, then unnecessary suffering results.

To take a poignant example, tactile stimulation from a caregiver appears to be necessary for robust growth in infants and young children. It has long been noted that children in orphanages who have adequate food and health care frequently show a "failure to thrive." The effects of touch on growth have recently been demonstrated in controlled studies (reviewed in Field, 1995). An evolutionary interpretation is that the child's growth rate is calibrated on-line by cues to how much social investment is likely to be available in the future, during possible periods of deprivation. Touch would be a cue to the level of commitment by the caregiver and the amount of free energy the caregiver has. Throughout our evolutionary history, infants were born into different caregiving environments: The mother might be healthy or sick, well fed or undernourished, alive or dead. Low levels of touch would predict an environment in which investment is likely to be cut back at any time, and a child with little social support may be better off sequestering reserves against the possible dramatic reduction of support that is likely to be imminent rather than embarking on growth that would only increase mandatory energy requirements. This touch–nourishment relationship no longer obtains in post-Dickensian caregiving institutions, yet the infant's adaptations are designed for a harsher ancestral world. This is an example in which calibrational adaptations interacting with an ancestrally normal range of environmental variation can produce a harmful result.

Conflict

Conflict is another source of value–condition divergence. Conflict is often claimed to be pathological but is often an evolutionarily functional phenomenon. Intragenomic conflict, already discussed, is one example (Cosmides & Tooby, 1981; Dawkins, 1982). More importantly, adaptations acting in different individuals (between mates, family members, mothers and fetuses, competitors, coworkers, friends, or enemies) may impel the interactors to pursue mutually inconsistent agendas, leading to many forms of suffering that may cause participants to seek treatment. Marital discord, parent–offspring conflict (of which the most well-known expressions are sibling rivalry and weaning conflict; Trivers, 1974), intergroup conflict, defects in reciprocity mechanisms (Glantz & Pearce, 1989), deception, and maternal–fetal conflict (Haig, 1993) are all cases. More generally, adaptations to pursue agendas at the expense of others may be operating adaptively while causing suffering to one or all parties. The world was not built so that others volunteer to be simple extensions of one's will, and so the nonconformity of others to one's wishes is a major cause of human suffering not attributable to dysfunction. The legal system can be seen as a set of collectively mobilized incentives to counteract motivational adaptations that impel individuals to injure or exploit each other.

Remaining categories include evolved design compromises that are no longer necessary, senescence, and proximate motivations in which the long term payoff no longer corresponds to anything sensible in the modern world. For example, this last category includes (a) paternal sexual proprietariness, which evolved to function in a world of small-scale bands where daughters were exchanged by patrilines, and (b) the impulse to form male coalitions, which was designed to operate in a world of small-scale feuding and warfare.

The Importance of Characterizing Adaptations

Adaptationist analysis is not a post hoc semantic game. It is an inferential tool that helps to guide empirical investigations into psychological and medical phenomena (Nesse & Williams, 1994; Tooby & Cosmides, 1992). Carving the architecture into its constituent adaptations provides a privileged framework for building adequate theories of what elements in the system will be causally connected to each other, and why. Because mental organization is created by adaptations, the clusters of symptoms constituting a syndrome will tend to be explained by the organization and features of the adaptation or adaptations that are impaired.

As evidence about the inventory of cognitive adaptations accumulates, new and more precise ways of conceptualizing known disorders may result. For example, individuals exhibiting neural damage or psychiatric disorders are often identified as reasoning oddly, but it is vital for neuropsychologists and therapists to be able to characterize exactly which pieces of functional machinery have been impaired. To do this, one needs to correctly inventory and characterize the set of species-typical reasoning competences present in normal humans.

In our own work, we have been involved in a controversy about whether performance on certain kinds of social reasoning tasks is caused by (a) a general-purpose mechanism handling all types of reasoning, social or not, (b) a single *permission schema* designed for reasoning about deontic rules from many domains (e.g., Cheng & Holyoak, 1985, 1989), or (c) two functionally distinct reasoning systems (out of a larger constellation of reasoning systems). In this last case, one mechanism is specialized for detecting cheaters on social contracts and another is specialized for detecting when a person has failed to take appropriate precautions in a hazardous situation. We believe that the two-mechanism view is supported by a range of evidence, including cognitive experiments using transformations of input and context, priming experiments, cross-cultural experiments, and experiments with patients with focal brain damage (Cosmides & Tooby, 1992, 1997; Fiddick, Cosmides, & Tooby, 1995; Rutherford, Tooby, & Cosmides, 1996; Stone et al., 1996, 1999).

In particular, if we are correct in believing that, in addition to a social exchange system, there is also an inference system specialized for detecting violations of precautionary rules (rules of the form, "If you are in a hazardous situation H, then take precaution P"), then this identifies a new species-typical competence present in ordinary individuals. It also suggests a new approach to conceptualizing obsessive–compulsive disorder (OCD). OCD would be caused as a breakdown in this system. Alternatively, it might turn out that OCD results only when two conditions are met: There

is an overactivation of the precaution schema and a defect in the mechanisms that govern task-switching, similar to that seen in certain kinds of frontal lobe damage (e.g., Duncan, 1995; Schwartz, 1996).

The proposed precaution circuit is, in essence, a checking mechanism: It causes one to seek out evidence about whether a precaution has been taken against a potential danger. This is independent of facility with reasoning about cheaters. If such a system exists, an evolutionary approach would suggest that such a system might be calibrated by life-history variables, because the costs and benefits of risk taking vary systematically across the lifespan. So, for example, its threshold of activation might be low in adolescent males who are taking many risks, as well as in first-time parents. If so, then one can imagine situations in which behavior genetic variation in an individual's cocktail of neurotransmitters, changes in biochemical state (caused, e.g., by pregnancy, drugs, or prolonged stress), or even experiential factors cause the precaution mechanism to become overactivated. This could result in the kinds of behaviors one frequently sees in people suffering from OCD: compulsive checking to make sure that the stove is off, that the front door is locked, that one has washed germs off of one's hands, that one hasn't accidentally thrown out something of value that will be needed later, and so on.

Moreover, a precaution system might itself evolve secondary adaptations designed to modulate its sensitivity depending on life stage and situation. For example, it seems plausible that children are less able to identify and avoid hazards. As a result, adults, when they become parents, would have had added precautionary computational problems engendered by the need to look out for their children. Over evolutionary time, mothers who were more successfully vigilant about the safety of their small children, anticipating dangers and averting them, raised more children to adulthood. If this were true, then selection plausibly might have designed the precaution schema to be activated more easily in new mothers, and the hormonal changes of pregnancy might modulate this activation. In most women, this would manifest itself as mothers spending more time than nonmothers imagining possible dangers and ways of averting them, and the condition would be an adaptation, not a disorder. For a woman whose precaution schemas are already more easily activated than average (e.g., as the result of normal genetic variation), the hormonal changes of pregnancy might push her ruminations and checking behavior beyond the bounds of normal variation. Indeed, the symptoms of OCD often do increase after pregnancy (Kaplan & Sadock, 1995).

Further investigations of the ancestrally functional computational structure of precaution schemas might throw new light on the various ways in which OCD manifests itself. Analysis of the adaptive problems involved suggests that the precaution module ought to include functionally distinct subcomponents for dealing with physical hazards, disease and contamination, preparation for the future, social disasters, and so on. If so, then one of these components could (in principle) be damaged or sensitized in some way while the others remain intact, giving rise to different forms of OCD and their clustered symptoms (e.g., compulsive handwashing versus fear of harming others). It also seems likely that some phenomena now categorized as OCD may be more parsimoniously construed as different clinical conditions. For example, compulsive sexual thoughts may be better conceptualized as a dysfunction in another cognitive adaptation, one related to mating.

In sum, trying to decide where a condition belongs in an evolutionary taxonomy is not a sterile exercise in categorization. Achieving a genuine understanding of the adaptations that comprise our bodies and minds, how they interact, and how they break down, can produce new and important insights into how to conceptualize and, therefore, treat conditions that cause human suffering.

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