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Series Editors: Todd K. Shackelford · Viviana A. Weekes-Shackelford

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The Evolution of Psychopathology

Springer

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Preface

In April 2016, we welcomed several dozen scholars from North America, South America, Europe, and Asia to join us at Oakland University in Rochester, Michigan, for a two-day interdisciplinary conference on "The Evolution of Psychopathology." We invited as panelists some of the leading minds in psychopathology from many different disciplines, including psychology, neuroscience, criminology, biology, anthropology, archeology, law, philosophy, and medicine. These scholars had conducted and published substantial work addressing various aspects of psychopathology from an evolutionary perspective. This volume showcases the groundbreaking empirical and theoretical work from several of these panelists and other distinguished conference guests.

The volume opens with a wide-ranging contribution from Fabrega, Jr. and Brüne, "Evolutionary Foundations of Psychiatric Compared to Nonpsychiatric Disorders." The authors discuss why psychiatric disorders, when examined from an evolutionary perspective, are different from nonpsychiatric disorders. Fabrega and Brüne draw on ideas and insights from evolutionary social and life sciences, including population genetics, gene-culture coevolution, evolutionary psychology and anthropology, evolutionary medicine, behavioral ecology, and human evolutionary developmental biology. The authors discuss how and why these and related sciences provide an appropriate framework for identifying and understanding the distinctive features of psychiatric disorders. According to the authors, principles and generalizations derived from evolutionary biology provide cogent and epistemically penetrating facts about *Homo sapiens* and help explain fundamental aspects of the nature of human medical disorders. However, their relevance to psychiatric compared to nonpsychiatric disorders is insufficiently appreciated. According to Fabrega and Brüne, teasing apart elements of this framework helps to explain the origins of unique sociological, political, cultural, as well as biological problems that psychiatric disorders (i.e., as compared to nonpsychiatric disorders) impose on human populations including prehistoric, ancestral, and even nonhuman ones.

In Chap. 2, "Taking People as They Are: Evolutionary Psychopathology, Uncomplicated Depression, and the Distinction Between Normal and Disordered Sadness," Wakefield, Lorenzo-Luaces, and Lee review the results of a research

program designed to test a controversial hypothesis generated by an evolutionary approach to psychopathology. This evolutionary approach is in conflict with the standard symptom-based approach to diagnosis advocated by the American Psychiatric Association's official *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (DSM-5). Wakefield and colleagues note that the conflict arises because evolved features that are part of normal human functioning may be undesirably "symptomatic" or undesirable from our current value perspective. Such problematic normal conditions include grief, anxiety regarding potential threats, and the taste for fat and sugar. Evolved features such as these were useful or neutral when they evolved but may be undesirable in our current, quite different context. Such features, although often deserving treatment or some other social response, are not necessarily medical disorders simply because they entail suffering or socially disvalued behavior. According to Wakefield and colleagues, distress and the perceived need for help also occur in normal conditions in which nothing is going wrong in the individual because evolution has favored fitness over comfort and pleasure. This perspective has important implications for the classification, research, and treatment of mental disorders as well as for DSM-5's nosological distinction between disorders and "Z Codes"—non-disorders about which clinicians may nonetheless be consulted.

Kennair and colleagues contribute Chap. 3, "Depression: Is Rumination Really Adaptive?" Within evolutionary approaches to depression, there is antagonism between theories that propose that depression and symptoms of major depressive disorder are not adaptive and those that propose that depression is adaptive. An example of the latter, the Analytical Rumination Hypothesis describes how rumination and depressive symptoms provide solutions to complex social problems and, therefore, should be promoted rather than treated. Paralleling the conceptual development of the Analytical Rumination Hypothesis, within mainstream clinical science a different approach to depression and the treatment of major depressive disorder has been developed. This approach views rumination as a maladaptive maintaining factor of depression, rather than as the healing, problem-solving factor. Treatment in the form of Metacognitive Therapy involves eliminating rumination to reduce depressive symptomology. As such, the Analytical Rumination Hypothesis and Metacognitive Therapy agree that rumination is central to depression, but they disagree on whether depressive symptoms are adaptive and desirable. Kennair and colleagues review the Analytical Rumination Hypothesis and Metacognitive Therapy and consider the arguments from both positions.

In Chap. 4, "The Evolution of Social Anxiety," Brosnan, Tone, and Williams begin by reviewing evidence that social relationships constitute a rewarding context for most people, providing a source of support and nurturance, as well as protection against loneliness, depression, and even death. Interpersonal relationships can also be stressful and can contribute to psychological and physical health problems. A common manifestation of such difficulties is social anxiety (SA), an excessive fear of negative evaluation that can lead people to avoid social engagement. Its associated behavior patterns may result in a clinical diagnosis of social anxiety disorder (SAD). In recent years, researchers have begun to explore alternate approaches to understanding this SA, including the hypothesis that, although SA has detrimental

effects that may require treatment, it is an adaptive behavior pattern that has been selected for the benefits it may have provided and that continue to be relevant. Understanding why this suite of traits was under positive selection pressure would not only provide a better understanding of when to expect SA symptoms, but also may help us decide when treatment is needed and, when indicated, to more successfully treat severe manifestations.

Brosnan and colleagues first summarize the history of thought surrounding SA, then outline evolutionary frameworks that scholars have proposed. They next discuss the ways in which recent work on social behavior in animals, particularly other primates, informs our understanding of these models. Although the original models focused on aggression and dominance in eliciting interpersonal difficulties, recent work emphasizes the formation and maintenance of peaceful social relationships and highlights situations in which it may be to an individual's advantage to hesitate in social circumstances. Brosnan and colleagues review new research demonstrating that there are times when appeasing or avoiding may be prudent social strategies. Building on this more recent work in primates, Brosnan and colleagues outline an updated evolutionary model of SA. They highlight several open questions around SA, and its evolutionary history, that will need to be answered to improve prediction and treatment of SA.

In Chap. 5, "Jealousy, Infidelity, and the Difficulty of Diagnosing Pathology: A CBT Approach to Coping with Sexual Betrayal and the Green-Eyed Monster," Buss and Abrams argue that humans have adaptations for infidelity, as well as defenses against a partner's infidelity—centrally the emotion of jealousy. Both create problems that bring couples to therapy. Diagnosing jealousy as pathological versus normal is difficult, because infidelity has evolved to be concealed from the betrayed partner, producing a signal detection problem. Because missing a partner's infidelity has been more costly in evolutionary currencies than falsely suspecting a partner of infidelity, selection has created an error management cognitive bias to over-infer a partner's infidelity. Moreover, adaptations for jealousy become activated by predictors of infidelity, such as mate value discrepancies, when no actual infidelities have occurred. Buss and Abrams argue that cognitive-behavior therapy (CBT) offers several ways to address these complexities. One way is to highlight potential mismatches, distinguishing between jealous emotions that were functional in ancestral environments but are less so in modern environments. A second is to distinguish between the goal of personal well-being and reproductive outcomes. According to Buss and Abrams, understanding the evolutionary logic of jealousy provides patients with conceptual tools for cognitively reframing jealousy and infidelity.

Hill, Hunt, and Duryea propose in Chap. 6, "Evolved Vulnerability to Addiction: The Problem of Opiates," that the involvement of the opioid system in social attachment and physical pain contributes to the current prevalence of addiction to opiate drugs. Hill and colleagues first describe the process and course of addiction. Then they briefly review the brain systems involved in opiate addiction and note the common opioid drugs of abuse. The authors then discuss factors that may explain why opiate addiction has become such a problem today. Hill and colleagues argue that an evolutionary perspective is helpful in understanding addiction, as this perspective led to the current understanding that physical and social pain use the same brain pathways. The authors present recent evidence for the role of opioid brain systems in social and

physical pain. They note that, although these vulnerabilities are shared by all humans, individual differences exist in the opioid systems in the brain that contribute to both types of pain. Hill and colleagues conclude the chapter by describing several promising avenues of treatment, intervention, and prevention of opiate addiction.

In Chap. 7, "Criminology's Modern Synthesis: Remaking the Science of Crime with Darwinian Insight," Kavish, Fowler-Finn, and Boutwell showcase recent efforts to unify criminological knowledge using evolutionary concepts, with a particular focus on life history theory. Kavish and colleagues review recent theoretical work in which a host of crime correlates are integrated using insights from evolutionary biology. Kavish and colleagues first briefly outline the basics of life history theory by drawing on examples from research throughout the animal kingdom and discuss why these same principles are relevant to the study of human behavior. The authors conclude by discussing some of the ideas proposed by evolutionary criminologists and highlight the additional research that is likely to be fruitful in this area. Kavish and colleagues draw attention to evolutionary approaches to understanding human behavior and encourage others to empirically test the hypothesis that criminal behavior does, in fact, have a biological basis and relevant evolutionary explanations.

In Chap. 8, "Excruciating Mental States," Perry notes that current models of suicide emphasize the connection between suicide and mental illness. According to Perry, the focus on mental disorders and the rare phenomenon of suicide has shielded a more important problem from view, one that is obvious upon reflection but rarely named: extreme subjective mental suffering, or excruciating mental states. Perry makes a compelling case that excruciating mental states mediate the relationship between mental illness constructs and suicide. Furthermore, prolonged excruciating mental states do not serve any purpose that can justify refusing to ameliorate them. According to Perry, excruciating mental states can be relieved directly, rather than as a hoped-for consequence of the treatment of a mental illness. Unfortunately, drug prohibition policies preclude the use of safe, effective solutions. Perry argues that the difficulty of communicating extreme mental pain prevents sufferers from being helped, and that simply having a conceptual category for the phenomenon may ease the difficulty of communication.

In the concluding Chap. 9, "Anthropathology: The Abiding Malady of the Species," a view of negative human evolution is advocated by Feltham, as a balance against the many positive, sometimes romantic, or academically cautious and narrow accounts. Feltham advances "anthropathology" as a hypothetical, quasi-singular phenomenon with multiple roots and manifestations, and in the process suggests a chronology and a speculative etiology. According to Feltham, anthropathology is characterized by damaging features such as violence, greed, deception, extended niche construction, and complex suffering on a scale not known among other species.

The Evolution of Psychopathology showcases the considerable and sweeping intellectual value of an interdisciplinary approach to human psychology and behavior. Guided by Darwin's insights, the contributions to this wide-ranging volume provide a compelling case for an evolutionary analysis of psychopathology.

Rochester, MI, USA

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Evolutionary Foundations of Psychiatric Compared to Nonpsychiatric Disorders

Horacio Fabrega Jr. and Martin Brüne

Introduction

With the recent publication of a new edition of a prominent classification system of psychiatric disorders (i.e., DSM-5), and the prospect that the next version will retain the conventional zeitgeist of psychiatry, this chapter discusses why such disorders, when examined from an evolutionary standpoint (e.g., as per cause, manifestation, and impact), are different from disorders germane to the rest of medicine. Specifically, we draw on ideas and insights of evolutionary social and life sciences, including population genetics, gene-culture coevolution, evolutionary psychology and anthropology, evolutionary medicine, behavioral ecology, and human evolutionary developmental biology. We discuss how and why these and related sciences provide an appropriate framework for understanding distinctive features of psychiatric disorders and its practical exigencies (see Fabrega, 1997, 2006a, 2009, 2013 for general background).

Principles and generalizations derived from evolutionary biology provide cogent and epistemically penetrating facts about *Homo sapiens* and help explain fundamental aspects of the nature of human medical disorders (Gluckman, Beedle, & Hanson, 2009; Nesse & Williams, 1994; Stearns & Koella, 2008; Trevathan, Smith, & McKenna, 2008). However, their relevance to psychiatric compared to nonpsychiatric disorders is insufficiently appreciated. A frame of reference embracing

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evolutionary social behavioral sciences is of special relevance to comprehending distinctive theoretical and empirical implications tied to *both* psychiatric and nonpsychiatric medical disorders. However, teasing apart elements of this framework helps explain the origins of unique sociological, political, cultural, as well as biological problems that psychiatric disorders (i.e., as compared to nonpsychiatric disorders) impose on human populations including prehistoric, ancestral, and even nonhuman ones (Fabrega, 1997, 2002, 2006a, 2006b, 2009, 2011, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d; Goddard, Wierzbicka, & Fabrega, 2013).

General Background

More than 50 years ago, George Engel pointed out that for a full understanding of all medical problems, psychiatric as well as general medical, one needed to integrate diverse factors which range across biology, psychology, and sociology (Engel, 1960, 1977). He emphasized that no one part of a "biopsychosocial" approach to a medical disorder was scientifically more valid than the other two (Fabrega, 1974, 1997). Both types of conditions are fully comprehended when viewed in terms of factors spanning levels and types of interconnected human systems, processes, and problems, a cornerstone of Engel's (1960) unified theory of health and disease.

Our chapter builds on Engel's formulation and situates it intellectually in a biological/genetic and cultural evolutionary frame of reference. How and why biological systems which constitute an individual's breakdown, and the medical disorders which they bring in their wake, are central tenets of the evolutionary social behavioral sciences. The costs of imperfection, disintegration, and breakdown of naturally selected programs governing biologically adaptive functions are responsible for medical disorders that all human societies through their social, psychological, cultural, and behavioral resources (i.e., "ethnomedicines"—see Fabrega, 1974, 1975, 1976a, 1997, 2013, n.d.-a) have disvalued and sought to control.

Principles of Darwinian adaptation provide compelling explanations for the causes and consequences of human disease of all types (Fabrega, 1975, 1976a, 1976b). The relevance of the role of evolutionary biology in general medical disorders, including psychiatric ones, is evident and stands firm for all life forms. It manifests in animal models of disease, medical problems of higher primates, ancestral human population history, medical ideas and practices of human nomadic and agricultural groupings in diverse regions of the world, the medical literatures of "Great Civilizations" of antiquity (i.e., India, China, Mesopotamia, Meso-America, and Africa), and societies and medical traditions of early modern as well as Anglo-European history. In human history broadly conceived, cultural factors have played a significant and special role in how psychiatric disorders have been perceived, labeled, and dealt with in social, political, and moral as well as in medical terms (Brüne, 2001, 2007; Fabrega, 1997, 2002, 2006a, 2013).

A scientific theory about psychiatric conditions involves understanding both their neurobiological nature, on the one hand, and relevance of social, interpersonal,

and cultural psychological factors, on the other. The theory and practice of general medicine underscore how modern biological sciences have contributed to the welfare of human populations as a moral imperative. Psychiatry shares this ethos. However, its position in society and in Western intellectual imagination is set apart from that of general medicine institutions, partly because manifestations of psychiatric disorders impinge on and sometimes intrude into the workings of and political and cultural traditions in *unique and distinctive ways*. We discuss in this chapter why and how the political and cultural baggage of human behavioral problems associated with psychiatric disorders in modern societies have deep evolutionary roots (Fabrega, 1997, 2002, 2006a, 2006b, 2009, 2011, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d). We maintain that basic biological (brain-based), philosophical (i.e., ontology and epistemology), symptom expression (psychological and interpersonal), and cultural and political impact of psychiatric contra nonpsychiatric disorders are sharply different.

Relevance of Evolutionary Social Sciences for Medicine

Tenets of evolutionary behavioral social sciences provide an encompassing and a penetrating frame of reference for the study of the general behavior and medical disorders of human populations. They are as comprehensive *and basic* as are those of genetics and molecular biology which encase clinical neuroscience psychiatry. The study of the influence of genes on neurocognitive foundations regulating behavior is an idea which the modern Neo-Darwinian synthesis has incorporated (Dobzhansky, 1973). Such sciences provide a vision and explanatory rationale for understanding the origins, functions, and changing dynamics wrought by natural selection in light of the tenets of adaptation and biological problem solving in ancestral as well as contemporary environments. They are thus relevant to understanding the nature of general medical as well as psychiatric conditions (Brüne, 2011; Brüne & Hochberg, 2013).

A primary focus of the emerging field of evolutionary medicine is understanding the causes, mechanisms, ontogeny, and consequences of somatic pathologies and disease processes, viewed in the context of Darwinian ideas, known as the proximate and ultimate causes of traits (Brüne, 2014a, 2014b; Nesse, 2013; Tinbergen, 1963). As explicated in several compendia about the effects of adaptations to conditions of life in ancestral human environments, evolutionary factors have had a profound impact on the social biology of internal bodily functions and structures of modern human populations relevant to medicine (Gluckman et al., 2009; Nesse & Williams, 1994; Stearns & Koella, 2008; Trevathan et al., 2008). An evolutionary perspective has thus provided insights into the prevalence, causes, characteristics, and consequences of modern human diseases. This translates as differences in constellation of medical problems keyed to distinctive changes in the social biology and behavior of modern human populations and corresponding social and cultural circumstances distinctive of modern physical and social environments (i.e., the

mismatch theory of "diseases of modern civilization"; Gluckman et al., 2009; Stearns & Koella, 2008).

Natural selection of human biological genotypic traits associated with the health-influencing environments of early human populations was determined by social ecological constraints affecting diets; physical activity patterns; alterations in expression of molecular genetic systems and processes; and distinctive levels and forms of diseases caused by pathogens, parasites, immunological demands, and general physiological and metabolic processes and functions (Fabrega, 1974, 1997, 2013; Gluckman, Low, Buklijas, Hanson, & Beedle, 2011; Gluckman et al., 2009). Such constraints influenced levels, values, and modalities of internal regulatory system functions creating different patterns of vulnerability, resilience, as well as health responses and outcomes (Fabrega, 1974, 1975; Gluckman et al., 2009). Differences involving medical and health ecology were mirrored in high levels of injuries, physically unpredictable risks and stressors, and high prevalence of parasites, vectors, and other pathogens, which have inhabited and plagued human populations for hundreds of thousands if not millions of years (Barnes, 2005; Ewald, 1994; Moore, 2002; Nunn & Altizer, 2006; Van Blerkom, 2003).

Evolutionarily determined developmental plasticity (West-Eberhard, 2003) is integral to the functional operation of internal regulatory and stress response systems of organisms (Chandak et al., 2009; Ellis, del Giudice, & Shirtcliff, 2012; Gluckman et al., 2009). The ways such systems are shaped influence characteristics, timing, and trade-offs associated with strategies that individuals adopt in regulation of major biopsychosocial processes and mechanisms, termed life history strategies (Charnov, 1993; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Stearns, 1992). Such strategies, discussed in more detail later, play a critical role in modulating levels of vulnerability and resilience to harsh, adverse, and unpredictable physical and social environments (Korte, Koolhaas, Wingfield, & McEwen, 2005; Ellis et al., 2009, 2011; Del Giudice, Ellis, & Shirtcliff, 2011). Negative outcomes of this nexus of factors involve general medical disorders and in addition are especially relevant to psychiatric disorders. The requirements of adaptation to life conditions and health problems encountered in ancestral environments correspond to and are partly causative of organic biological health costs which are particularly medically burdensome in modern human environments and especially psychosocially and in a cultural psychological sense with respect to psychiatric disorders.

By explicating the evolutionary logic of processes and mechanisms surrounding and responsible for disease, principles and dicta of evolutionary behavioral social sciences enhance the effectiveness of all types of clinicians. It furnishes them with a powerful objective and impersonal "naturalistic" explanatory framework for construing forms of pathology and disease as a recurring universal predicament, and potential for suffering. By generating therapeutic activities and advice consistent with the logic and rationale of evolutionary social behavioral sciences, the impact of a physician or psychologist on his or her patient is rendered more cogent and compelling. Information presented to patients during therapeutic exchanges would take into consideration the latter's behavioral resources, cognitive executive functions, and potential power over causes and manifestations of disease. Employment of an

evolutionary logic of explanation is of relevance to psychiatric as well as to general medical disorders. However, the distinctive cultural psychological and sociological factors tied to psychiatric compared to general medical disorders (i.e., devolving from their special ontology and epistemology) are made especially problematic for reasons addressed later.

Accordingly, evolutionary medicine is of considerable value not just as a theory for understanding disease processes. It functions as a basic science comprising an explanatory calculus for ontogenetic development, natural selection of biological mechanisms, evolutionary history, and pathophysiology of human disease. It provides a modern clinician with a scientific ethic about the ethos or "spirit" of disease, which broadens intellectual understanding of etiology, pathogenesis, and natural history. Disease is seen as a natural, recurring, and universal outcome, which all human populations have suffered. Factors which are distinctive of psychiatric disorders compared to general medical ones and which play a central role in their causation manifestations and impact are addressed later.

An Evolutionary Social Behavioral Sciences Approach to Medical Disorders

Problems posed by disease and pathology are principal agents of natural selection and like all biological problems have to be solved for organisms to persist and this fact requires an evolutionary understanding of the attributes of organisms that encompass their cognition, semantics, language of thought and experience, and forms of awareness and consciousness (Baars, 1989; Dehaene, 2014; Tononi & Edelman, 1998), modes of thought, interpersonal and cultural behavior, and even evolutionary semantics (Fabrega, 1975, 1997, 2002, 2013; Goddard et al., 2013). As discussed later, these parameters of behavior are captured in a concept that exemplifies the social and evolutionary biology of "mentalization." Put simply, when cast in an evolutionary framework, medical problems are universal occurrences and require explication through theory, ideas, and methods of traditional evolutionary social behavioral sciences which include neurobiology of consciousness and related forms of psychological behavior as exemplified in cognitive ethology (critical anthropomorphism) and comparative cognitive psychology (Fabrega, 2006b, 2011, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d). Their causes, pathogenesis, manifestations, and effects are as fundamentally important to human societies as they are to nonhuman primates and other animals (Fabrega, 1997, 2002). In light of modern human biomedical science, constructs referring to general diseases and disorders are explained as breakdowns of internal regulatory systems which give rise to disturbances in well-being and function and the need for correction action (Fabrega, 1974, 1975). Morbid effects of medical disorders include pain, misery, sickness, and impairments in function and well-being, which individuals universally find aversive and in response to which they institute corrective actions. Studies in medical anthropology

substantiate this generalization (Fabrega, 1974, 1975, 1976a, 1976b, 1997). This would seem to support the view that medical problems belong "naturally" under the umbrella of evolutionary theory which, starting with representatives of genus Homo, is a cornerstone of a general anthropological understanding of the human condition and its staying power as a consequence of solving biological problems generated by the exigencies of natural selection.

Evolutionary theory centers on the dynamics and dialectics of human populations pitted against prevailing unpredictable and potentially adverse environments. Conversely, from a philosophical standpoint, qualifications about biopsychosocial parameters of medical disorders do not fit as comfortably in a strict evolutionary biology frame of reference as they do in a general medical one. Biological problem-solving routines are fundamental necessities and imperatives tied to the logic of evolutionary explanation. On the other hand, deviations, breakdowns, and normality (and diseases along with them) are human concepts that are socially, politically, morally, and culturally as well as biomedically value laden. In cultural anthropology, the biomedical sciences are a product of and germane to the social biological characteristics of populations, as based on prevailing social ecologies and symbolic conventions that impact on definitions and approaches to health and disease. Medical sciences are about things that are "bad" and "undesirable" and hence "not wanted" (Fabrega, 1974, 1975, 1997). The backdrop of modern medical concepts, in other words, is based on populations that happen to have lived and continue to live in ecologically, historically, and culturally distinctive (i.e., modern) environments. However, the constructs of evolutionary biology are for the most part objective, impersonal, timeless, universal, and value free.

In light of the ubiquity of somatic pathologies in animal and human populations and because conditions which they give rise to are central to necessities and vicissitudes tied to evolutionary processes, it is proposed that disease and disorder are constructs that should and need to be grounded in the logic and rationale of evolutionary social science. They constitute costs, trade-offs, and constraints imposed by evolutionary imperatives and necessities tied to optimality theory. To reconcile apparent discontinuities in the semantics of key constructs of medicine compared to evolutionary biology concerning matters relevant to disease and disorder (e.g., normality, deviation), it is desirable to delve into differences between the philosophical emphases of these two scientific traditions.

First, the tenets of Neo-Darwinian science and theory exemplify objectivity and impersonality. Second, in contrast to the natural theology and world views of ancient civilizations (i.e., Ancient Greece, India, and China), evolutionary as well as biomedical dicta surrounding the logic of disease are not framed in ultimately metaphysical notions involving personal design, sanctity, and spirituality (Fabrega, 1997, 2009, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d). Third, from the standpoint of evolutionary theory, many diseases and disorders of medicine and psychiatry are understood as stemming from perturbation, fragmentation, or breakdown of inherited generic categories, mechanisms, and multileveled regulatory systems, which are constrained or adaptively costly in relation to prevailing social, behavioral, and cultural ecologies, especially as these are now exemplified in modern environments. Fourth, virtually

all descriptions of basic medical categories, especially psychiatric ones, are out of sync with the scientific ontology and epistemology of ideas germane to evolutionary social science. In the latter science, relevant constructs covering medical problems refer to natural and necessary costs and constraints associated with trade-offs which are intrinsic to natural selection, life history, and evolutionary imperatives and necessities more broadly. Finally, viewed comprehensively and in no way to demean the advantages of medical specialization, the causes and adaptive costs of diseases and disorders, regardless of which discipline owns jurisdiction over them, all conform to the logical imperatives and dialectical unfolding of processes germane to evolutionary social science and its inventory of concepts and principles (e.g., natural selection, social ecology contingencies, gene-environment correlations, adaptation, constraints, optimality theory).

To put it baldly, many of the "deviations" and "breakdowns" that constitute nonpsychiatric and psychiatric disorders represent inevitable and even sometimes adaptive response patterns of biological functioning due to inevitable trade-offs between benefits and costs associated with the recurring clash of organisms and environment (Fabrega, 1997, 2002, 2013; Nesse & Williams, 1994).

Theoretical and empirically validated understanding of medical conditions when examined from a strict evolutionary point of view does not reflect political and philosophical quandaries found in the medical discourse typical of modern societies. Differences involving the social and cultural valuation and consequences of evolutionary compared to medical explanations of health disturbances (i.e., their ontology and epistemology) stand firm with socially, symbolically laden labels prominent in medical discourse compared to evolutionary ones. As suggested earlier, it is arguable whether conventional ideas and explanations of medical disease and/or psychiatric disorder properly "belong" in an evolutionary framework. The former are modern human concepts the cultural overtones and general meanings of which correspond roughly to analogous terms in (modern and non-modern) human languages and cultures and in this respect loosely refer to human universals (Fabrega, 1974, 1997). Because of their quintessential modernist, social culturally centered ontology and epistemology (i.e., what medical disorders are and how they are expressed and meaningfully explained), when referring to phenomena relevant to the study of disease and disorder in a general and especially evolutionary frame of reference, preferable terms to adopt are "condition of medical interest" or CMI, and "condition of psychiatric interest" or CPI.

The present discussion acknowledges the cogency of Engel's (1960, 1977) unity conception of health and disease as exemplifying biopsychosocial phenomena. It also provides a more precise analytical language for navigating across two types of conceptual territories: exemplifying the logic of evolutionary biology compared to medicine, on the one hand, and that is associated with the evolutionary signatures of general medical compared to psychiatric conditions, on the other.

Adopting a unitarian rationale about medical problems (as conditions of general medical interest or CMI) concedes the relative influence of changing environments on the prevalence, natural history, and biopsychosocial manifestations of CMI as well as CPI. A unitarian conception of both conditions accepts the relevance of

evolutionary and medical science explanations about causes, origins, and consequences of all medical conditions. The qualifier "of interest" underscores contemporary differences in the logic of explanation and significance (the *raison d'être*) as well as practical directives germane to value-laden clinical medical discourse and value-neutral evolutionary discourse.

To recapitulate, explanations in evolutionary social science operate from a distant and objectively descriptive vantage point which is different and somewhat inconsistent if not in contrast to the vantage point of medicine. The latter by definition exemplifies a correctional directive which targets value-laden problems stemming from agonies, miseries, and consequences of human biopsychosocial pathologies in its various forms (Fabrega, 2002, 2013). Such a rationale has constituted the focus of medicine throughout human evolutionary and cultural history (Fabrega, 1974, 1997, 2002, 2006a, 2006b, 2009, 2011, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d). Disparate connotations and social consequences regarding the rationale of evolutionary social science compared to medicine may seem counterintuitive. Its special connotations need to be made clear, especially in light of historic examples where the two vantage points were lumped together, leading to horrific consequences of "social Darwinism" by confusing "is" with "ought" (Brüne, 2001, 2007).

Differences in the Evolutionary Biology and Cultural Psychobiology of Medical and Nonmedical Disorders (CMI and CPI)

General Considerations

The relevance of evolutionary social behavioral science on the special character of CPI compared to CMI is made clear by considering how characteristics of these two types of medical conditions play out in social behavioral settings and stages determined by evolutionary dialectic. In general, CMI exhibit somatically centered stigmata which are perceivable, unwanted, distressing, and disvalued, and characteristically give rise to a need for corrective action (Fabrega, 1974, 1975). They tend to exemplify authentic handicaps, are difficult to feign, and provide few opportunities for free rides (Zahavi & Zahavi, 1997). This biopsychosocial signature explains much of the evolutionary backdrop and significance of CMI compared to CPI. When chimpanzees (our closest extant relatives) exhibit CMI (e.g., telling signs of physiological impairment, physical lesions, weakness and suffering, impaired physical capacity) they elicit attention, interest, supportive responses, and sometimes even direct care (Goodall, 1986; Boesch, 2009, 2012; see Ritchie & Fragaszy, 1988; Fragaszy & Simpson, 2011 for monkeys; Fabrega, 2002, 2006b, 2011, 2013). It is plausible to contend that among early *Homo sapiens*, roughly modeled by anthropological studies involving contemporary hunters and gatherers, behaviors surrounding CMI conformed to and expanded upon the preceding

primate backdrop: they elicited compassion, gave rise to a need for corrective action, associated with roles of sickness and healing, and had social consequences which extended beyond the envelope of genetic relatives (Fabrega, 1974, 1997, 2011, 2013).

Factors Shaping Biopsychosocial Character of Medical Disorders

During human prehistory, the medical ecology and epidemiology of CPI and the form and effects of their morbidity were differently constituted compared to CMI (Cohen, 1989). Among nomadic hunters and gatherers as well as settled agricultural groups, occurrences of CMI tend to bring together or unite family and group. This occurs also in complex modern societies. Evolutionary developments conducive to understanding and coping with CMI either fed on or helped cause major transformations involving social cognitive capabilities and shared problem-solving routines in the genus *Homo* (Fabrega, 2013; Tomasello, 1999, 2008). Such behavioral resources and capabilities and related cultural patterns (e.g., social values, myths, existential and metaphysical beliefs), together with obvious socio-emotional consequences tied to behavioral play of CMI, were in part responsible for the biological and cultural selection of traits embodied in naturalistic, practical, and meaning-based social ensembles (i.e., systems of [ethno] medicine; Fabrega, 1975, 1976a, 1976b). The precursors of such approaches to sickness and healing would have played a significant role in enabling hominins to transcend the biological problem of altruism (Fabrega, 1997, n.d.-a, n.d.-b, n.d.-c, n.d.-d).

In contrast to CMI, CPI rarely exhibit clear and tangible markers of somatic pathology and physiological breakdown. In general, when viewed from within social behavioral spaces, manifestations of CPI embody psychological fractiousness and social conflicts configured through emotional and interpersonal relationships. Their manifestations alienate, divide, and problematize social conventions and sometimes are alarming and frightening (Fabrega, 2002, 2009, 2013). Although some CPI may elicit compassion, many do not and are socially distancing and costly to the affected individual as well as the social group or community. Compared to CMI, CPI do not invariably unite and tie members of a group together (e.g., integrate them through shared rituals—Fabrega, 1974, 1997, 2013), but instead often contribute to social separation and segregation and sometimes fractionate and break apart groups (fission). Socially divisive, fractious emotions, beliefs, and actions integral to some CPI derive from and impact heavily on preexisting (social, sexual) tensions and rivalries posing a threat to the often tenuous social and ecological grip that a group has on its environment. It is plausible that salient, visible parameters of CPI were evident since *Homo erectus* approximately one million years ago (see Fabrega, 2006b and chapter on Neanderthal CPI in Fabrega, 2013).

Evolutionary Imperatives Shaped Social Environments

Among peoples of small-scale societies, egalitarianism is a distinctive social feature and an "egalitarian ethos" is said to be responsible for smoothing, controlling, or forcing social conformity (Boehm, 1999; Fabrega, 2013). A strong egalitarian ethos and group commitment maintains integration and social conventions and plays a role in curbing behaviors that are disruptive of social order. During human biological and early cultural evolution, manifestations of CPI would arguably have played out in and been constrained by behavioral conventions which promoted egalitarian syndromes of behavior. However, during evolution (as in present hunters and gatherers), the divisiveness and fractiousness of some CPI would have cracked through the constraints of egalitarianism, leading to the breakup of social groups. They may even have played a significant role in emergence of fission-fusion patterns of group composition (Stevens & Price, 2000).

In agricultural settings, on the other hand, the signs and symptoms of CPI paint a complementary although somewhat different picture. Manifestations of CPI would have more patently fed on, impacted on, and intensified the tensions produced by competition, proximity, and lessened opportunities of leaving the fixed abodes corresponding to sedentary social life. In such settings, if for no other reason than for their persistence, CPI would have incurred higher levels of morbidity than in smaller scale societies because they were mired and festered in the tense fixity of preexistent social arrangements and intragroup dependence and connectedness.

From a population genetics and evolutionary biology standpoint, the prevalence and fitness costs of CPI compared to CMI may differ as a consequence of factors that influence how such conditions play out in relation to social ecology and related cultural circumstances. For both types of medical conditions, environmental hardships and ecological stressors would have compromised fitness: signs and symptoms of medical disorders would have embodied and highlighted vulnerability to organic, somatic disturbances (Engel's "bio" part of the biopsychosocial or pathophysiology). However, the social demographic and population genetic impact of CPI (as per fitness) is far more complex. The costs of biological organic compared to social psychosocial components and related cultural psychological effects of CPI compared to CMI (e.g., prevalence, manifestations, behavioral visibility and salience, social impact) are not only complex but also complicated.

Some contend that at the level of population the pathological nature of potentially harmful alleles of CPI is "neutralized" or behaviorally moderated if not concealed by characteristics of social, cultural environments and for this reason not significantly fitness consequential. In other words, if in ancestral environments and for social cultural reasons the alleged "pathology" of CPI alleles was not salient (or even manifest), then such alleles are not necessarily deleterious in a universal sense. The claim is that in modern compared to historically ancient societies the fitness costs of CPI would have differed significantly. Such factors as selective salience versus neutrality of CPI manifestations, on the one hand, and of environmental time lags responsible for pathogenicity, visibility, and social effects, on the other hand,

would make manifestations of the corresponding CPI alleles in modern environments evolutionarily anomalous and socially costly and consequential (a so-called environmental mismatch between impact and sheer visibility of signs of CPI and prevailing social cultural patterns and beliefs; Fabrega (1993, 2006a, 2013)).

Instead, it is plausible to contend that ancestral and indeed any group's environment is materially and socioculturally instrumental in the production of its special varieties of CPI (as well as CMI) but that the special, distinctive manifestations of varieties of CPI are responsible for their social causes and effects, and that their evolutionary implications (or "signatures") differ from CMI in social and biologically consequential ways (Fabrega, 2013). This interpretive framework as per CPI is contestable (Keller & Miller, 2006), but its supporting arguments ignore data and information involving the medical anthropology of CPI, essentially skimming over cultural psychological and biological relevance of causes, manifestations, and social behavioral consequences of CPI compared to CMI (i.e., encompassing their distinctive ontology and epistemology). It is such matters that are the focus of this chapter.

To contextualize the argument more vividly, consider that in the modern era, the potential perniciousness of environmental influences on the prevalence, visibility, and determination of "pathology" is mirrored in CPI ascribed to runaway slaves in the American South during the nineteenth century and CPI as per schizophrenias ascribed to Soviet Union dissidents during the twentieth century. Departures from the logic of objectivity and impersonality germane to evolutionary science compared to the value-laden and frequently but not always politically perverse ramifications of social cultural biases and labels constitute a signal property of the way an evolutionary dialectic can play out with respect to CPI compared to CMI (Fabrega, 1987, 2009, 2011, 2013). It shows the potentially darker and politically harmful consequences of ontology and epistemology foundations of CPI, although clear instances of psychotic conditions frequently have been socially masked and even valorized in alternative cultural settings (Fabrega, 2009).

Social cultural consequences of CPI in local communities and settings are complex and difficult to integrate with a population genetics vantage point. It is possible that adversity and salience compared to appropriation and neutrality devolve from the character and severity of CPI alleles and their manifestations, and the contexts in which they occur. Differences in social physiology and cultural psychology of ancestral populations can be presumed to have shaped the biopsychosocial matrix responsible for CPI in ways different from CMI (Fabrega, 1972, 1974, 1976b, 1987, 1997). In small-scale societies, constraints of egalitarianism can blunt and neutralize, and in settled agricultural societies, myths and ritual, extended families, and socially incorporative institutions do some of the neutralizing, yet in both settings morbid and fitness costs are prominent (Keller & Miller, 2006). The bottom line is that whether viewed from the standpoint of actual local social behavioral spaces or from the vantage point of populations, the nature and impact of CPI produce effects that are complex, overlapping, and difficult to set apart and predict. However, their differences compared to CMI stand out and this generalization is relevant to the argument presented here, namely, that there are differences in the evolutionary

signatures and implications of CMI compared to CPI (Fabrega, 1993, 1997, 2002, 2006a, 2009, 2013).

Evolutionary Imperatives and Necessities Shaped the Internal Environments of the Genus Homo

In general, the interplay of generic evolutionary imperatives and necessities considered in relation to features of environment (i.e., evolutionary dialectic) has a long-lasting and changing effect on human adaptation throughout lifetimes and involves diverse levels of human functioning. This is reflected in the dicta of "life history theory" or LHT (Charnov, 1993; Stearns, 1992), which addresses the allocation of resources, effort, energy, and time that the organism devotes to somatic growth and reproduction. LHT and the behavioral strategies linked to them are especially relevant in evolutionarily situating and contextualizing CPI.

In human populations, life history activities consist of repair of body, reproduction, parenting, care of offspring, and transfers of support and resources from older to younger relatives (Kaplan & Gangestad, 2005). In animal populations, and in humans living in different conditions and settings, LHT helps to explain basic social biology and social organization features of populations: for example, age at sexual maturity, gestation period, birth weight, litter size, postnatal patterns of growth, longevity, and length of juvenile dependency (Stearns, 1992). Natural selection is responsible for designing life history traits and activities concerning the complex optimization problem of how and when to allocate resources to gain maximum reproductive success. A defining characteristic of LHT is that its mechanisms, functions, and schedules are *developmentally plastic* and a product of biological evolution (Gluckman et al., 2011; West-Eberhard, 2003).

Fundamental tenets of LHT include the following. Organisms differ as per genotypes and phenotypes, and the nature of their environments and ecologies differ. Environmental resources are finite and they (i.e., ecologies, habitats, and social and cultural circumstances) present a different mix of opportunities and potential hazards. Early in development and as a consequence of sampling information about prevailing biologically important markers predictive of harshness or support as per future circumstances including fitness imperative in particular, organisms fashion life history strategies which are afforded by their genotype and its fit to the social ecology into which it was born (Odling-Smee, Laland, & Feldman, 2003). In conforming to dictates of ecology of a species, evolved reproductive "strategies" exemplify different mix of expenditures or investments of resources of time and energy. As suggested earlier, it is the "solving of biological problems of adaptation" which influenced and sometimes determined rates of reproduction, patterns of growth, aging, mating behavior, and parental investment, referred to as life history (LH) strategies (which by no means imply conscious reflection).

Life history strategies, while adaptive in function, are important to an individual's vulnerability and health. Costs of life history strategies are exemplified in the ways in which dysregulated, disintegrated internal response systems are structured and expressed. The goal of an adaptive strategy is to transcend recurring stressors and hindrances so as to maximize fitness. This is particularly relevant for CPI, because many, if not most, CPI manifest during reproductive years (i.e., postpuberty and young to mid adulthood; Paus, Keshavan, & Giedd, 2008), and their negative effects on fitness are longer lasting and impose higher evolutionary costs than CMI, which are more prevalent during late adulthood, and while their morbid effects are distressing and significant (e.g., osteoarthritic pains, obesity, cancers, cardiovascular disease) their effects on reproduction may be less consequential than CPI.

Dysregulated internal biological processes and especially mechanisms encompassing stress response systems are major factors determining a creature's health status. The costs of dysregulation affect visceral, vegetative, somatic, and behavioral routines which give rise to signs and symptoms whether these result from outright breakdowns of equipment (i.e., diseases) or simply from compensatory reactions to suboptimal life history strategies (Nesse & Jackson, 2006; Nesse & Williams, 1994). In general, functions of visceral vegetative and stress response systems in particular are responsible for differential susceptibility to environmental circumstances and pervasive person/environment interactions resulting in high sensitivity to environmental factors during development (Belsky, Steinberg, & Draper, 1991; Boyce & Ellis, 2005; Del Giudice et al., 2011; Ellis et al., 2009, 2012).

For example, with regard to human psychological and somatic development, LHT predicts that harsh environmental conditions reduce the amount of parental investment in individual offspring (including the amount of parental emotional commitment), which in turn biases the offspring toward immediate resource extraction and opportunistic behavior, accelerates biological maturation of offspring, and fosters short-term pair bonds and risky sexual behavior (Belsky et al., 1991; but see Barbaro et al., 2017, for important qualifications). In contrast, resource abundance during ontogeny increases the willingness of parents to invest heavily in individual offspring, hence encouraging the development of mutually rewarding relationships, deferring sexual maturation, and supporting enduring intimate relationships. Importantly, either strategy can be adaptive in terms of an individual's reproductive fitness, because each reflects a flexible response to environmental contingencies, depending on the availability and predictability of resources (Belsky et al., 1991; Chisholm, 1996). Put another way, the first type of behavior resembles a strategy of increasing the quantity of offspring at the expense of quality, whereas the latter type reflects the opposite, in behavioral ecology known as "r" and "K" strategy, respectively (the terms "r" and "K" strategy are usually used for between-species comparison only; MacArthur & Wilson, 1967; Trivers, 1972).

This model of the behavioral ecology of socialization has been expanded to include the prenatal environment during which the amount of parental investment in individual offspring is contingent upon current environmental resources and unconscious extrapolation of resource availability in the postnatal period. Such factors may, in turn, accentuate or ameliorate conflict between mother and offspring (Haig, 1993;

Trivers, 1974). According to Belsky et al. (1991), high contextual stress would bias the amount of parental investment in offspring, the offspring's psychological orientation toward interpersonal relationships including choice of sexual partners, and the speed of somatic development in the direction of the former type as described above. Such individual variation in life history strategy may be reflected in personality traits or even personality disorder (Brüne, Ghiassi, & Ribbert, 2010).

Architecture and Manufacture of Health Problems and Pathologies

To understand problems tied to adaptation and medical disorders from an evolutionary standpoint it is useful to distinguish two regulatory systems and features of adaptive behavior. One involves purely somatic visceral vegetative functions and responses (e.g., blood pressure, hormone imbalances, smooth muscle-generated pain) (Damasio, 1999; Sapolsky, 2004). This system is particularly relevant to CMI and was inherited by hominins and the primate line which preceded it. The visceral vegetative apparatus exhibits mechanisms and processes involving regulation of primary or essential chemical and physiological functions. Disturbances here are produced by genetic abnormalities, infection, and physical and social stress-related breakdown of general organ systems (e.g., cardiovascular, renal, gastrointestinal, and hormonal).

The second regulatory system that is responsible for medically adaptive problems involves mental or psychological phenomena such as awareness/consciousness, thought/cognition, emotional experience, and meaning-centered interpersonal behavior. This system emerged during biological evolution of representatives of genus *Homo* (i.e., first with *Homo erectus* and later *Homo neanderthalensis*) approximately one million years ago and was followed by the advent of more complex cognitive-emotional functions and behaviors of *Homo sapiens* approximately 100,000 years ago (Fabrega, 2013). However, in conformance to Darwinian tenets involving continuity of behavior from animals to humans, it is not just plausible but reasonable to consider that the treasured crown of behavior usually tied to *Homo sapiens* alone has a natural history, which is to say that it (i.e., mental phenomena) embodies behaviors of species that preceded the genus *Homo*, a fundamental tenet of academic scholarship that is still controversial and contested.

The label "mentalization" will be used here with a slightly different meaning compared to the neuroscience literature (see, for example, Brüne, 2014a, 2014b). It encompasses diverse "mental" phenomena germane to psychological, social, and interpersonal behavior. It includes thought/cognition, language, meaning and understanding, experience, and feelings and emotions. Mental phenomena are unproblematically ascribed to *Homo sapiens*. Contemporary emphases in diverse evolutionarily inspired behavioral sciences probe and document the relevance of mentalization as a descriptor of forms of behaviors among animals, for example,

critical anthropomorphism in cognitive ethology, voluntarism and implicit cognition in comparative associative and reinforcement psychology, the hybrid field of psycholinguistics, and in the evolutionary neurobiology of consciousness and awareness of animals (work summarized and discussed in Fabrega, n.d.-a, n.d.-b, n.d.-c, n.d.-d). Activities and functions of mentalization apply to behaviors of chimpanzees and hominins and presuppose the advent of a sense of meaningfulness (semantics) and of forms of language and culture (and cultural psychology) in "evolutionary" creatures that preceded the last common ancestor of humans and apes (Fabrega, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d; Goddard et al., 2013).

To return to our present line of argument, the regulatory system responsible for mentalization enabled profound intellectual capabilities and resources that were biologically adaptive, starting with *Homo erectus*. However, it also added vulnerability to medical disturbances through what one can term the *social, cultural, and political problematization* of mental phenomena and behaviors exemplified in varieties of CPI (Fabrega, 2006b, 2011, 2013). Put baldly, in human populations generally and in modern populations especially vividly and explicitly, the second ("internal," "mental") system adds an experiential and social cognitive-emotional dimension to health problems which influences causation, manifestations, and costs of CPI in a primary and more profound way than with CMI.

Evolutionary social scientists (Belsky et al., 1991; Del Giudice et al., 2011; Ellis et al., 2009; Ellis et al., 2012; McEwen & Stellar, 1993) have proposed working models for functions of regulatory systems (e.g., adaptive calibration model, allostatic load model, differential susceptibility theory) and discussed health problems associated with constraints involving the regulation of stress. To what extent such types of problems were exhibited in earlier environments—including those of ancestral humans—compared to modern environments represents a tantalizing question about evolutionary signatures of psychiatric conditions.

The line of argument followed thus far implies that during human evolution hominins and especially representatives of genus *Homo* evolved life history strategies that influenced not just automatic nonconscious, primary neurobiological vegetative behaviors. In addition, it involved and required mentalization parameters of adaptive functions and behavioral strategies responsible for optimal solutions to social psychological and interpersonal problems which were heightened and especially consequential in face-to-face groups which, ordinarily, were under the sway and control of an egalitarian ethos (Boehm, 1999; Fabrega, 2013). Social relationships of daily life laid the groundwork for ways of relating, ways of competing, ways of gaining status and mates, as well as mate selection, partner commitment, and parenting. Viewed in behavioral context, activities involving "biosocial goals" (Gilbert, 1998) bring into play and impact on social, biological, and cultural patterns that exemplify functional domains of life history strategies in *Homo sapiens* and other highly social species leading up to hominins. During human evolution, miscalculations in choices which individuals make in organization and expression of motivational needs, social psychological dispositions, and interpersonal and reproductive behaviors (involving misalignments in set points involving goals of

related social strategies) are not only potentially divisive and fractious but also highly consequential in a social biological and evolutionary sense.

Intra- and Intersystem Connectedness and Effects

In societies of *Homo sapiens* social, psychological, and cultural behaviors are traits through which individuals pursue goals that complement otherwise nonvoluntary, more or less "automatic" behavioral patterns, timing, and set points of life history strategies. What this means is that when such strategies are perturbed, disintegrated, and/or destabilized, the individual's social effectiveness with respect to social and cultural conventions and valued goals and end points dictated by evolutionary imperatives and necessities is undermined. Adverse consequences include typical parameters of CPI, specifically, deficiencies, excesses, and ineffective and inefficient patterns of social, cognitive, and emotional behavior that conform to cultural conventions and values. In short, in the context of gene-environment correlations, disruptions in the organization and efficacy of life history strategies of genus *Homo* represent major causes not just of child and adolescent psychopathology but of CPI across all age categories (see reviews by Belsky et al., 1991; Ellis et al., 2009; Ellis et al., 2012; Del Giudice et al., 2011; Korte et al., 2005 Matthews, 2005; Low, Thurston, & Matthews, 2010).

An individual's sensitivities, resources, and capabilities for filtering information and cues prevailing in the social and physical environment are responsible for setting and changing goals and set points of optimality involving largely nonconscious, involuntary visceral vegetative aspects of life history strategies of animals. Resources of basic systems of regulation involving autonomic and hormonal processes also influence and modulate the chemistry, metabolism, and functions of the brains of animals which are responsible for life history strategies. In "higher" animal forms (mammals, primates, hominins, humans) basic visceral vegetative activities and functions come under the sway of larger and more complex neurological structures that are responsible for associative and reinforcement behaviors that correlate with implicit or tacit forms of consciousness and awareness which in turn modulate life history strategies (Fabrega, n.d.-a, n.d.-b, n.d.-c, n.d.-d). Natural selection underpins the origins of larger, more complex brains and forms of mentalization, thereby adding higher levels of (voluntary and conscious) control in the way parameters of life history are governed and implemented. Adaptations of representatives of genus Homo exemplify higher functions of mentalization that come to the fore with *Homo erectus*. The second system of internal regulation, in other words, relies on consciously articulated goals and strategies subtending interpersonal behaviors and mental phenomena more broadly (i.e., dimensions and functions of mentalization) which in representatives of genus Homo are voluntary, conscious, and deliberative.

The level of efficacy and the resilience of each of the two regulatory systems described earlier are vulnerable to perturbation, fragmentation, and/or breakdown as a consequence of physical and social environmental stressors, challenges, and

harms. Such sources of stress, physiological and immunological dysregulation, and social psychological culturally dysregulated behaviors can take place in ontogenetic time lines (i.e., lifetimes) and produce changes in set points, priorities, and goals via balances and trial-and-error learning, all of which are hallmarks of life history strategies. When this occurs, behavior is costly and produces stigmata of dysregulation (which can involve signs and symptoms an observer would qualify as medically relevant). When dysregulation and system-environment mismatch take place over evolutionary time lines, natural selection and genetic assimilation can bring about change (Jablonka & Lamb, 2005; Waddington, 1959, 1961) through modifications of innate and epigenetic responses.

During individuals' lifetimes and across generations human biological and cultural patterns undergirding adaptation have involved (i.e., caused, shaped, and been affected by) CMI as well as CPI. The latter have rested on basic perturbations of visceral somatic vegetative response systems and on cognitive, linguistic, and experiential parameters of systems responsible for mental life or mentalization (see Fabrega, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d; Goddard et al., 2013). However, and to reemphasize, CPIs are distinctively characteristic of perturbations and dysregulated cognitive emotional systems of meaning responsible for social psychological behaviors and functions keyed to regulating behavior in conformance to cultural conventions. Developmental psychopathology, not just of adolescents, but of adults as well, is a distinctive domain of general medicine that richly exemplifies the social psychological costs of dysregulated and disintegrated life history strategies of *Homo sapiens*.

For understanding the special character of CPI it is hard to overstate the importance of perturbation and disintegration of somatically ingrained and physiologically situated life history strategies and related cultural psychological motivations, dispositions, and social behaviors. Clinical features of a number of contemporary psychiatric disorders of adolescents and adults (e.g., personality disorders, conduct disorders, types of depression, anxiety, traumatic, somatoform antisocial personality) reflect fragments of dysregulated behavior associated with interactions of behavioral dispositions and environmental circumstances consistent with the adaptive calibration model of stress responsivity that was developed to explain and predict costs associated with life history strategies (Del Giudice et al., 2011). It is likely that in alternative evolutionary, ecological, and cultural settings ("evolutionary conditioned dialectic relationships") the architecture and integrative character of the biopsychosocial nexus would have shaped alternative varieties of CPI as well as CMI (Fabrega, 1974, 1975, 1997, 2002, 2009, 2013, n.d.-a, n.d.-b, n.d.-c, n.d.-d).

Explaining Distal Effects in Proximal Terms

It is well established that in modern populations and from an early age, exposure to adverse environmental conditions poses susceptibility risks for cognitive, emotional, and general health problems to individuals of all ages and especially children

and adolescents. Susceptibility to environmental hazards is mediated by neurobiological visceral somatic regulatory responses involving physiology and metabolism as per CMI, on the one hand, and by disturbances involving cognition, emotion, and related psychological behaviors (i.e., mentalization), on the other hand (Ellis et al., 2012). Visceral somatic vegetative and basic stress responses (activities of regulatory system number one) continue to operate and correlate with activities of regulatory system number two, namely, of mentalization as discussed earlier. Differences in susceptibility and health status of individuals are evolutionarily determined, ultimately a product of factors generated by natural selection. They are mediated through both regulatory systems. They are based on evolutionarily imposed constraints and developmentally mediated mechanisms which regulate how individuals adapt behaviorally from early life onwards.

In conditions of life prevalent in ancestral human settings, natural selection pressures in contexts of unpredictable, fluctuating, and potentially harsh environments (i.e., involving physical, social, and cultural ecological stress-filled circumstances) were responsible for generating alternative programs of development which balanced and optimized fitness costs and benefits across the life cycle. An evolutionary vantage point suggests that adverse as well as supportive environments have been the recurring fate of experiences throughout human evolution. Developmental programs and systems influencing neurobiological, visceral somatic or vegetative, and social psychological functions were adaptively shaped and regulated through developmental strategies of behavior which balanced benefits and costs that differed in terms of stressful and harmful consequences. Exposure to ecological parameters of physical environments interact with genotypes and phenotypes of individuals in differentially shaping not just neurobiological response sensitivities, resourcefulness, and resilience but also patterns of social and psychological maladaptive behavior and by extension types of general medical disorders.

Two contemporary theories explaining the adaptive consequences of adverse, harsh environments on general patterns of development throughout life cycle (i.e., differential susceptibility theory and biological sensitivity to context theory; Boyce et al., 1995; Boyce & Ellis, 2005; Belsky, 2012; Ellis et al., 2012) both explain harm to programs responsible for neurobiological, visceral vegetative, and social psychological responses and both explain vulnerability and negative health consequence, specifically, CMI and CPI. The greater behavioral (i.e., psychological, social, and cultural) visibility of signs and symptoms constitutive of CPI poses additional individual, familial, social, and societal costs compared to those correlated with CMI. This difference suggests that evolutionary imperatives and necessities (e.g., adaptive optimization of benefits and costs, life history theory, natural selection) are special factors conditioning CPI.

In support of the differential susceptibility theory it has been observed that gene-environment interaction with regard to psychological traits can produce diametrically contrasting and opposed phenotypes, depending on whether early environmental conditions in terms of emotional availability of caregivers are beneficial and supportive (i.e., trustful and compassionate) or adverse (i.e., harsh and rejecting). Several genes moderating responses to such factors have been identified that

predispose to CPI such as antisocial personality disorder, ADHD, or depression when meeting early adversity, whereas the very same variants are associated with lower than average risks for the same conditions when combined with optimal parenting (Belsky & Pluess, 2009; Brüne et al., 2012). Differential susceptibility has, to the best of our knowledge, not been described for any CMI.

Aside from differences between CPI and CMI seen from a LH perspective, there is evidence to suggest that modern environments impact in different ways on CPI versus CMI. To begin with, modern environments are in some ways grossly different from those of our evolutionary past. The so-called mismatch hypothesis (discussed earlier) points out that with the emergence of large, dense, sedentary, and stratified populations exemplifying modern culture and complex civilizations the human mind and body were and still are not prepared or readied for what involves unprecedented adaptive constraints and hindrances inherent in (and responsible for many of the challenges) associated with modern compared to ancestral conditions of life (Buss, 1995). These involve, for example, caloric oversupply, lack of physical activity, different selective environments created by exposure to levels and types of pathogens, and a dramatic change in social structure of societies (Barnes, 2005; Gluckman et al., 2009; Stearns & Koella, 2008; Trevathan et al., 2008). Studies governed by principles of evolutionary biology and human cognitive developmental biology have produced unprecedented advances in scientific understanding and control of many diseases.

Additional Considerations

Among the most welcomed technical achievements of modern society and medicine one can point to are the detection, technical understanding, and precise deployment of radiological methods for the examination of the body and development of anesthetizing and analgesic agents which have enabled conducting and perfecting surgical treatment of many hitherto untreatable conditions. To these developments one can add the discovery of infectious agents and parasites and the development of preventive measures of contagion and the discovery of antibiotics. These and related advances in the modern science and practice of medicine have expanded understanding of developmental biology and medical ecology factors responsible for the constitution of modern human populations which, as suggested earlier, are mirrored in modern profiles of distinctive health problems and susceptibilities which, in turn, are responsible for many diverse varieties of somatic pathology as well as their control, cure, and palliation. Complementarily, such changes in social biology and ecology of human populations have created new selective environments that have changed the genetics, developmental biology, metabolism, physiology, and immunological constitution of modern human compared to ancestral populations. The results and corresponding profiles of mechanisms and processes tied to the consequences of new dialectics of population, environment, society, culture, and disease are mirrored in the distinctively evolutionary contingencies and imperatives

discussed earlier. The effects of changes in medical ecology of health of human populations involve the complex cycles of deviation, compensation, costs, benefits, trade-offs, and adjustments in internal general bodily and brain functions discussed earlier. Together they constitute a medical inheritance of human populations reflected in physiological, molecular biological, and social psychological changes linked to constraints and costs attending human adaptive behavior, constraints and costs which are roughly subsumed under historically inherited medical categories as discussed earlier.

Put another way, unprecedented changes in the social biology of human populations and their CMI and CPI have changed the organization and structure of mechanisms and processes responsible for "modern diseases of civilization." Relaxation of natural selection pressures and changes in the mix of harmful and beneficial environmental characteristics associated with modern human populations have changed the balance responsible for protection of and susceptibility to disarticulation of internal regulatory systems which are expressed in different levels and constellations of somatic pathology. On the other hand, nowadays individuals have the opportunity to reproduce who a few generations ago were excluded from contributing to the genepool of future generations. Tragically, for example, Charles Darwin's daughter, Annie, died of scarlet fever, for which effective antibiotic treatment is now widely available. In the last couple of decades, the list of previously untreatable diseases has expanded, including type I diabetes mellitus and other endocrinological diseases, such that the number of individuals reaching the reproductive age has increased by the score. We are aware of the complex problems and limitations in terms of which these statements could be interpreted. However, while a "naturalistic fallacy" would confuse "is" with "ought," it is clear that past and present medical progress is a desideratum of interest to all.

Whether modern conditions of life have a differential influence on prevalence of CMI compared to CPI brings into focus two types of questions. These are the following: (1) Does the vulnerability to psychiatric compared to nonpsychiatric conditions differ in relation to developments surrounding the cultural ecology of modernity? (2) Leaving aside the possible influence of genetic vulnerability, is pathogenesis or mechanisms responsible for the production of CPI compared to CMI influenced in a special way by modern conditions of life? In view of earlier discussion regarding the special (i.e., ontological, epistemological, and moral) features of CPI, answers to these questions appear to be yes. This line of thinking suggests the need for continued emphasis on research initiatives in social, cultural, and evolutionary psychiatric theory and practice. Unfortunately, the relevance of evolutionary social behavioral sciences for psychiatry is all too often less emphasized in the light of psychiatry's understandable excitement associated with expansion of knowledge in molecular biology and brain sciences and technologies. One can propose that the logical and empirical ties that CPI has with social environmental influences regarding pathogenesis, content of manifestations, persistence, and general consequences support and strengthen an argument for a general evolutionary emphasis in psychiatric theory and practice. As indicated earlier, evolutionary social sciences uniquely underscore interpretations of human adaptive or nonadaptive

behavior in light of precisely those same environmental influences, specifically, the interplay of populations, society, social biology, and ecology, and the inevitable costs attending attempts to transcend natural selection. While the environment, population, and adaptation dialectic influence clinical and epidemiological dimensions of both CMI and CPI, the social psychological cost of CPI is higher for reasons discussed earlier.

Comment

For most of their evolutionary history humans have been adapted to life in small close-knit communities consisting of approximately 150 adults and children which any one individual was personally acquainted with and connected to via complex patterns of exchange (Dunbar, 2003). As indicated earlier, in contemporary urban environments, such conditions of social life are not common: people are confronted with a much larger, more diverse, and denser concentration of coresidents, not fully comparable with the more open and communitarian settings found in the social environments of the ancestral past. Similarly, establishment and maintenance of mutually shared values and goals through relationships supported by trust, all based on mutual give and take, constitute phenomena difficult to achieve in modern urban societies. Conversely, research has shown that a stable personal social network increases the likelihood of survival by 50%, independent of gender, age, and initial health status (Holt-Lunstad, Smith, & Layton, 2010). Moreover, modernity has shaped considerably the ways of upbringing, including a lack of physical proximity between mothers and infants, which measurably leaves its mark on stress physiology (Morgan, Horn, & Bergman, 2011).

These considerations underscore the point that the social biology and evolutionary nature of psychiatric conditions, which by definition are special, because of the interconnected character of their causes, mechanisms, manifestations, and natural history (Engel's biopsychosocial amalgam or holism), are importantly determined and constituted by differences in the conditions of social life of modern societies compared to the character of ancestral environments wherein representatives of genus Homo (i.e., immediate evolutionary ancestors humans) evolved and lived for hundreds of thousands if not millions of years ago.

A final, though no less important, difference of CPI compared to CMI relates to what we would like to call "tolerance of variation." With respect to blood pressure, for example, the American Heart Association recommends that the systolic blood pressure be under 140 mm Hg and under 90 mm Hg for diastolic blood pressure (American Heart Association, 2005) and blood sugar ought to be below 100 mg/dL. In other words, for somatic disorders, the medical conventions or standards concerning variation in definition of normality (compared to abnormality) have traditionally been low. Today, one observes among representatives of the medical establishment an increase in or relaxation of medical conventions involving definition of normality, such that measures, which in the past were construed as abnormal,

no longer apply. For example, with regard to anatomical structures, an increase in prevalence of "anomalous" arteries has been documented for the median artery of the forearm and the thyroidea ima branch of the aortic arch (reviewed in Rühli & Henneberg, 2013). However, one needs to keep in mind that even though criteria for hypertension, diabetes threshold, etc. seem to be based on empirical grounds, "normality" is defined by determining the skewedness of a Gaussian curve in a particular population. Put another way, what is determined as "normal" in one population is not necessarily consistent with what is found in different populations, but culturally biased (Fabrega, 1975). This becomes most evident when comparing population averages of blood pressure and body mass indices (BMI), for example, of Western populations with hunter-gatherer societies, where the mean systolic blood pressure is considerably lower than in Western samples, and the same applies for BMI (Lindeberg, 2010; Lindeberg & Lundh, 1993). Conversely, and ironically, it is taken for granted by Western medical scientists and academic scholars that some age-related changes such as the development of atherosclerosis is—to some degree—"normal," although no such evidence exists from studies in hunter-gatherer societies (Stearns & Koella, 2008).

In comparison to manifestations of CMI, during past historical eras attitudes and dispositions in Anglo European societies about manifestations of CPI (e.g., involving psychological characteristics and especially personality traits) and high level of intolerance and social censure have been prominent among gatekeepers and administrators of social institutions (one exception here is leprosy during the premodern era). Tenets of Anglo European social and moral philosophy held by politically influential experts have supported the proposition that every person constitutes a unique entity, an individual in the word's best sense. Even twins can easily be distinguished based on their personality and behavior, not necessarily their outer appearance. However, psychiatric theory, explanation, practice, and language for describing human action question and problematize traditional assumptions about individual motivations. Conventions extolling the tenets surrounding humanism, good will, social responsibility, morality and ethics, and political neutrality are challenged when motivations and goals undergirding behavior are explained as expressions of medical and personality disorders. The drift toward a medicalization if not pathological signaling of behavior of individuals has been a feature of psychiatry for a considerable period of time.

To a critical observer it appears that variation in personality and character is nowadays more acceptable and accepted as a moral imperative in the community at large. However, modern psychiatry's reliance on categorical thinking and value-laden notions respecting motivation "personality functions" sometimes amounts to a social moral condemnation of persons. What should ideally constitute a reliable and reasonably objective accounting of a person's adaptive behavior in matters involving fitness are hardly possible in modern psychiatric theory and practice since cardinal features with respect to CPI are that the latter are caused by, partake of, manifest through, and impact on motivations, understandings, and feelings inherent in behaviors of persons situated in social and cultural environments. Put in formal terms, the distinctive nature of CPI (compared to CMI) is anchored in their basic

ontology and epistemology which directly manifests in socially consequential settings and circumstances which place psychiatric explanations and claims in a contentious environment that distinguishes it from nonpsychiatric medical disciplines (Fabrega, 2006a). A fully neutral and value-free description of behavior exemplifying CPI and especially personality disorders can hardly be said to be easily attainable but it nonetheless has to be argued for in courts of law (Fabrega, 2004).

The language of descriptive psychopathology problematizes and brings into question the moral standing of the person. Psychiatry's formulations can appear to support and reinforce a societal tendency toward condemnation of medically ill persons. Here, psychiatry leaves its mark on the threshold at which personality traits are being diagnosed as beyond normal variation and as requiring rectification through treatment. Such a widening of the diagnostic frame (i.e., pathologizing of psychological variation) is exemplified in the epidemic growth of cases of childhood ADHD, the relaxation of diagnostic criteria of ADHD in adults in DSM-5, and perhaps even the invention of conditions such as childhood bipolar disorder, or the medicalization of grief akin to major depression (Frances, 2013). These examples indicate that psychiatric disorders and especially personality disorders are highly contrastive to nonpsychiatric disorders (CMI) with regard to their etiology, pathogenesis, and manifestations, including their social consequences and implications. We assert that the salience of negative social labels attached to CPI can best be appraised and appreciated by comparing them to labels and valuations of CMI, as reflected in an evolutionary perspective. The latter account takes into consideration our evolved history and environmental changes that may cause new or relax old selection pressures all of which do not bring into play negative social labels. This can only be fully appreciated in an evolutionary perspective that takes into account our evolved history and environmental changes that may cause new or relax old selection pressures.

Evolutionary Focus: A Complement to the Categorization Approach in Psychiatry

General Considerations

The clinical humanistic basis of medicine underscores the centrality of disorder of individuals that is harmful, constraining, and inimical to happiness and productivity. Framed in different arrays of symbols, such an approach is highly consistent with the cultural psychology and medical epistemology found in societies across human history (Fabrega, 1974, 1997). The cultural foundations of biomedical sciences presuppose value-laden categorization of negative ("badness") versus positive ("goodness") descriptors and of specificity versus generalization as exemplified in treatment. Such a framework for categorization of CPI was based on and began to consolidate in the ethos of late nineteenth century in Anglo European societies.

Modern psychiatry was spawned in this general medical culture (Porter, 1987; Scull, 1993). Like in the rest of medicine, a predominant focus in psychiatry is on categorization and specificity of disorders. Emphasis is placed on a disorder's pathogenesis, manifestations, diagnosis, natural history, and treatment. Such an approach, which underscores nosology, classification, and clinical description, remains predominant in psychiatry. It is mirrored in the anticipation of a new and carefully thought-out approach to diagnosis and classification.

Distinctive evolutionary features of CPI are comparatively de-emphasized in the conventional theory of clinical neuroscience psychiatry. A general point is the short shrift devoted to causal, functional, and evolutionary implications of the biopsychosocial and cultural characteristics and impairments which embody and constitute CPI and which set them apart from CMI. Many researchers of the psychiatric community have emphasized that the conceptual limitations of the way CPI are portrayed in the contemporary academic establishment of medicine. Representatives of the reigning clinical neuroscience perspective (CNS psychiatry, aptly put) have come to apply their approach in a disproportionate, essentially reductionist way when they cast aside and/or marginalize fundamental Darwinian insights and knowledge in evolutionary behavioral sciences. Put in stark terms: Neuroscience principles exercise a dominant and almost exclusivist influence in teaching psychiatric knowledge and principles in the academy and in organizing and sponsoring research involving basic theoretical and practical tenets of "CNS psychiatry." The cost of this glorification involves beclouding and marginalizing the basic (biopsychosocial) and cultural dimensions embodied in the pathology, diagnosis, and treatment of victims of psychiatric disorders.

Knowledge generated by evolutionary behavioral social and cultural sciences exemplifies the fruits of what constitute fundamental "basic sciences" of psychiatry. They have contributed a rich body of information that, depending on focus and emphasis, sometimes supports and sometimes underscores limitations of the reigning clinical neuroscience approach.

In psychiatry, emphasis on evolutionary factors is illustrated through several textbooks (Brüne, 2015; McGuire & Troisi, 1998; Stevens & Price, 2000) and chapters in handbooks and compendia of evolutionary psychology (Nesse & Lloyd, 1992; Troisi & McGuire, 2000). Study of various types of CPI have probed relationships between clinical manifestations and environmental circumstance and sharply brought into question whether symptoms should be construed as breakdown or defects of physiological mechanisms (i.e., biologically based pathology) or as responses of physiological systems under siege from intra-organismic or environmental circumstances and consequently possibly biologically adaptive (Nesse, 2000; Nesse & Berridge, 1997; Troisi, 2001).

As discussed earlier, life history theory and strategies represent a central organizing principle of evolutionary biology (Del Giudice et al., 2011). It is highly relevant to both the theory and practice of psychiatry. This is the case because life history parameters exemplify the connectedness between somatic developmental and reproductive functions, on the one hand, and descriptive biopsychosocial and culturally laden manifestations of behavior and psychopathology, on the other. For example,

many researchers have underscored the evolutionary rationale or ethic of a constellation of clinical manifestations of a particular diagnostic category (e.g., personality disorders, mood disorders, sexual disorders), seeing some of these "disorders" as contingent on evolutionarily determined sexual differences and sometimes as alternative life history strategies and hence adaptive and less as forms of "clinical psychopathology" (Brüne et al., 2010; Mealey, 1995; Nesse, 2000; Troisi, 2005).

A Prototype of Evolutionary Impressionability

One area where evolutionary factors can be expected to have inscribed differences in behavior is in relation to the biological ramifications of sexuality. Sex differences have been an important theme in evolutionary biology and anthropology and in studies of social stress in social psychology and neuroendocrinology. They figure importantly in the evolutionary anthropology and deep history of CPI (numerous chapters in Fabrega, 2013). Sex differences are responsible for alternative life history strategies and exemplify the wide-ranging biopsychosocial behavioral effects of this cardinal biological attribute (Campbell, 2006, 2008; Del Giudice et al., 2011). The influence of evolutionary factors on sex differences in behavior involves subsistence (e.g., males predominantly do the hunting and females the gathering), social and mating behavior (e.g., males generally indiscriminate with multiple partners), parenting (females more protective and bonded to their offspring and children), and social status seeking (males more individualistic and socially active and competitive, females more socially affiliative and connected to group relations and support building). The importance of age in adaptive contribution to support and caring of younger generations, a major factor thought to have contributed to the evolution of unique life history milestones of *Homo sapiens* (Gurven, 2012), is generally construed as involving influence of postmenopausal role of females in transfer of social capital to female offspring and grandchildren (Kaplan & Gangestad, 2005).

Female responsiveness to social circumstances represents a central focus in social and evolutionary psychology (Campbell, 2006, 2008) and has commanded the interest of most evolutionary psychiatrists (Brüne, 2015; Nesse, 2000; Troisi, 2005). Studies of responses to stress indicate that women are more physiologically reactive (i.e., involving cortisol response) to social rejection challenges (Stroud, Salovey, & Epel, 2002), and sex-specific effects of glucocorticoid responses indicate that females are low cortisol responders yet display higher levels of social cognition and apparent emotional reading (Smeets, Dziobek, & Wolf, 2009). Cortisol differences in female stress responses are variable although a critical review of this literature (Kudielka & Kirschbaum, 2005) concluded that "the overall picture seems to indicate that adult men respond to psychological stress with greater increases in cortisol compared to women" (Kudielka & Kirschbaum, 2005, p. 125).

Sex differences in stress response patterns have been equated with possible health consequences and medical conditions (e.g., Lundberg, 2005; Wang et al.,

2007). It is generally conceded that males are more susceptible to infectious disease, hypertension, cardiovascular disease, aggressive behavior and problems linked to aggression, and abuse of drugs and alcohol whereas females exhibit more autoimmune diseases, chronic pain, and depression and anxiety. Wang et al. (2007) compared the regional cerebral blood flow (CBF) responses to psychological stress with males showing CBF increases in the right prefrontal cortex and CBF reduction of left orbitofrontal regions whereas females exhibited primary activation of the limbic system (i.e., ventral striatum, putamen, insula, and cingulate cortex). Men's stress responses in prefrontal cortex were associated with physiological measures (i.e., salivary cortisol) whereas female activation showed lower correlations. "Conjunction analyses indicated only a small degree of overlap between the stress networks" (Wang et al., 2007, p. 227). This study was seen as a first and important step in discovering neurobiological factors responsible for contrasting health consequences of psychosocial stress in males and females. In general, this brief review provides support for the "tend and befriend" biobehavioral response pattern to stress said to exemplify women compared to the "flight-or-fight response" of men, a difference postulated by Taylor et al. (2000).

The polyvagal theory of Porges (2011) offers a comprehensive and integrated perspective on social behavior of humans which is deeply imbued with phylogenetic and related evolutionary behavioral considerations and which is relevant to understanding psychopathology differences in females compared to males. His theory is complex and complicated, and weaves together findings in neural sciences, evolutionary biology, and social science studies involving social communication, social "neuroperception" of the environment, and social engagement through relations of trust and protection. It acknowledges the important and distinctive role of females in matters involving social support, social relatedness, and coping through interpersonal relationships. Porges links neuroanatomical and neurophysiological factors involving interrelationships between cranial nerves V, VII, IX, X, and XI (e.g., controlling head and face gesturing, social communication, vocal and voice quality, face-to-face and social communicative engagement) with efferent and afferent nerve influences involving different fibers of the vagus nerve, particularly in controlling and dampening heart rate and contributing to respiratory sinus arrhythmia. Admittedly, the central organization of the autonomic system is very complex and remains to be worked out (see chapters in Llewellyn-Smith & Verberne, 2011). The scope and complexity of the neurological, neurophysiological, neuroendocrine, social behavioral, and evolutionary factors brought together in Porges' theory make it difficult to unqualifiedly support or reject the polyvalent and somewhat inconsistent or at least paradoxical functions of the vagus nerve on parameters of behaviors that incorporate cardiac functions and aspects that connect neural activity of cranial nerves to social relations. His theory is unifying and provocative and generally supports the emphasis of sexual differences in response to social and psychological stress reviewed earlier, and furthermore has been used in empirical theories of psychopathology.

With regard to clinical considerations it is important to emphasize that females, compared to males, are about twice as likely as men to experience a depression

during their lifetime (Cyranowski, Frank, Young, & Shear, 2000). Such vulnerability toward depression emerges at the time of puberty. Proneness toward early depression is a vulnerability that persists throughout women's reproductive lives and is generally accompanied by higher preexisting anxiety symptoms. Moreover, depression is a CPI which is particularly associated with disturbances involving social attachments, higher levels of social distress, and dysregulation of the hypothalamic-pituitary adrenal stress regulation mechanisms. It would be surprising if evolutionary factors did not represent a major conditioning influence in the high prevalence of anxiety and depression disorders among females and on their clinical significance.

Cyranowski, Hofkens, Swartz, Salomon, and Gianaros (2011) have studied characteristics of female anxious depression and reported common patterns of emotional, social, and cardiac hemodynamic dysregulation. They suggest that this vulnerability may be associated with impaired vagal control mechanisms as propounded by Porges' (2011) polyvagal theory. Vagus nerve activation promotes flexible adaptation to changing environmental demands. It rapidly shifts autonomic nervous system activity in response to environmental cues, which elicit relaxation and social affiliation (e.g., relative parasympathetic dominance via the vagal brake) versus fight-or-flight responses to environmental threats (e.g., relative sympathetic dominance via vagal withdrawal). Cyranowski et al. (2011) surmise that anxiety depression syndromes in females may be an outcome of impaired ability to induce cardiac vagal control during daily social interactions featuring diminished social interpersonal support among vulnerable females.

Cyranowski et al. (2008) contend that postpubertal females' sensitivity to interpersonal life stress may partly be influenced by the neurohormone oxytocin. This represents a factor stressed by Porges in his polyvagal theory. It is also consistent with the proposition of Taylor et al. (2000) about distinctive features of female neuroendocrinology, psychology, and behavior and of what amounts to a female alternative life history strategy compared to males. As mentioned earlier, Taylor et al. (2000) emphasize that females exemplify a built-in "tend-and-befriend" response, in other words, a biopsychosocial response pattern sculpted in the ancestral environments of females which contrasts with the male "fight-or-flight" response. Cyranowski et al. suggest that elevated peripheral oxytocin may serve as a marker of vulnerability of social separation and/or distress. Availability of oxytocin may thus ameliorate female affiliative behaviors under conditions of stress. In support of an evolutionary explanation involving such a response Cyranowski et al. cite the work of Grippo et al. (2007) involving female prairie voles exposed to extended periods of social isolation who display high levels plasma oxytocin and higher amounts of oxytocin-immunoreactive cells in hypothalamic nuclei. The same prairie voles displayed a depression-like behavioral response, which featured an anhedonic decrease in sucrose intake and sucrose preference (Grippo et al., 2007).

Heightened sensitivity to perturbation of interpersonal relationships and impaired vagal tone and dysregulation of oxytocin response in females exhibiting clinically significant anxiety and depression symptoms represent neurobehavioral underpinnings of evolutionary signatures of a subclass of female CPI. They implicate the

workings and strains imposed on life history strategies in modern environments compared to ancestral ones. Modern environments make women especially vulnerable for developing cardiac sensitivity reactions, sensitivity toward social relationship deficits, and signs and symptoms of anxiety and depression. Ancestral environments prepared females to rely on close supportive social interpersonal relationships, which prove difficult to attain in impersonal modern environments. Thus absence of close social relationships among women may constitute a risk factor for depression. Females with prior histories of social traumas were especially vulnerable to experience interpersonal stress in the absence of social supportive relationships and it is likely that mediation of perturbation of life history strategies may have played a role.

Such distinctions involving the social biology and psychology of females may have important theoretical implications. The plight of females exhibiting anxious depressions reinforces a possible evolutionary dictum that during human biological evolution reproduction and parenting responsibilities placed women in situations which required integrated social relations with group mates and sharing of responsibilities in pursuit of group support and protection. Social emotional mutualistic activities involved subsistence, child care, and protection of predator attack through emphasis on group cooperative relationships. Such a dictum also reinforces the theoretical and practical implications of an evolutionary stance respecting CPI compared to CMI. Evolutionary factors which are responsible for human vulnerability toward some CPI may give rise to clinical manifestations that exemplify and in a sense communicate (symptomatically) the significance of such factors in the biopsychosocial economy of health of an individual. This tenet supports evolutionary foundations of many CPI and points to appropriate uses that can be made of evolutionary theory in psychotherapeutic interventions based on implications of the functional significance of symptoms of psychopathology. This formulation is consistent with and reinforces a logical tie among features of CPI which are not exhibited in CMI, a contention referred to as a "feature situation-symptom complex" as described by Keller and Nesse (2006).

Conclusions

Conditions of modern life contribute in special ways to the development and manifestations of psychiatric disorders which are ontologically and epistemologically distinctive among nonpsychiatric medical disorders. When an evolutionary vantage point is adopted, emphasis is given to complex causal influences impacting on the life of individuals. Conditions of contemporary social life require taking into consideration factors outside the envelope of experience of ancestral human populations. The fluid, changing, interconnected, and complex character of factors impacting on individuals and their social relationships of modern life today constitutes contingencies, imposes necessities, and exemplifies imperatives that during their evolution representatives of *Homo sapiens* were unlikely to have been

identified and encountered. On the other hand, in contemporary modern societies they are forced to do so (Fabrega, 2002, 2013). In other words, Engel's biopsychosocial vantage point has not diminished in significance with respect to logic and science of medicine and including psychiatry.

For CPI, the aforementioned issues may lead to the following conclusions. First, modern environments increase the risk for psychiatric disorders, such that action has to be taken to reduce the impact of novel stressors on psychological and biological well-being. Second, therapy can benefit from explaining to patients the interaction of genes and environmental conditions. For many genetic variants, it is not true that being a carrier of a certain variation is merely associated with increased vulnerability, as is suggested by the prevailing diathesis-stress model. One can be much more encouraging by making clear to the patient that he or she has probably inherited an increased susceptibility for both better and worse, which is profoundly different from leaving a patient with the burden of information that he or she simply constitutes an unlucky victim which carries a "risk allele." Third, in contrast to many CMI, prevention of CPI is fraught with many more complex problems associated with the early detection of (nonspecific) precipitating conditions and identification of "toxins," including psychological adversities. Put another way, it is perhaps impossible to develop means by which primary prevention of CPI becomes a realistic goal, which further sets CPI apart from CMI. Finally, society has to decide how much tolerance it is willing to express toward people with more extreme temperament and character traits. In particular, environmental precipitants of disorders such as ADHD should carefully be studied, and the medicalization of normal psychological reactions be resisted, all this in view of the fact that diversity is the key to adaptation, not uniformity.

We contend that scientific validity of the underlying system for understanding, classifying, and treating patients exhibiting psychiatric disorders which has been the prevailing *raison d'être* of clinical neuroscience psychiatry is problematic. The promissory note of the "decade of the brain" to satisfactorily uncover and explain causal mechanisms in relation to specific psychiatric disorders does not integrate seamlessly with inherited ideas and principles about the special ontological and epistemological nature of CPI. At present there are simply no bodies of clinical neuroscience (CNS) clinically relevant data (e.g., neuropsychological, neuroimaging, genetic) that have proven specific to any particular disorder as conventionally described and categorized. Contemporary psychiatry has to acknowledge the sobering possibility that clinical neuroscience may not be capable of delivering a unifying frame of reference for psychiatric theory and practice. Integration of information from clinical neuroscience with a system of classification of psychiatric conditions based on tenets of evolutionary social and cultural behavioral sciences will require time, research, and concerted thought. At present, psychiatry has yet to fully acknowledge the power of evolutionary contributions to the understanding of CPI, at the level of diagnostic categories such as depression (e.g., Nettle, 2004), anxiety disorders (e.g., Price, 2013), or schizophrenia (e.g., Burns, 2004; Brüne, 2004), as well as at the level of more nosology-independent approaches such as the analysis of nonverbal behavior (e.g., Troisi, 1999; Geerts & Brüne, 2009), genetics (Keller & Miller, 2006;

Belsky et al., 2009), and last but not least at the therapeutic level (Glantz & Pearce, 1989; Gilbert & Bailey, 2000). The irony of the present line of reasoning is that, on the one hand, many scientific approaches to the understanding of human behavior have adopted a strong evolutionary frame of reference or vantage point (e.g., Barkow, Cosmides, & Tooby, 1992) whereas, on the other hand, distinctly genetic, physiological, and social and behavioral ecology factors are heavily reliant on general evolutionary principles to the relative neglect of social, psychological, and especially cultural factors (Stearns & Koella, 2008; Trevathan et al., 2008; Gluckman et al., 2009).

Appreciation of the importance of a comprehensive approach to CPI can function as a first step toward an individualized and evolutionarily grounded medical model of psychiatric conditions that fully acknowledges the fact that each case develops before a complex background shaped by individual differences in susceptibility (both genetic and nongenetic) and upbringing, including differences in the uterine environment that shapes psychology to a considerable degree (Brüne, 2015). Our proposal is that framing CPI in light of principles and dicta of evolutionary social sciences will go a long way toward unifying and individuating psychiatric theory and practice as a medical institution.

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Taking People as They Are: Evolutionary Psychopathology, Uncomplicated Depression, and Distinction between Normal and Disordered Sadness

Jerome C. Wakefield, Lorenzo Lorenzo-Luaces, and Jane J. Lee

Introduction

In this chapter, we review the results of a research program aimed at testing a controversial hypothesis that was generated by an evolutionary approach to psychopathology. The evolutionary approach is in conflict with the standard symptom-based approach to diagnosis utilized by the American Psychiatric Association's official *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5; American Psychiatric Association (APA), 2013). The conflict arises because many biologically designed features that are part of normal human functioning may be undesirably "symptomatic" or undesirable from our present value perspective. Such problematic normal conditions range from the pain of childbirth and the discomfort of teething to the intensity of grief, anxious vigilance regarding potential threats, and taste for fat and sugar. Evolutionarily shaped features such as these were presumably useful or neutral when they evolved but may be undesirable in our present quite

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different context. Such features, although often deserving treatment or some other social response (Cosmides & Tooby, 1999), are not necessarily medical disorders simply because they entail suffering or socially disvalued behavior. Distress and perceived need for help also occur in normal conditions in which nothing is going wrong in the organism because evolution has favored fitness over comfort and pleasure. This perspective has implications for the classification, research, and treatment of mental disorders as well as for DSM-5's nosological distinction between disorders and what DSM-5 lists as "Z Codes," that is, non-disorders about which clinicians may nonetheless be consulted.

The specific hypothesis tested by the research reported here concerns a certain subset of the conditions now classified as major depressive disorder (MDD), a prominent category of disorder in DSM-5. We label the target subset "uncomplicated depression" (Wakefield, Schmitz, First, & Horwitz, 2007) which is defined below. The hypothesis is that this uncomplicated subset of MDD is not actually a disorder, but instead describes a normal form of suffering (Wakefield, Horwitz, & Lorenzo-Luaces, 2017). This hypothesis challenges the current received view within psychiatry about its most diagnosed and paradigmatic category. Testing this hypothesis also raises tricky questions about how to empirically evaluate a conceptual hypothesis, a challenge that this research program has attempted to meet by systematically addressing objections and producing an interrelated set of findings that bolster the claim that uncomplicated depression is closer to normal distress than depressive pathology.

As far as we know, the research program reviewed here is the only research program of its kind. That is, it is the only active empirical research program that is systematically investigating whether, as is often claimed (Maj, 2011a), certain forms of depression currently classified as MDD by DSM-5 diagnostic criteria may describe normal emotional reactions that are being invalidly pathologized. If these claims are correct, then the diagnosis of MDD suffers from a potentially serious problem of "false-positive" diagnoses (i.e., misdiagnosis of non-disordered cases as being disordered). False positives can lead to unnecessary and harmful treatment choices (Andrews, Thomson, Amstadter, & Neale, 2012). Moreover, if false positives occur in research sample selection, they can bias and misdirect research on the psycho*pathology* of depressive states and render the results uninterpretable specifically with regard to depressive disorder, thus undermining efforts to find treatments for and understand the etiology of depressive disorder. Finding treatments for mental disorders is hampered—if not undermined—if one's research samples include an unknown mixture of normal sadness and pathological conditions. The critical perspective on diagnosis provided by an evolutionary perspective can guide us to more carefully formulate the distinction between normality and disorder and thus to establish a firmer foundation for psychiatric research.

Why Is Evolutionary Psychopathology Important?

It is worth taking a moment to briefly comment on the "big picture" of why it is crucial to bring an evolutionary perspective to the diagnosis of psychopathology. The obvious reason for taking an evolutionary approach to psychiatric nosology is that validly defining psychopathological categories depends on a prior understanding of what is normal. Evolutionary psychology is needed to establish what is normal, given that normality refers to human biological design (Nesse & Stein, 2012). Normal distress tends to have a prognosis that is quite different from pathology (Clayton, 1990; Wakefield & Schmitz, 2014a; Wakefield et al., 2007), and judging a condition as pathological often influences the treatment approach (Garb, 1998; Gove, 1980). Thus, from a practical and bioethical perspective, mislabeling a normal biologically designed human response as a disorder not only distorts the process of decision making about appropriate treatment but also undermines informed consent by misleading the patient in assessing the benefits versus the risks or costs of treatment. To take a common example, a normal response of intense sadness may have little chance of a recurrence once it remits, making lengthy treatment after symptoms subside unnecessary. Yet, guidelines sometimes suggest that treatment should be continued well after remission to prevent recurrences, which are expected to happen anywhere from 35 to 85% of the time (Bockting et al., 2008; Clarke, Rohde, & Lewinsohn, 1999; Eaton et al., 2008; Geddes et al., 2003; Lorenzo-Luaces, 2015; Nierenberg, Petersen, & Alpert, 2003). Consequently, the difference between a diagnosis of an intense normal response of sadness to real loss and a diagnosis of a disorder in which intense sadness is generated in a pathological way can translate into a major difference in how the patient is treated and the consequent risk of treatment side effects to the patient.

Taking an evolutionary approach to psychopathology involves reclassifying some conditions currently considered illnesses as normative experiences, and thus some individuals currently considered ill as normal individuals whose suffering is part of the normal range of human response. This approach is sometimes maligned for risking under-treatment of medical needs (Hickie, 2007). The resistance to reclassifying some conditions currently considered disorders as non-disorders goes beyond the sheer intellectual or medical and makes taking an evolutionary perspective on psychopathology more controversial than it might seem. Several interests contribute to the pressure to find high levels of illness in the population. These include aspirations by patient organizations to reduce stigma by emphasizing how common disorder is in the population, the self-interest of Big Pharma in having the broadest possible indication for use of medication, the interests of the psychiatric profession in legitimizing its treatment efforts, and the admirable desire to help suffering people despite an insurance system that demands disorder diagnosis for reimbursement.

Moreover, conceptions of normal human functioning versus disorder and what is and is not biologically designed are not topics limited in their importance to scientists and professionals. Rather, such views of human nature are also at the heart of

social and religious ideologies that form part of the cement of human communities. Views of what is natural are systematically exploited by culture as a way of rationalizing social arrangements, and such views then come to seem obvious. Challenging them can offend our basic sense of what is acceptable and cause not just disagreement but outrage. Thus, for example, efficiency and competitiveness are idealized within our culture, and negative emotions such as sadness and grief can be impediments to productivity; thus one might not be surprised to find that we are living through a period that pathologizes these emotions to an unprecedented degree (Prigerson et al., 1999; Shear, Ghesquiere, & Glickman, 2013; Wakefield, 2012, 2013a, 2013b, 2015). Similarly, in American society we see individual autonomy ("be yourself") as an ideal of social functioning, and consequently some developmental theories ensconce autonomy at the pinnacle of human development (Loevinger, 1976). Yet many cultures value devotion to the group more than they do autonomy, and high autonomy is likely a local value and not a biologically designed outcome. The challenging of socially anchored ideologies and values is what makes evolutionary psychology so interesting, and so controversial. Only a hard and honest look at ourselves through a scientific lens that confronts us with who we truly are in terms of our biologically designed evolutionary heritage can offer a corrective to potentially oppressive excesses of such sociomedical ideologies. Moreover, such knowledge can guide us in better coping with the problems raised for modern societies by our human nature designed for very different environments.

The Struggle to See Ourselves as We Are: Some Historical Asides

These issues of conflict between social idealizations and ideologies about human nature and scientific reality are not new. There is a long-term tension between who we are and what we want to believe about ourselves. Consider, for example, the following quote published in 1885 from Carl Lange, the co-originator of the classic James-Lange theory of emotion, in which he states part of his motivation in exploring the theory of emotions within an evolutionary framework:

> Kant, in a passage in his *Anthropologie*, qualifies the affections [i.e., emotions] as diseases of the mind. He considers the mind normal only as long as it is under the incontrovertible and absolute control of reason. Anything that causes it to be disturbed seems to him to be abnormal and harmful to the individual. To a more realistic school of psychology, which knows no abstract 'Ideal' man, but rather 'takes men as they are,' such a doctrine of the soul must appear strange. It must be but a meager conception of man's existence, to consider pain and pleasure, pity and anger, defiance and humility, as conditions foreign to normal life, or even as something from which one must turn away if one wishes to recognize the actual nature of man-kind. A theory which makes the power of admiring the great, of deriving pleasure from the beautiful, and of being moved by misfortune, a disease, results in a limitation of the extent of our mental life. Such a theory will consider the imperturbable arithmetic teacher, to whom every impression is merely an impulse to draw rational conclusions, as the only normal, healthy individual (Lange, 1885/1922, p. 33).

Both James and Lange were explicitly motivated in their theorizing about emotion by Charles Darwin's book, *The Expression of the Emotions in Man and Animals* (Darwin, 1872), on the evolution of the emotions. They attempted to create a more scientific theory of the emotions than the philosophers had provided. In contrast, Kant was inspired by his Enlightenment vision of enthroning the special power of rationality as a central value in life, a worthy aspiration given the irrationality of the exercise of power by Church and State in his time. However, Lange suggested that Kant, echoing claims by the Stoics, illegitimately transformed that admirable goal of being more rational into an incorrect doctrine about human normality and pathology. According to Kant's view, Lange says, intense emotions are classified as diseases of the mind and the mind is normal only as long as it is under the incontrovertible and absolute control of reason. In the face of such constricting ideologies, one must agree with Lange about the need for scientists to provide "a more realistic school of psychology which … takes [people] as they are." Such a view is provided by evolutionary psychology.

This idea that to act effectively we must know the truth of who we are goes back at least as far as the Delphic Oracle's aphorism, "know thyself." The meaning is thought by scholars to be that one must know who one really is to understand how the Oracle's advice fits into one's personal context and thus to be capable of effectively using the information imparted by the Oracle. This seems right for us as well; to use our growing technological powers effectively, we need to understand the organisms that will be wielding those powers, namely, ourselves. One presumes that this admonition applies to today's mental health professions' attempts at diagnosis of mental disorder as well; we must know who we are as human beings when normal to understand how to recognize when something has gone wrong and we are suffering from some form of pathology.

At the site of the Delphic Oracle, there are actually 146 oracular aphorisms carved in stones. Five of them in effect give you a mini-developmental stage theory by way of advice about how one should behave in each phase of life: "As a child, be well behaved; as a youth, be self-disciplined; in middle age, be just; as an old man, be sensitive; on reaching the end, be without sorrow" (one might feel that through our own cultural lens the Oracle's developmental comments are virtually inconsistent: if you actually managed to control all contrary tendencies and limit yourself to the proper behaviors as described in the first four stages, surely at the end of your life our culture suggests that you are going to regret many of the things you missed out on in life!). Our more modern psychological developmental theories are framed as scientific statements about how we are designed to develop rather than as outright moral advice. Although they are presented in the guise of scientific knowledge about human development, developmental theory is often implicitly more about what we as a society consider desirable in terms of self-control, discipline, and meeting socially valued demands than about who human beings really are. When developmental theorists suggest that a certain trait should occur by a certain age, they may be describing the development of psychological features as they are shaped by social training and thus reflect what is valued as an outcome of socialization in the present, not as features as they are biologically designed to develop.

Evolutionary psychopathology has the potential to challenge this use of theory for motives of power and social control rather than truth.

The Challenge of Valid Diagnosis

A similar process to that pointed out by Lange and exemplified in the Delphic Oracle's description of desirable developmental stages seems to be occurring with psychiatric diagnosis today. Some of the conditions being categorized as mental disorders by DSM-5 diagnostic criteria seem inappropriate because they may fall under what most people think of as normal-range human reactions. For example, this is the first time in the modern history of psychiatric diagnosis that there is a concerted effort to pathologize grief reactions to genuine loss in various forms. There is a specific new disorder added to DSM-5—often called complicated or prolonged grief disorder (although having the more elaborate name in DSM-5 of "persistent complex bereavement-related disorder")—based strictly on grief symptoms after a loss. It is true that grief can become pathological (Parkes, 1964), and physicians have long distinguished normal grief from intense grief that persisted indefinitely or had such profound intensity that it appeared pathological. However, the diagnostic criteria proposed by grief researchers seem surprisingly broad (Wakefield, 2012, 2013a, 2013b, 2015). This diagnosis remains sufficiently controversial that it was placed in DSM's list of conditions needing further study, but seems on the way to full acceptance as a diagnostic category.

Admittedly, psychiatry is particularly challenged among the medical disciplines with regard to false-positive diagnoses. This is partly because we know so little about the etiology of mental disorder and have no hard-and-fast indicators, making the distinction between disorder and non-disorder depend on indirect considerations. However, it is also because psychiatry is unique within medicine in that virtually every symptom used for diagnosis in DSM-5 can occur under some circumstances as a normal-range response. Moreover, mental reactions are designed to be highly contextually dependent (e.g., anxiety is designed to occur in response to perceived danger or threat, not randomly as in panic disorder; Wakefield & First, 2012). Thus, attempting to diagnose on the basis of symptoms without careful consideration of context—as many DSM symptom-based diagnostic criteria sets require—makes achieving validity extremely challenging. Symptoms that might indicate abnormality some of the time but are also normal in other contexts might be diagnosed as disordered all the time based on the symptoms alone if the DSM criteria fail to take context into account.

Certainly, psychiatric diagnosis is expanding rapidly in ways not seen before. In many cases, these expansions of the domain of pathology seem—as Lange put it—"strange." Indeed, the issue of "false-positive" diagnosis due to the expansion of psychiatric categories into areas of normality was a major issue in the debates over the recent revision of DSM that led to DSM-5, in terms of both introducing new categories and broadening diagnostic criteria for existing categories. The DSM-5

work groups were very responsive to clinicians' need to expand the diagnostic labeling of individuals coming for consultations but relatively tone deaf to issues of mistakenly labeling normal distress as mental disorder. With psychiatry strongly embracing a brain-disease approach to mental disorder, critics feared that the expansion of diagnostic categories portended an even more overly medicated society than we already have.

Allen Frances, who had been Task Force Chair in the development of DSM-IV (APA, 1994), emerged as a vehement critic of DSM-5, arguing that DSM-5's changes would unleash a tidal wave of false-positive diagnoses, transforming many normal conditions into bogus disorders: "Many millions of people with normal grief, gluttony, distractibility, worries, reactions to stress, the temper tantrums of childhood, the forgetting of old age, and 'behavioral addictions' will soon be mislabeled as psychiatrically sick" (Frances, 2012, para. 22). One can almost hear Frances pleading for psychiatry not to idealize human normality but to "take people as they are" in formulating diagnostic criteria.

The Special Challenge of Validly Diagnosing Depressive Disorder

The most controversial area in the debates over potential DSM-5 expansion of false-positive diagnoses concerned changes to the diagnosis of major depression. DSM-5 failed to examine the proper threshold of this burgeoning diagnosis and in fact made a change—eliminating the "bereavement exclusion" (see below)—that further expanded the diagnosis by eliminating one consideration of context that formerly limited the diagnosis of depression. The overall challenge of diagnosing whether a given grief reaction is normal or disordered poses the central question that this chapter addresses: When do depressive symptoms represent a normal intense sadness response versus something having gone *wrong* with the functioning of the emotions?

Depressive disorder was much more narrowly diagnosed from Hippocrates until the DSM-III in 1980 (Horwitz, Wakefield, & Lorenzo-Luaces, 2016; Horwitz & Wakefield, 2007), when psychiatry adopted primarily symptomatic criteria for diagnosis. The question most physicians throughout history have asked when confronted with a patient who presents as depressed is some variation of this: Is anything happening in the patient's life that might explain these feelings without postulating a mental disorder? The move towards a symptom-based classification largely set aside the traditional role of context.

The normality of intense sadness as a biologically designed human response to loss is supported by the cross-cultural, developmental/infant, and comparative (across-species) literatures, among others (Horwitz & Wakefield, 2007). Although to some extent it remains an evolutionary mystery as to why human beings are designed with such painful and often debilitating emotions, there are many theories

of how these reactions might have been adaptive under the circumstances in which human beings evolved. The trigger for these studies is the clear evidence that these emotions are biologically designed species-typical reactions, and therefore that it is a mistake to classify them in a blanket way by symptoms as disorders. Because normal sadness reactions can be intense and painful, it is a particular challenge to distinguish normal from disordered sadness responses.

The idea that depression can be over-diagnosed when people react intensely to sad life circumstances was already well recognized in antiquity. Ancient physicians such as Galen presented cautionary case histories of individuals thought to have melancholia (the ancient label for depressive disorder, meaning "black bile disease" based on the humoral theory popular at the time) but in fact suffering from various forms of reaction to life's vicissitudes. Perhaps the most famous such tale of a false-positive diagnosis of melancholia is the story of Antiochus and Stratonice. Antiochus was the son of the Syrian King Seleucus, an aging monarch who lost his wife and remarried through an arranged marriage to a beautiful and very young woman, Stratonice. Antiochus subsequently fell ill, took to his bed, and was thought to be suffering from melancholia. In desperation, the king sent for a famous physician from Alexandria, Erasistratus, to diagnose Antiochus. Erasistratus was known as the first physician to use pulse rates as a diagnostic technique. Erasistratus soon noticed that whenever Stratonice came to pay Antiochus a visit, Antiochus's pulse would begin to race. The physician wisely diagnosed Antiochus' condition as love sickness and not melancholia. Galen reports that when the young man's ardor was returned by Stratonice, his symptoms quickly disappeared. The king subsequently was convinced to divorce Stratonice so that she and Antiochus could wed, and gave them a third of his kingdom as a wedding gift. The story illustrated to early physicians that not all intense sadness is MDD and that correct diagnosis of non-MDD sadness can lead to radically different ideas about how to rapidly "treat" depressive symptoms.

How Do You Tell a Disorder from a Non-disorder? The Harmful Dysfunction Evolution-Based Analysis of the Concept of Medical Disorder

This chapter is largely about distinguishing disorder from non-disorder. We approach all such questions about normality versus medical disorder within the framework of Wakefield's "harmful dysfunction analysis" of the concept of medical disorder (Wakefield, 1992a, 1992b, 1993, 1999a, 1999b, 2000a, 2000b, 2007; Wakefield & First, 2003). According to this analysis, a medical disorder requires both dysfunction—the failure of some mechanism to perform a function that it was biologically designed to perform (where "biological design" is understood in terms of the natural selection of evolutionary functions)—and harm, where the dysfunction causes harm to the individual *as judged by social values*. The dysfunction component, construed as a failure of biological design, is taken to be a purely factual criterion

(Wakefield, 1995, 2003). However, overall the harmful dysfunction analysis is a "hybrid" fact/value view due to the insistence that a value component is also involved (Wakefield, 1992a).

DSM Diagnostic Criteria for Major Depressive Disorder

In 1980, in order to address a host of problems besetting psychiatry, the third edition of DSM created a diagnostic revolution by formulating operationalized symptom-based criteria for all the major mental disorders. Diagnoses based on brief descriptive statements, as they were represented in prior editions of the DSM, were known to be highly unreliable with different mental health professionals coming to radically different conclusions based on the same evidence. There was a growing number of schools of psychiatric thought—cognitive, behavioral, psychoanalytic, and biological—that each used its own approaches to defining disorder for research studies. Because of this, studies could not be compared and the small but growing research was not cumulative. The antipsychiatry movement used this as ammunition to criticize psychiatry for being a bogus medical field that was really about the social control of undesirable behavior (Horwitz, 2002). Adding to this, the traditional usage of psychoanalytic concepts in diagnostic descriptions was bothersome to those in other schools of thought. All of these problems were addressed at once in the use of more reliable and theoretically neutral symptom-based diagnostic criteria purged of psychoanalytic assumptions and providing a level conceptual playing field for all schools of thought to compete with cumulative research based on similarly defined samples. Since it was introduced by DSM-III in 1980, the definition of major depression has stayed fairly constant throughout the iterations of the DSM.

The current definition of MDD in the fifth edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; APA, 2013), which distinguishes depressive disorder from normal sadness, requires the presence of symptoms from at least five out of nine depressive symptom groups for a minimum duration of 2 weeks of symptoms. The symptom groups are depressed mood or sadness, diminished interest or pleasure in usual activities, sleep difficulties, changes in weight or eating, psychomotor retardation or agitation, fatigue, feelings of worthlessness or guilt, diminished ability to concentrate, and thoughts about death or suicidal ideation. The criteria also require that the symptoms not be better accounted for by the physiological effects of a substance or general medication condition, or by uncomplicated bereavement.

The precursors of these symptom-based criteria for MDD were originally formulated in research studies to distinguish depression from other medical disorders among hospitalized, clearly ill populations for research purposes (Horwitz et al., 2016; Kendler, Muñoz, & Murphy, 2014; Wakefield, Schmitz, & Baer, 2010). The evidence for their validity was thin, and as psychiatry moved from the asylum where severe cases were almost always clearly disordered to the community with its large

reservoir of emotional suffering that can often look similar to disorder, the criteria were never adequately revalidated to distinguish normal anguish from disorder. An extremely heterogeneous range of conditions can meet these criteria, from mild one-episode cases to chronic, severe, or recurrent episodes (see below; Lorenzo-Luaces, 2015; Monroe & Harkness, 2011, 2012; Zeiss & Lewinsohn, 2000). It has been hypothesized that the current DSM-defined diagnostic criteria encompass many "false-positive" misdiagnoses of normal sadness that result in inflated prevalence rates that "strain credulity" and "undermine the [diagnostic] model's credibility" (Parker, 2008, p. 842; see also Friedman, 2012; Parker, 2007).

Establishing any kind of a diagnostic boundary separating MDD from normal sadness is a challenging enterprise. Although it is possible to produce a boundary that can be reliably made, the validity of this boundary is suspect because normal reactions to stress exhibit many of the same symptoms (e.g., sad mood) as depressive disorders (Horwitz & Wakefield, 2007). Crystallizing the need for a scientific approach to this question, Maj (2011a) challenged researchers to undertake the difficult **t**ask of answering the question "When does depression become a mental disorder?" and urged an empirical approach to validating hypotheses about an adequate threshold for disorder (Maj, 2011b).

The publication of DSM-5 provides an additional reason for investigating the threshold for MDD. Indeed, it is noted in the DSM that simply satisfying the diagnostic criteria does not indicate the presence of a major depressive episode. Responding to criticism that the criteria are overly expansive and that it was a mistake to eliminate the "bereavement exclusion" (see below), DSM-5 did not alter its official diagnostic criteria and instead added a note to the MDD criteria set that explicitly recognizes that some conditions satisfying the symptom criteria are in fact not depressive disorders but normal and understandable reactions to stressors:

> Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss (APA, 2013, p. 151).

This note makes some important points, such as that there is no specific duration that is associated with normal sadness (whereas earlier editions had included an implicit 2-month limit) and that any loss or stressor could give rise to a normal reaction that resembles in symptoms a disordered depressive episode (whereas before only uncomplicated bereavement was recognized as a circumstance in which this could happen). Rather unfortunately, however, the note leaves decisions to the clinician about whether a loss-related condition that meets the criteria for MDD is in fact disordered or not disordered. The vast majority of major depressive episodes are identified by individuals as occurring after a stressor (Wakefield et al., 2007). Thus, the lack of symptom-based criteria for making this distinction throws the diagnosis

of one of the DSM's central categories into doubt; it undermines the DSM's primary rationale of providing reliable operationalized diagnostic criteria for research as well as clinical diagnosis (Maj, 2010). Further understanding of this boundary issue is thus urgently needed, and the research program presented here is one step in trying to meet this need.

The Ubiquity and Heterogeneity of DSM-Defined Major Depression

Are the DSM criteria valid indicators of a depressive disorder? To understand one of the major reasons for suspicions that the current MDD criteria may be invalid—and thus one of the major motivations for reassessing the valid threshold for diagnosing MDD—one needs to only consider the implausibly high rates of disorder that are produced when current DSM MDD criteria are applied at the population level. Until just a few decades ago, psychiatrists were trained to believe that depression as a disorder was relatively rare and severe and that perhaps 2–3% of the population would suffer from it in a lifetime (Klein & Thase, 1997). In fact, the lifetime risk of clinical depression was considered to be so low that when the pharmaceutical manufacturer Geigy evaluated the first antidepressant, imipramine, after it was identified in the 1950s, it was concluded that there were too few potential depressed patients to justify taking the drug to market (Healy, 2008). The advent of the DSM-III symptom-based criteria for MDD changed all of this. Epidemiological studies could then be cost-efficiently conducted in large populations by simply having trained interviewers—not necessarily mental health professionals—administer structured questionnaires asking respondents to report symptoms they had experienced, and using computer programs to evaluate whether the symptoms reported satisfied criteria for disorder, with no other information needed (Horwitz & Wakefield, 2006). Using DSM criteria in such a survey, the National Comorbidity Survey (NCS; Kessler et al., 1994) reported a lifetime rate of 15% for MDD. The National Comorbidity Survey Replication (NCS-R; Kessler et al., 2003) reported a similar rate of 16%, and the National Epidemiologic Survey of Alcoholism and Related Conditions (NESARC; Grant et al., 2008) reported a lifetime rate of 13%.

The magnitude of the prevalence rates reported in these nationally representative epidemiological surveys puzzled some prominent epidemiologists, who acknowledged that normal reactions to stress or other life events might have been misclassified as depressive disorders by the DSM-based diagnostic criteria used in the studies. For example, Darrel Regier, who went on to be Vice-Chair of the DSM-5 Task Force, observed that some conditions diagnosed by DSM criteria in the community as depressive disorders may be "transient homeostatic responses to internal or external stimuli that do not represent true psychopathologic disorders" (Regier et al., 1998, p. 114). However, even more provocative findings were to come due to improvements in methodology that addressed weaknesses in these initial studies.

The initial DSM-based epidemiological surveys relied on cross-sectional assessments in which individuals were interviewed at a point in time and asked to recall whether they ever experienced the symptoms of depression in the past, to produce a lifetime estimate. The heavy reliance on memory of past symptoms suggests that the rates obtained from these studies are actually *under*estimates. This is because it is known that individuals often fail to recall symptoms of depression—especially milder depression or depression in response to adverse life events—when asked to think about them over the life course (Eaton, Neufeld, Chen, & Cai, 2000; Eaton et al., 1997). Indeed, even more severe symptoms, such as those leading to hospitalization, are often forgotten years later (Andrews, Anstey, Brodaty, Issakidis, & Luscombe, 1999). Recent longitudinal studies have corrected for this problem by following participants over time and interviewing them on multiple occasions about recent symptoms that the subjects are more likely to recall.

The results of recent longitudinal studies suggest a markedly higher MDD rate than was indicated by the cross-sectional studies. In one of the first waves of published reports of the prospectively assessed prevalence rate of MDD, Wells and Horwood (2004) reported on a representative cohort of youths born in the New Zealand city of Christchurch in 1977. In just the 7-year study period between the ages of 14 and 21 years, 37% of Wells and Horwood's sample met DSM MDD criteria at least once during the study period. Similar findings were reported by Wilhelm et al. (2006) in a cohort of students from a Sydney, Australia, university, who were interviewed every 5 years during a 25-year period. Almost half of the sample (42%) was reported as meeting MDD criteria at least once during the study period. A selected cohort of young adults from Zurich, Switzerland, who were followed from the ages of 20–50 years, reported a similarly high lifetime prevalence rate of 32.5% MDD at some point during that period (Angst et al., 2015).

Some even more methodologically rigorous longitudinal studies have now replicated these early findings. Moffitt et al. (2010) interviewed a representative cohort of individuals in Dunedin, New Zealand, at ages 18, 21, 26, and 32 years about symptoms they had experienced during the past year. In contrast to the standard estimate of 17% lifetime prevalence of MDD in New Zealand, the more careful longitudinal Dunedin study yielded roughly a 17% prevalence rate of MDD in *any one given year in which participants were interviewed immediately following that year*. The cumulative Dunedin lifetime rate of MDD over the four 1-year measurements (i.e., the percentage who satisfied DSM criteria for MDD at any one or more of the four 1-year evaluation points) was 41.4%. This rate does not include individuals who had depressive episodes only before age 18 or after age 32, or had them only during the other 10 years between the ages of 18 and 32 that were not sampled in the four 1-year evaluations. Consequently, full lifetime prevalence of MDD in Dunedin should be expected to be considerably higher than 41.4%.

Rohde, Lewinsohn, Klein, Seeley, and Gau (2013) reported on a series of prospective assessments of MDD in a US cohort of Oregon adolescents followed longitudinally from childhood to age 30 years. In this study, 51% of the total sample met DSM criteria for MDD. Even this study is subject to recall biases during the sampled 6-year intervals, and so can be expected to underestimate lifetime MDD

prevalence relative to what would be obtained from a more continuous assessment, such as with ecological momentary assessments. Additionally, the Moffitt et al. (2010) and Rohde et al. longitudinal studies, which are the most rigorous available, reported MDD prevalence up to the last assessment at age 32 or 30, respectively, not lifetime risk. There are no longitudinal data that address the lifetime prevalence of MDD across the entire lifespan. In the cross-sectional NCS-R, for example, 50% of the cases reported first onset *after* the age of 32. Extrapolating from the Moffitt et al. and Rohde et al. reports, the true lifetime DSM-defined MDD prevalence would then rise to over two-thirds of the entire population (69%). Overall, it seems that the more sophisticated the methodology, the higher the rate of MDD that emerges.

There are many within the mental health field who question whether a condition with such high prevalence is a genuine disorder or is confusing normal negative human emotions with disorder and may need an adjustment of its diagnostic threshold. Leading figures have called for empirical investigation of the proper threshold for MDD diagnosis (Maj, 2011b), but there has been little response to what amounts to a call to potentially reduce the extent of the primary domain of psychiatry.

Uncomplicated Depression and the Bereavement Exclusion

As noted, DSM-5 (APA, 2013) diagnostic criteria for major depressive disorder (MDD) require the presence for at least 2 weeks of a syndrome consisting of at least five symptoms from at least five out of nine groups of specified symptoms, one of which must be depressed mood or lack of interest or pleasure implying a depressive condition. The symptoms used to identify depressive disorders vary enormously in nature, ranging from common phenomena such as insomnia and fatigue to such extremes as marked impairment that can reach almost total immobilization, psychotic ideation, and psychomotor retardation in which there is an observable slowing down of thought and action. The criteria were originally formulated to distinguish depressive disorders among hospitalized patients as distinct from physical conditions or schizophrenia, so this range of symptoms could be useful in making those distinctions. However, the DSM-5 criteria for diagnosis of depression treats all nine symptom groups as equal, drawing no qualitative distinctions among them for the purposes of the count that determines whether the five-symptom threshold is reached for diagnosis with MDD. The problem is that as psychiatry has emerged into the community, the challenge of distinguishing normal sadness from depressive disorder is not effectively addressed by these same criteria because many of the less severe symptoms that might distinguish depression from a physical disorder or from schizophrenia—for example, insomnia or fatigue—do not effectively distinguish depressive disorder from intense normal sadness.

The distinction examined in the research reported here between “uncomplicated” and “complicated” depression is an attempt to refine the MDD criteria by identifying likely non-disorders representing normal-range human emotional responses to loss and stress—the “uncomplicated” group—that are now mistakenly subsumed by the

DSM-5 criteria under MDD. The uncomplicated/complicated depression distinction is based on a division of the DSM-5 depressive symptoms into two groups, uncomplicated versus complicated symptoms. The complicated symptoms are the following six pathosuggestive features uncommon in normal distress responses: psychomotor retardation, suicidal ideation, psychotic ideation, sense of worthlessness, episode duration greater than 6 months, and marked functional impairment. Uncomplicated depression includes depressive moods lasting 2 weeks or longer that include none of the six complicated symptoms, and thus include only other "uncomplicated symptoms" that are frequently present in normal distress reactions to loss and stress, such as sadness, lessened appetite, insomnia, difficulty concentrating, and fatigue. Both complicated and uncomplicated depressions as discussed here satisfy DSM-5 criteria for MDD in requiring that the individual experience a total of at least five of the nine DSM-5 symptoms for at least 2 weeks, and thus under current criteria are diagnosed as MDD. They are distinguished by the nature of the specific symptoms that they include. The central hypothesis driving this research program is that uncomplicated depression is not in fact a mental disorder but is normal intense sadness that has mistakenly been included as a disorder within the DSM-5 category.

The selection of complicated symptoms derives not from a quantitative algorithm but from a mix of clinical, theoretical, and research considerations. Uncomplicated depression is in fact an extension and modification of the "bereavement exclusion" (described below) that was part of the DSM-IV criteria for major depression but was eliminated in DSM-5. Whereas the bereavement exclusion excluded from MDD diagnosis normal depressions occurring during grief after losing a loved one, the concept of uncomplicated depression generalizes the bereavement exclusion to include stressors other than grief. The use of 6 months as the threshold between uncomplicated and complicated cases is supported by extensive empirical analysis (see below). Informal attempts to test possible additional symptoms, such as guilt or psychomotor agitation, as potential complicated symptoms have thus far not yielded changes in validity.

The distinction between complicated and uncomplicated symptoms originally emerged from studies of the symptoms that differentiate normal intense sadness during grief from pathological depression (Clayton, 1990; Clayton, Desmarais, & Winokur, 1968). An initial longitudinal investigation by Clayton et al. (1968) examined depressive symptoms experienced during normal grief by a nonclinical sample of individuals who had recently lost a loved one. During the first week after their loss, most participants reported symptoms such as depressed mood (87%), sleep disturbances (85%), and crying spells (79%), and about half reported each of diminished interest in usual activities, difficulty concentrating, and lessened appetite. Such episodes remitted on their own within a brief period and did not cause the kind of marked impairment that frequently leads to psychiatric consultation and care (Clayton, Halikas, & Maurice, 1971; Clayton et al., 1968), and thus were consistent with cultural and societal expectations. In contrast, psychotic ideation, self-condemnation and feelings of worthlessness, suicidal ideation, and psychomotor retardation were rare in these cases of normal grief but more common in a comparison group of pathologically depressed

hospitalized cases (Clayton, Herjanic, Murphy, & Woodruff, 1974). A substantial percentage approaching half of the normal grief cases satisfied criteria for a DSM-style symptom-based definition of MDD at some point within the first year after the loss, even though presumably they were not disordered (Bornstein, Clayton, Halikas, Maurice, & Robins, 1973; Clayton et al., 1971). These findings suggest the presence of two types of symptoms following the loss of a loved one, some symptoms that commonly express normal distress and others that may be more suggestive of depressive pathology and thus "pathosuggestive."

The data by Clayton and colleagues motivated the creation of a bereavement exclusion for the diagnosis of MDD in the earlier DSM editions, in which the diagnosis of major depression was not applied if the symptoms were better accounted for by normal bereavement. The bereavement exclusion is perhaps more properly referred to as the uncomplicated bereavement exclusion because it allowed for the well-established possibility that the death of a loved one could trigger a pathological state of depression (Parkes, 1964). Even after loss of a loved one, depression was still considered pathological if it involved psychoticism, suicidality, morbid feelings of worthlessness, psychomotor retardation, or marked impairment—the symptoms found to be rare in normal grief but more common in pathological cases—or if the reaction was of inordinately prolonged duration, which was defined as a duration of more than 2 months irrespective of the nature of the symptoms.

In addition to their empirical pedigree, some of these criteria for complicated depression comport with major clinical theories about what distinguishes the pathology of depression from normal sadness. For example, the "worthlessness" criterion goes back to Freud's (1917) analysis of what distinguishes sadness from pathological depression and is likewise a staple of the cognitive theory of depression. The psychomotor criterion reflects the view that depressive disorder often takes the form of melancholic depression of which psychomotor disturbances are a core feature (Parker, 2011). The criteria also address issues of clinical necessity (e.g., marked functional impairment, suicidal ideation).

The uncomplicated bereavement exclusion was a somewhat puzzling feature of the diagnosis of MDD. On the one hand, it made common sense and it was consistent with the history of medical thinking about depression that had always highlighted the fact that symptoms of depressed mood after losses can resemble pathological depression and used severity or duration to distinguish pathological from normal cases. On the other, it implied that there was something specific about grief that was not true of other stressors (e.g., marital dissolution or other relationship disruption, loss of job or financial reversals, negative medical diagnosis) because no other stressor had an "uncomplicated" exclusion. Indeed, the DSM revision process never considered whether such an exclusion of uncomplicated reactions to other stressors might be warranted.

The uniqueness or nonuniqueness of depressive reactions to grief was finally addressed in the run-up to the revision of the DSM that yielded DSM-5. Wakefield and colleagues undertook a series of empirical investigations to test not only whether the bereavement exclusion constitutes a justifiable exception to the DSM's

symptom-based definition of MDD, but also whether uncomplicated reactions to a broader range of loss events should also represent exceptions to the diagnosis. This research program emerged out of a review of psychological, comparative, infant, anthropological, sociological, epidemiological, evolutionary, and historical considerations regarding depression (Horwitz & Wakefield, 2007). Based on the harmful dysfunction evolutionary approach to disorder, the review suggested that uncomplicated reactions of the sort identified in the bereavement exclusion are likely a form of evolved species-typical emotional response that is most often non-disordered.

An initial question in exploring uncomplicated depression was the critical question of whether grief-related uncomplicated depression is indeed unique or whether the uncomplicated/complicated distinction is applicable homogeneously across, and looks somewhat similar across, different stressors. Wakefield et al. (2007) used data from the nationally representative National Comorbidity Study (NCS; Kessler et al., 1994) to compare uncomplicated versus complicated MDD episodes after death of a loved one versus after other reported events that triggered depressive episodes. This was the first time the DSM's distinction between complicated versus uncomplicated depressive reactions during bereavement was applied to depressive reactions after other stressors. Consistent with their homogeneity hypothesis, the bereavement- and other-triggered groups were comparable in terms of demographics, clinical history, percentage of individuals qualifying as "uncomplicated," and symptom profiles. With regard to symptom profiles, the only difference was that reactions following bereavement were more likely to include the symptom "thinking about death," which makes sense without postulating pathology.

Most importantly, bereavement-triggered and other-triggered depressions that were classified as uncomplicated were uniformly low in pathology-indicating validators and statistically indistinguishable from each other on eight out of nine pathology validators used to measure severity and likelihood of pathology, which included number of symptoms, likelihood of meeting criteria for melancholic depression, rates of suicide attempts, duration of symptoms, interference with life, prior episodes, and rates of mental health service utilization. The only exception was a modest but statistically significant difference in those reporting that depression "interfered a lot" with their life: more individuals in the uncomplicated other loss-triggered group than in the uncomplicated bereavement-triggered group (12.4% vs. 4.6%) reported this. These results supported the primary hypothesis of a homogeneous uncomplicated-depression category across triggering stressors that has a substantially more benign clinical profile than standard MDD does.

Wakefield et al. (2007) went further and systematically tested whether uncomplicated depressive reactions, whether after the death of a loved one or after other stressors, were less pathological as indicated by pathology validator levels than complicated depressions. Across validators, the uncomplicated cases had significantly and generally substantially lower levels. Uncomplicated cases reported fewer symptoms, their longest lifetime episode was briefer, they were less likely to report a lot of interference with life by depressive symptoms, they engaged in less help-seeking behavior, and they reported fewer lifetime depressive episodes and fewer suicide attempts. Individuals with complicated depressive responses to either kind

of stressor scored much higher than individuals with uncomplicated responses to either kind in terms of pathology validators. Based on these data, the authors concluded that uncomplicated depression is not only homogeneous across stressors but also basically different from major depression and in all likelihood a non-disordered response, and that the bereavement exclusion consequently should be expanded to include other stressors.

A subsequent broadly similar study using a different sample (Kendler, Myers, & Zisook, 2008) also failed to find any evidence that bereavement-triggered depression meaningfully differed from depression triggered by other stressors. However, these authors interpreted their findings to mean that the bereavement exclusion should be eliminated rather than expanded. They reasoned that, because uncomplicated reactions to stressors other than grief are currently classified as disorders, and the bereavement-related uncomplicated episodes are no different from the other-stressor-related uncomplicated episodes, there is no reason to single out bereavement as a unique stressor after which uncomplicated depressive reactions are not disorders. However, Kendler et al. (2008) failed to analyze the overall relationship of uncomplicated and complicated episodes, thus leaving ambiguous how different these two classes may be.

Thus, Wakefield et al. (2007) and Kendler et al. (2008) agreed that uncomplicated depressive reactions to the death of a loved one and to other stressors should be homogenously classified, but disagreed on whether the "uncomplicated" reactions should be classified as disordered or non-disordered. Although many studies subsequently supported the validity specifically of the bereavement exclusion addressing depressive symptoms during grief (Mojtabai, 2011; Wakefield & Schmitz, 2012b, 2013a), based on the 2007 and 2008 studies the focus of further research shifted to the broader question of the validity of the general distinction between uncomplicated and complicated depression across all contexts.

Objections to the 2007 Study

Although the DSM made the controversial decision to eliminate the bereavement exclusion, Wakefield and colleagues continued to pursue a program of research to quell the debate surrounding uncomplicated depression. Although the initial findings were impressive given the general lack of validity of proposed subcategorizations of depression (Baumeister & Parker, 2012), and they were consistent with the history of thinking on depression and the early grief studies, the claim that uncomplicated depression is different from the rest of MDD and more like normal sadness was met with much skepticism.

The tautology bias. Critics of Wakefield et al.'s (2007) analyses drew attention to potential confounders that may have biased the magnitude of the differences between uncomplicated and complicated depressions (Kendler & Zisook, 2009). For example, for episodes to be classified as "uncomplicated," they must be brief, yet the duration of an individual's longest depressive episode was also used as a

validator of depressive pathology. Similarly, "suicidal ideation" is a "complicated" definitional symptom but is closely conceptually linked to the "suicide attempt" validator; "marked role impairment" is a "complicated" definitional symptom, but is closely related to the "interference with life" validator; and, although not strictly a conceptual connection, the "complicated" definitional symptom "suicidal ideation" is closely related to the "hospitalized" validator because suicidal ideation is frequently used as a reason for hospitalizing a depressed patient. It is worth noting that this criticism, although valid, applied only to some of the indicators of pathology used. For example, the number of prior episodes was not inherently biased by the definition of uncomplicated depression.

To address the criticism of definitional biases, Wakefield and Schmitz (2013a, 2013b) conducted further studies on bereavement-related depression and on depression occurring in the presence of other stressors. They addressed potential biases by reconducting each validator analysis after removing the symptom questioned as biased. For example, when comparing the validator lifetime suicide attempts between complicated and uncomplicated cases, they did not use current (in the episode) suicidal ideation as an indicator of complicated depression. They repeated this process of "purifying" the analyses of potential biases for all the relevant validators. The results were that the validator findings remained virtually identical after these corrections, showing that the results had not been due to the proposed biases. Uncomplicated cases still had a significantly more favorable and less pathological presentation than complicated cases. These findings support the conclusion of the original study by Wakefield et al. (2007) that the differences between uncomplicated and complicated depression represent real syndromal differences and are not due to biases in the definition of uncomplicated depression.

Clinical significance criterion. Another notable objection to Wakefield et al.'s (2007) findings focused on the details of the diagnostic criteria for MDD. Peter Kramer (2008a, 2008b) of *Listening to Prozac* fame observed that the analysis used a definition of MDD that excluded the "clinical significance criterion" introduced in DSM-IV (APA, 1994) because the NCS used earlier DSM-III-R-based diagnostic criteria for MDD. The clinical significance criterion (CSC) imposed an additional requirement on depression diagnosis: it required that the depressive symptoms must cause clinically significant distress or functional impairment. Kramer argued that the uncomplicated versus complicated distinction in the NCS was moot because the uncomplicated cases likely did not satisfy the CSC and so would likely have been eliminated from MDD altogether by the more rigorous DSM-IV diagnostic criteria.

The CSC was originally introduced to reduce false-positive diagnosis of harmless feelings of sadness, a goal consistent with the goal of the uncomplicated depression analysis. However, due to conceptual flaws, the CSC did not seem to be a useful tool for reducing diagnosis. The MDD diagnostic criteria already seem to implicate distress (e.g., sadness, worthlessness/guilt) or impairment (e.g., loss of sleep, difficulty concentrating) and had been argued to be essentially useless in reducing false positives because both normal and pathological sadness caused distress and role impairment (Spitzer & Wakefield, 1999). This "redundancy" criticism

was subsequently empirically validated by Zimmerman, Chelminski, and Young (2004), who studied a large group of patients from a clinical practice. In their sample of 1500 outpatients who met the symptom and duration criteria for MDD, not a single patient was eliminated from MDD diagnosis when adding the CSC as a requirement. A further study confirmed that the CSC does not eliminate a significant number of cases in community epidemiological samples (Wakefield et al., 2010), thus falsifying Kramer's claim that the uncomplicated cases in the NCS analysis would have been eliminated from NDD by the CSC.

Uncomplicated depression is a mild disorder. Another criticism to the exclusion of uncomplicated depression is the argument that uncomplicated depression is simply a milder form of MDD. Some have hypothesized that this accounts for its more favorable prognosis and clinical profile (Kendler & Zisook, 2009). In a sense, the definition of uncomplicated depression is biased towards milder symptoms because it excludes two symptoms—suicidality and psychomotor retardation—and a more severe version of a third symptom, worthlessness. However, the existing data suggests that the differences between complicated and uncomplicated depression are not better accounted for by severity. Wakefield and colleagues sought to examine the role of severity in determining complicated and uncomplicated depression. Wakefield and Schmitz (2012b) examined three different definitions of severity, derived from the DSM-IV and DSM-IIIR, and compared their overlap with that of uncomplicated depression. The findings of that study suggest that whereas uncomplicated status is related to severity, the two are not equitable. For example, using DSM-IV criteria for severity about 58% of the cases were of moderate or severe severity, suggesting that uncomplicated depression is not literally the same as or a subset of mild depression.

Not only is it impossible to reduce uncomplicated depression to mild depression, but also the existing data suggest differences in the correlates of the groups. Wakefield and Schmitz (2013b) reported that the pathological validators' interference with life, past suicide attempts, melancholic depression, depression duration, the number of prior hospitalizations, and the number of symptoms was predicted by uncomplicated depression status above and beyond depressive symptom severity. Additionally, uncomplicated depression has greater predictive validity in predicting low rates of depression recurrence than does mild severity using a standard number-of-symptoms measure (Wakefield & Schmitz, 2013c). Uncomplicated depression is simply not reducible to mild depression as generally defined.

Recurrence of Depression

All the evidence considered above regarding the uncomplicated/complicated depression distinction concerns concurrent validation. That is, it explores the validity of the distinction when applied to the nature of the uncomplicated or complicated depressive episodes themselves. A more powerful form of evidence concerns predictive validity, in which differences in the outcomes over time of the two types

of depression are studied. Here, the initial variable to be considered was the occurrence of further episodes of depression in the years following the target uncomplicated or complicated episode.

A hallmark of depressive disorder throughout its history has been the observation that the disorder tends to recur. Indeed, a course distinguished by recurrent episodes was one of the characteristics used in the nineteenth century by Emil Kraepelin to identify depressive disorder as distinct from intense normal responses (Horwitz & Wakefield, 2007), and remains the most important validator of depressive disorder (Kendler, 1990). Recovery from a depressive episode is thus considered distinct from recovery from depressive pathology per se (Frank et al., 1991). Recurrence in depression, then, is interpreted to represent the activation of an internal vulnerability. The number of depressive episodes that an individual experiences can thus be considered a proxy for latent depression vulnerability. If depressive states are contextually bound, they may be expected to dissipate with the passage of time, the activation of internal coping, or the changing of circumstances. If an individual has an underlying pathology that manifests as a vulnerability to depressive episodes, then depression may be expected to persist or recur. Thus, recurrence has often been used in empirical studies as the most revealing validator of the presence of depressive disorder (Kendler & Gardner, 1998). Substantially elevated recurrence rates are considered to be characteristic of MDD in both clinical and community samples (Burcusa & Iacono, 2007; Colman et al., 2011; Coryell, Endicott, & Keller, 1991; Greden, 2001; Judd et al., 1998; Monroe & Harkness, 2011; Mueller et al., 1999), justifying the standard conceptualization of MDD as an episodic and chronic condition for which prevention of recurrence through maintenance of therapy is a major treatment goal (Bockting et al., 2008; Clarke et al., 1999; Farb, Irving, Anderson, & Segal, 2015; Nierenberg et al., 2003; Solomon et al., 2000; Vittengl, Clark, Dunn, & Jarrett, 2007; Vittengl, Clark, & Jarrett, 2010).

Given the strong theoretical and empirical rationale for using depressive recurrence as a pathology validator, Wakefield and Schmitz (2013c, 2014a) used recurrence to evaluate the validity of the uncomplicated/complicated depression distinction using two nationally representative epidemiologic datasets, the Epidemiologic Catchment Area Study (ECA; Regier et al., 1990) and the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; Grant et al., 2004). The ECA study had a wave 2 follow-up interview 12 months after the initial wave 1 evaluation. Wakefield and Schmitz (2013c) explored the recurrence of depression during this follow-up period in individuals with complicated versus uncomplicated depression. As a stronger test of the validity of uncomplicated depression, they also compared recurrence in complicated versus uncomplicated MDD to the occurrence of major depression in members of the population with no prior history of major depression. If the category of uncomplicated depression indeed does not capture pathological responses, one would expect that the rates of recurrence of MDD would be comparable to the rate at which depression otherwise occurs. In this dataset, the rate of MDD recurrence was significantly lower in uncomplicated than in complicated depression, as predicted. Moreover, the rate of

recurrence in uncomplicated depression did not significantly exceed the rate of the initial occurrence of major depression in individuals with no prior major depressive disorder. Adding support for the specificity and validity of uncomplicated depression as a distinct group, this pattern of findings was not obtained for mild (vs. more severe) or non-melancholic (vs. melancholic depression). Each of these categories, mild depression and non-melancholic depression, had lower recurrence rates than their counterparts, moderate to severe depression, and melancholic depression. However, in both cases, the rate of MDD recurrence exceeded the rate at which MDD occurs in individuals with no prior history. Thus, while mild and non-melancholic depressions appear to be less pathological subgroups of depression, using the recurrence rates as validators suggested that the groups still exhibit a vulnerability to depressive pathology. The same is not true for individuals with uncomplicated depression. In other words, knowing that an individual has uncomplicated depression is as informative, in regard to predicting depression risk, as knowing that they have no history of depression.

Although the ECA analyses provided strong predictive evidence that uncomplicated depression is a valid category that may capture false-positive diagnoses, the study was not without limitations. The study had a relatively short follow-up period. Moreover, the data did not allow for the examination of the DSM-IV clinical significance which, although redundant in practice, left the results open to criticism. Thus, Wakefield and Schmitz (2014a) replicated and expanded upon their work using the NESARC data to address these and other limitations. A trade-off was that a limitation of the NESARC data is that the distinction between complicated and uncomplicated could only be derived for an individual's worst depressive episode, so that the analysis of the outcomes of uncomplicated versus complicated episodes had to be restricted to single-episode cases. Thus, the authors compared the recurrence of depression in the NESARC's 3-year follow-up in individuals with (1) no history of depression, (2) single-episode uncomplicated depression, (3) single-episode complicated depression, and (4) recurrent depression (complicated or otherwise). The results of that study again supported the validity of uncomplicated depression. The rates of recurrence of depression in uncomplicated cases were lower than the rates of recurrence in recurrent and in complicated depression. More importantly, individuals with single-episode uncomplicated major depression did not have higher recurrence rates than the rates of the occurrence of MDD in individuals with no prior history of depression. These findings supported the authors' earlier assertion that uncomplicated depression is not indicative of pathology. The findings are impressive if one considers the study's lengthier 3-year follow-up period. Whereas the identification of depressive subgroups that differ in their recurrence rates is not impressive in and of themselves, the identification of a subgroup that more closely resembled individuals with no history of depression is unique and could represent a step towards refining the category of major depression to better distinguish those warranting extended treatment to prevent recurrence.

Suicide

Suicide attempts are among the most devastating sequelae of depression, and depressive disorder is known to have substantially elevated suicide rates over the general population. Thus, an important concern in any endeavor surrounding the classification of depression is to balance the sensitivity and specificity of the diagnostic criteria in relation to the prediction of suicidality. This is an area in which false negatives—mistaking someone who is suicidal for someone who has no problem—are of the greatest concern. Although most individuals who have depression are not suicidal, nonetheless for good reason proposals to change the threshold at which depression is diagnosed are frequently challenged on the grounds that they risk underdiagnosing depression in potentially suicidal patients and thus leading to inadequate treatment that might prevent the patient's death.

Because the risk of missing suicide cases was raised as a major criticism of the proposed exclusion of uncomplicated cases from MDD, Wakefield and Schmitz (2014a, 2014c) undertook an empirical examination of suicidality following uncomplicated versus complicated depression. In a comprehensive analysis, they analyzed the four major epidemiological surveys (ECA, NCS, the NCS replication [NCS-R], NESARC) in which relevant data were publicly available to explore the association between uncomplicated depression and suicidality. Prior analyses of the NESARC (Wakefield & Schmitz, 2014a) had suggested that the rate of lifetime suicide attempts was lower among those with uncomplicated depression compared to other MDD. The subsequent four-dataset analyses, however, demonstrated more conclusively that rates of both concurrent and later suicide attempts were no greater—and, in fact, in some instances were lower—among those with uncomplicated depression than among those who did not have a history of depression (Wakefield & Schmitz, 2014c). Moreover, the uncomplicated/complicated depression distinction still prospectively predicted suicidality even if the related criterion capturing current suicidal ideation during the episode was removed. A subsequent analysis of the data suggested that aside from suicidal ideation a specific symptom, feelings of worthlessness, explained much of the predictiveness of suicidality of the uncomplicated/complicated distinction (Wakefield & Schmitz, 2016). In other words, not only was the uncomplicated depression distinction valid and unbiased, but there also appeared to be a specific link between one theoretically relevant symptom and an important validator, yielding robust syndromal predictions even when some criteria were altered.

Generalized Anxiety Disorder as a Predictive Validator of Major Depression

From the time of ancient Greek medicine until the late nineteenth century and even in the diagnostic manuals of the early twentieth century, anxiety was considered a basic symptom of depression/melancholia (Crocq, 2015). The strict distinction between anxiety and depression occurred only recently in the DSM-III with the attempt to cleanly separate the diagnosis of generalized anxiety disorder (GAD) and related anxiety disorders from the diagnosis of depression. The recent re-"discovery" of high rates of comorbidity of GAD and MDD in epidemiological surveys has led to renewed exploration of their relationship, which has demonstrated a strong etiological relationship between the two disorders that suggests that they are often two aspects of a shared vulnerability to internalizing psychopathology.

Indeed, GAD and MDD "are the most common type of anxiety-mood comorbidity" (Gorwood, 2004, p. 27) with a relationship so strong that prior to DSM-5 a workgroup investigated whether GAD and MDD should be classified as a single disorder (Kendler & Goldberg, 2004). A large body of research supports the conclusion that GAD and MDD often lead one to the other, and that individuals with GAD often eventually develop MDD (Moffitt et al., 2007). Moreover, a strong genetic correlation between GAD and MDD indicates that they emerge from a similar underlying temperament and is further evidence that they share a common pathogenesis. For example, over 25 years ago, having studied genetic loadings of MDD and GAD, Kendler, Neale, Kessler, Heath, and Eaves (1992) stated: "[g]enetic factors … were completely shared between the two disorders" (p. 716), and a few years later observed that "[i]n both clinical and epidemiological samples, … the best-fitting twin models … found a genetic correlation of unity between the two disorders" (Kendler, 1996, p. 68). The same underlying etiological factors do not appear to apply in the same way to MDD and other anxiety disorders: "Genetic influences on these disorders are best explained by two factors, the first of which loads heavily on phobia, panic disorder, and bulimia nervosa and the second, on major depression and generalized anxiety disorder" (Kendler et al., 1995, p. 374). Thus, although MDD and GAD can sometimes represent independent conditions (Horwitz & Wakefield, 2012; Kessler et al., 2008), overall GAD is not just a related risk factor but also appears much of the time to be uniquely etiologically closely related to MDD among possible comorbidities.

Wakefield and Schmitz (2014a) therefore adopted GAD to serve as an additional predictive validator for MDD. To evaluate the predictive validity of the uncomplicated/complicated depression distinction, Wakefield and Schmitz (2014a) used data from NESARC to assess the 3-year follow-up rates of GAD. The study addressed both the nosological question of whether uncomplicated depression is like MDD in general in terms of the elevated level of GAD subsequently generated, and the practical concern of whether, if supposedly benign uncomplicated MDD cases are not diagnosed as MDD, this could result in missing negative sequelae of GAD that are characteristic of MDD.

Wakefield and Schmitz (2014a) tested the hypothesis that uncomplicated depression has a lower 3-year follow-up rate of GAD than does complicated depression. They compared 3-year rates of GAD among groups with (1) no history of MDD, (2) uncomplicated single-episode MDD, (3) single-episode complicated MDD, and (4) multiple-episode MDD. They found that the single-episode uncomplicated rates of GAD (4.2% [1.8, 6.7]) were not significantly greater than the no-MDD-history rates of GAD (3.4% [3.1, 3.7]). Additionally, single-episode uncomplicated MDD follow-up rates of GAD (4.2% [1.8, 6.7]) were significantly less than follow-up rates of single-episode complicated MDD (GAD, 8.4% [6.9, 9.9]). As uncomplicated depression does not increase the likelihood of GAD over those with no MDD history, these results validate uncomplicated depression as a condition that is not equivalent to other major depression and is more like no depression in terms of sequelae.

Objections to Prospective Validators

The prospective data suggest that uncomplicated depression does not confer increased risk for depression recurrence, suicidality, or generalized anxiety disorder. Although these data provided stronger evidence for the validity of uncomplicated depression than the correlational data, they too were met with criticism.

Is uncomplicated depression mild disorder? As with the findings regarding concurrent validators, it is possible that the findings that suggest differences in uncomplicated depression versus other depressions are better explained by symptom severity. However, using the NESARC data to test the "mild disorder" hypothesis, Wakefield and Schmitz (2014a) reported that, even after controlling for depression severity, uncomplicated (vs. complicated or recurrent) depression predicted a lower likelihood of subsequent MDD, GAD, or suicide attempts in wave 2. In effect, with regard to predicting future suicide attempts, symptom severity provided no incremental validity over the "uncomplicated versus other" categorization. Moreover, the follow-up rates of recurrence, GAD, or suicide attempts were higher in mild depression than in those with no history of depression. This pattern of findings diverges from the pattern observed in uncomplicated depression, which has a prognosis comparable to that of not having MDD. These results suggest that while there is a strong argument for considering uncomplicated depression as non-disordered, there is less of a case for mild depression, which indeed appears interpretable as disordered albeit less severely so.

Residual symptoms. Residual depression symptoms are clinically meaningful because subthreshold depressive symptoms may be impairing. Furthermore, residual depressive symptoms predict recurrence and may indicate a latent pathology (Judd et al., 2000). Thus, critics argued that individuals with uncomplicated depression, despite not experiencing elevated rates of full recurrences, may still have had high residual symptoms, suggesting an increased likelihood of future recurrence (Maj, 2014). To address this issue, Wakefield and Schmitz

(2014b) analyzed the average number of MDD symptom groups experienced during follow-up by individuals who did not have depression recurrences during the follow-up period, for each of the depressive subgroups. The mean number of residual symptoms experienced by those who had no wave 2 occurrences during the 3-year follow-up period was not significantly different in the no-lifetime-MDD-history group, 0.37 (95% CI: 0.35–0.39; n = 25,514), and the lifetime single-episode uncomplicated group, 0.49 (95% CI: 0.34–0.65; n = 379), both of which were significantly lower than the residual symptoms in the lifetime single-episode complicated group, 0.75 (95% CI: 0.65–0.85; n = 1756), and the lifetime multiple-episode group, 1.00 (95% CI: 0.91–1.10; n = 1876). Thus, the hypothesis that despite the lack of actual recurrences the uncomplicated group's higher likelihood of recurrence and more negative outcomes would emerge in an elevated rate of residual symptoms was falsified.

Were treatment differences responsible for the results? The use of epidemiologic datasets based on representative community samples for the exploration of nosological issues has many advantages in terms of scientific validity. However, these analyses do not address the issue of the possible influence of treatment on the results. If those with uncomplicated depression are particularly inclined to seek or receive mental health care, their lower follow-up pathology validators could reflect the result of a higher rate of successful treatment.

To address the potential confound that treatment might have accounted for the different rates of recurrence of depression, Wakefield and Schmitz (2014b) analyzed whether differences in treatment utilization explained their outcome finding. The NESARC survey included three lifetime service use questions specific to depression: whether the person ever saw a mental health professional, was hospitalized, or was prescribed medications for depression. Rather than receiving more treatment, the uncomplicated MDD group had a significantly and substantially lower rate of use of any of these services (34.0% [28.0, 40.0]) than either the complicated single-episode (51.0% [48.2, 53.9] or multiple-episode (65.6% [63.7, 67.6]) groups. Consequently, greater amounts of treatment at baseline of uncomplicated cases cannot explain the pattern of wave 2 results. Of note, reported treatment at wave 1 did not predict wave 2 recurrence outcomes for either uncomplicated or complicated single-episode depression cases. Wave 2 depression recurrence rates for wave 1 untreated versus treated uncomplicated cases were 7.0% (3.9, 10.1) and 6.9% (2.3, 11.4), respectively; for complicated single-episode depression cases, the untreated versus treated recurrence rates were 17.1% (14.1, 20.0) and 21.9% (18.8, 25.0), respectively (Wakefield & Schmitz, 2014b). These results raise questions about the effectiveness of treatment in lowering disorder rates (Jorm, Patten, Brugha, & Mojtabai, 2017).

Durational Limit for Uncomplicated Episodes

Variability between individuals in the duration of experiences of depressive mood states has been recognized and documented throughout history and across cultures (McCrae & Allik, 2002; Piedmont & Aycock, 2007). Despite a recognition that individuals normally vary in how often and for how long they experience negative moods, the extreme prolonged duration of states of depressed mood has been used since antiquity as an indicator that they are pathological, going all the back to Hippocrates's assertion that "[i]f fear or sadness last for a long time it is melancholia." The judgment that the duration of sadness is "excessive" may be restated as asserting that the duration of the sadness reaction is disproportionate relative to the magnitude of the loss that triggered it (Horwitz & Wakefield, 2007). Alternatively, and based on an implicit theory of the normal trajectory of recovery from loss, a prolonged duration may imply that the natural recovery process is somehow blocked, or derailed, or has somehow been inhibited (Wakefield, 2012). But, just how long can a reaction of intense sadness to a loss or stressor last before it is plausibly judged to be pathological?

The DSM-III (APA, 1980) and DSM-III-TR (1987) stated no specific duration threshold for depressive disorder, but simply used "prolonged duration" as the criterion. This was generally interpreted to imply a 1-year threshold, an interpretation that was consistent with the operationalization of the Research Diagnostic Criteria (see, for example, Weissman & Myers, 1978). DSM-IV dramatically shortened the allowable time someone who is bereaved who has depressive symptoms could be seen as normal to just 2 months. By eliminating the uncomplicated bereavement exclusion, DSM-5 effectively reduced the threshold for considering a depressive response to a loss to be a disorder to 2 weeks, the duration criterion for major depression.

In several studies, Wakefield, Schmitz, and colleagues have attempted to take an empirical approach to establishing the durational threshold for an otherwise uncomplicated episode lasting long enough to be considered a depressive disorder. Their basic idea was that if there is a natural cut point, then on average, other validators should shift significantly when that durational point is surpassed. They thus compared the relative predictive validity of the DSM-IV 2-month threshold to the earlier 1-year threshold and to a possible 6-month threshold (Wakefield & Schmitz, 2012a; Wakefield, Schmitz, & Baer, 2011a, 2011b). In these studies, depressions triggered by bereavement or other losses were significantly more severe than their briefer counterparts only if they lasted 12 months or more. These results suggested that duration substantially greater than the 2-month threshold used by DSM-IV was empirically supported for identifying pathology. Given the potential for false negatives and the importance of not missing true cases, Wakefield and Schmitz suggested a more conservative 6-month threshold to minimize false negatives. This duration cutoff was used to define uncomplicated versus complicated depression in a series of studies across several datasets, including the NCS, the ECA, and the NESARC (Wakefield & Schmitz, 2011, 2012a, 2012b, 2013c). In both concurrent

and predictive validity studies, the 6-month threshold yielded validator results supporting the distinction that were equally strong or stronger than results obtained with the DSM-IV's 2-month threshold, suggesting that the 6-month threshold is closer to a natural cut point.

Conclusion

The "harmful dysfunction analysis" of the concept of medical disorders dictates that disorder is a product of the failure of a biologically designed function which produces harm as judged by social values and norms (Wakefield, 1992a, 1992b, 1993, 1999a, 1999b, 2000a, 2000b, 2007; Wakefield & First, 2003). A cogent view of medical pathology requires an evolutionary approach to function, dysfunction, and nosology because our judgments about aberrant or abnormal functioning require a prior understanding of adaptive, or "normal," functioning that forms a baseline for judging pathology. The only plausible such objective baseline for normality is how human behavior has been shaped by evolution.

In no area of medicine is this reliance on an evolutionary foundation more important, and more challenging, than in psychiatry. This is because sociomedical ideologies have generally attempted to define pathology based on the presence of varying levels of harm, irrespective of whether there is evidence of disturbed biologically designed functioning. This in effect reduces diagnosis to judgments of value, thus reflecting what we desire people to be rather than who people really are. There is no better example of this than the elimination of the uncomplicated bereavement exclusion in DSM-5. That meant that after just 2 weeks of normal signs of bereavement such as sad mood, loss of interest, low energy, and disrupted sleep and appetite, an individual satisfies the diagnostic criteria for major depression. This may serve social needs for efficiency in intervening quickly with people impaired in their roles by intense negative emotion, but it does not reflect who human beings are or what grief is like by nature.

It is widely accepted that the category of MDD is heterogeneous and it has long been argued that some of the heterogeneity comes from its inclusion of individuals who are likely non-disordered (Horwitz & Wakefield, 2007; Lorenzo-Luaces, 2015; Parker, 2007). However, there has been little in the way of research findings that test this assumption. Research on uncomplicated depression is the first research program to have advanced this debate on empirical grounds.

It should be emphasized that the attempt to distinguish normal from pathological sadness is not about who should be helped. Of course we should help all people who have emotional problems in coping with the dramatic challenges of modern life, whether they are experiencing a normal intense emotional reaction to stress or loss or are suffering from a mental disorder. Even the DSM-5's own Z Codes for non-disordered conditions that are often the target of treatment imply that the mandate for the mental health field is broader than mental disorder. However, the way we help people is shaped by how we understand their condition and its prognosis.

Misclassifying normal-range reactions as mental disorders distorts treatment choice and can be harmful if treatment is more intrusive than needed or has side effects that could have been avoided. The first step towards rectifying this situation is to be clearer about the nature of the problems we diagnose, and one basic distinction that is needed is between disorder and normality.

There are many theories about the evolution of sadness and grief. However, we have little knowledge of the mechanisms underlying such responses, and none of the many theories about the evolution of sadness are developed enough to specify precise parameters of the normal evolved response as distinct from pathological states that represent disruptions of selected functions. This poses a formidable difficulty for nosologists attempting to specify criteria for depressive disorder in community samples where there is much intense normal sadness that needs to be discriminated from pathology. Without the ability to perform direct tests of the integrity of underlying mechanisms or infer precise theory-based thresholds, an alternative approach to exploring the boundary between normal and disordered depression is to propose a hypothesis about a cut point and perform multiple tests bearing on the validity of the hypothesis, creating a web of empirical findings that, although no one finding may be determinative or conclusive, taken together tend to support the hypothesis. That is what the uncomplicated depression research program has attempted to do, with the various results reported above.

The research program's results suggest, first, that there is nothing unique about bereavement as a stressor, yielding a general category of uncomplicated depression that applies across losses, stressors, and contexts. The subsequent research has provided clues as to what features of depression are more suggestive of pathology versus those that are more consistent with normality. It strongly suggests that the subgroup of individuals who meet the full criteria for major depression by endorsing symptoms of generalized distress that are of relatively brief duration and occur in the absence of marked functional impairment may be experiencing normal sadness, not pathological depression.

When compared to individuals who endorse the "complicated" symptoms of depression, individuals who experience uncomplicated depression tend to have less severe and complex clinical pictures characterized by overall fewer help-seeking behaviors and fewer adverse outcomes associated with depression. Most importantly, the predictive validity data reviewed above reveals that the sequelae of uncomplicated depression is unlike the sequelae of other conditions classified as major depression, but is very much like the sequelae of people who are normal and have never had a depressive disorder. In particular, when followed over time, the rate at which depression recurs in this group is not higher than the rate at which individuals in the general population meet the criteria for MDD. This is an important step in establishing validity given that recurrence is considered to be a hallmark of depression's pathology. Moreover, individuals who report uncomplicated depressive episodes are also not at an increased risk of meeting the criteria for GAD or attempting suicide. It is important to note that the definition of uncomplicated depression is not reducible to mild severity of depression; uncomplicatedness has incremental predictive power over and above that of predictions based

on mildness alone (Wakefield & Schmitz, 2013a). These results and the other results reported above provide a web of evidence that taken cumulatively strongly support the hypothesis that uncomplicated depression is an intense form of normal sadness. This program is unusual in that there are almost no other proposed subtypes of depression that have been similarly empirically validated (Baumeister & Parker, 2012).

These findings are frequently met with the criticism that any change made to the category of depression should make it more, not less, inclusive because there are many who fail to be helped. This is a non-sequitur; overdiagnosis of a disorder does not preclude simultaneous underdiagnosis. However, to the degree that there remains an underserved population, this is not because of the diagnostic issues on which we have focused. These individuals satisfy the criteria but for one reason or another either do not seek help or fail to get the help that they do seek. Another side to this opinion is that about half of individuals with DSM-diagnosable mood disorder do not believe that they have a need for treatment (Mojtabai, Olfson, & Mechanic, 2002). They are dismissed as misguided and underdiagnosed cases, but some may understand their conditions and contexts in a way that goes deeper than the DSM diagnostic criteria.

The overpathologization of depression can only be addressed by focusing on the distinction between normal distress and indicators of dysfunctional psychological functioning. The criteria for uncomplicated depression are one empirically supported attempt to distinguish between depressive disorder and distress that meets the criteria for major depression. This distinction should not be the only, or final, attempt at distinguishing pathological versus non-pathological cases of MDD. However, the strength and consistency of the research findings suggest that uncomplicated depression is one category of false positives and confirm the belief that the category of MDD requires rethinking in light of evolutionary considerations. If we take people as they are rather than as we would prefer that they be, then painful as it may be, uncomplicated depression is a normal-range, not disordered, emotional reaction, and informed consent and treatment strategies should be sensitive to this reality.

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Depression: Is Rumination Really Adaptive?

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Introduction

Within evolutionary approaches to depression there is a rather even split between theories that propose that depression and symptoms of major depressive disorder are not adaptive and those that propose that depression is adaptive. An example of the latter, the analytical rumination hypothesis, has since its first formulation (Watson & Andrews, 2002) suggested that the depressive symptoms are adaptive and help solve problems. In the most recent formulation of the theory (Andrews & Thomson, 2009), the analytical rumination hypothesis describes a theory of how rumination and depressive symptoms provide solutions to complex social problems and, therefore, should be promoted rather than treated. This adaptationist approach might seem unsurprising to nonclinician evolutionists, but breaks with a tradition among clinical evolutionary researchers such as Gilbert (1998), Nesse (2011) and Nesse and Williams (1996). Further, standard clinical approaches to depression typically assume that depression is not adaptive, if evolutionary questions are considered at all. Although most clinicians and researchers that work with anxiety have a functional approach to fear, an adaptationist approach has been atypical within clinical approaches to depression.

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Paralleling the conceptual development of the analytical rumination hypothesis, within mainstream clinical science a different approach to depression and treatment of major depressive disorder has been developed. This new approach considers rumination, similarly to the analytical rumination hypothesis, as the key underlying process involved in depression. However, in accordance with the work of Nolen-Hoeksema and her colleagues (Nolen-Hoeksema, 1991; Nolen-Hoeksema & Morrow, 1993), this approach views rumination as a maladaptive *maintaining* factor of depression, rather than as the healing, problem-solving factor. This new approach to treatment is called metacognitive therapy (Wells, 2009) and is a relatively new treatment of mental disorders. It grew out of cognitive behavioral therapy. However, whereas cognitive behavioral therapy focuses on the contents of negative automatic thoughts and cognitive schema (Alford & Beck, 1997; Beck, 2011), metacognitive therapy focuses on mental processing. The major aim of metacognitive therapy is to discontinue rumination in order to reduce depressive symptomology. As such, the analytical rumination hypothesis and metacognitive therapy agree that rumination is central to depression, but they disagree on whether depressive symptoms are adaptive and desirable.

Evolutionary medicine, including adaptationist psychopathology, can shed light on why humans are susceptible to disorders (Nesse, 2011; Nesse & Williams, 1996; Williams & Nesse, 1991). One insight from evolutionary medicine is that not all "symptoms" are diseases, but rather evolved bodily *defenses*. A typical example is fever: fever is an adaptation that protects and aids the host by fighting off infections (Kluger, Kozak, Conn, Leon, & Soszynski, 1998; Nesse & Williams, 1996). Although fever has subjective, behavioral, social, and caloric costs, it serves an important fitness-relevant function. This might be true for some specific symptoms of mental "disorders" as well. Does depressive rumination show this pattern?

This chapter aims to consider the analytical rumination hypothesis and metacognitive therapy and consider the arguments from both positions. The question of whether depression is adaptive cannot be answered solely based on whether depression is aversive. All pain is aversive; without pain one dies young (Melzack, 1973). Thus, adaptive pain may exist. Nevertheless, the pan-adaptationist suggestion that all phenomena that exist are adaptive is naïve and, therefore, rather than an *a priori* truth, adaptationism is a claim that is testable and must be tested.

An Illustrative Case of Depressive Rumination

Below we describe a fictional case. A woman we call Emma. We will refer to this case throughout the rest of the chapter as a means of illustrating differences in relevant approaches to depression and rumination.

Emma, who is in her mid-20s, fails to get along with her new colleagues in an office she started working at 6 months ago. Over time, she develops guilt and hopelessness. Her motivation fails and she no longer feels any joy from the things that used to thrill her, like that sitcom she always watches or those weekends with her

niece. She eats three meals a day but food does not taste as good as it used to and some days she does not even get hungry. Emma used to date a lot but since she started the new job, her romantic and sexual desire has all but gone. Her pace is slow and her posture is submissive. She notices that she takes longer to walk from her house to the subway. Thinking about all these alterations in behavior and mood keeps her up at night, but what she thinks about the most are all the mistakes she has made at work. When she wakes up, she feels exhausted and when she gets to her desk, it is almost impossible to keep her attention on work. Emma is having what clinicians call a depressive episode.

As her depressive symptoms develop, Emma ruminates. She asks herself questions such as these:" Why can't I get along with people?"" Why do people dislike me?"" What's wrong with me?"" What should I do differently?" "Why can't I sleep?" "How will my lack of concentration at work affect relationships with my colleagues?" Maybe the thought she ruminates most on is "why me? Why am I depressed?" One day Emma finds herself in a chair talking to a psychologist. Emma feels trapped by her own thoughts. Uncontrollable and depressing thoughts. Yet, when asked why she asks these questions to herself, her answer is that she must figure out what is wrong with her in order to fix it.

What is Rumination?

Rumination is a form of repetitive thinking. Loosely defined, repetitive thinking is "The process of thinking attentively, repetitively, or frequently about oneself and one's world" (Segerstrom, Stanton, Alden, & Shortridge, 2003, p. 909). Repetitive thinking has been the focus of investigation from many different fields in the social science (Sarason, Pierce, & Sarason, 2014; Uleman & Bargh, 1989). We are interested in the kind of repetitive thinking that we typically see in depressed people. This type of repetitive thinking has been called by many names but sometimes the same name refers to different ideas (Smith & Alloy, 2009). A conceptual clarification is therefore important when discussing the meaning and relevance of rumination in the depressed patient.

Today a common term for the type of repetitive thinking that occurs in depressives is "depressive rumination" (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Papageorgiou & Wells, 2004a). The researcher most closely associated with this term is the late Susan Nolen-Hoeksema, with her work on response styles theory. In early conceptualizations of response styles theory, the ruminative response to a depressed mood was referred to as "the ruminative response" or "rumination," which was defined as "repetitively focusing on the fact that one is depressed; on one's symptoms of depression; and on the causes, meanings, and consequences of depressive symptoms" (Nolen-Hoeksema, 1991, p. 569). However later in the development of the theory the articles describing the "ruminative response" use the more generic term "depressive rumination" about the *response* in response styles theory.

About the same time as Nolen Hoeksema and her colleagues developed response styles theory with a focus on the cognitive risk factors for depression, Martin and Tesser (1989) used the term rumination in their theory of repetitive thinking. Unlike response styles theory, their focus was on self-regulation and state-goal discrepancy, and their theory has been called goal progress theory (Martin, Shrira, & Startup, 2004). In the framework of goal progress theory, they define rumination as an ongoing thought that is focused on a special theme and continues even though there are no demands for the thought in the environment. Since the publication of Martin and Tesser (1996), there seems to be a consensus to reserve the word *rumination* to describe the perseverative nature of repetitive thinking. The broad definition of rumination thus focuses on the repetitive process rather than the content of thought. More specific or narrowly defined constructs use the word rumination together with some prefix or support word/words (e.g., *depressive* rumination Nolen-Hoeksema & Morrow, 1991), rumination *on sadness* (Conway, Csank, Holm, & Blake, 2000), *stress-reactive* rumination (Just & Alloy, 1997), and *post-event* rumination (Clark & Wells, 1995). Rumination more narrowly defined is closer to specific content (e.g., valence, see below). Following this logic, it is expected that the specificity of rumination to depression increases when rumination is defined narrowly.

Depressive rumination is often assessed with the Ruminative Response Scale in the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991; Smith & Alloy, 2009; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Factor analysis of this scale has shown that depressive rumination fits with a two-factor solution with Brooding and Pondering as separate entities, and that it is the Brooding factor that is the best predictor of symptoms of depression (Armey et al., 2009; Treynor et al., 2003). Brooding has been referred to as "moody pondering," indicating that it is the valence dimension that separates the two factors (Treynor et al., 2003).

The finding that positive versus negative valence is a relevant dimension in depressive rumination mirrors the results from another factor analytic study of several questionnaires (Segerstrom et al., 2003). It was found that, in addition to the valence dimension, the degree to which the items represented a search for meaning or an attempt to problem-solve differentiated between items across measures of repetitive thinking. This dimension can be called the purpose dimension in repetitive thinking (Segerstrom et al., 2003). Yet another review indicated that the level of abstractness or concreteness can differentiate between repetitive thoughts, again when considering alternate models and theories about repetitive thinking (Watkins, 2008). The more abstract the repetitive thinking was the less constructive consequences the rumination was found to have.

Evaluating depressive rumination as assessed by the Ruminative Response Scale on the dimensions discussed above we find that (1) *depressive* rumination has a searching purpose instead of a solving purpose; (2) the thoughts are abstract, not concrete; and (3) they have a negative valence. Examples of items that fulfil these criteria are the following: "Think: Why do I have problems other people don't have?" or "Think: Why can't I handle things better." Furthermore, there is a difference between the Brooding component and the Pondering component of depressive rumination in that brooding has a negative valence whereas Pondering is more neutral, albeit not positive, in valence (Treynor et al., 2003). An example of a Pondering item is "Go away by yourself and

think about why you feel this way." This thought is (1) searching for a meaning with or reason for the mood, and (2) abstract because it does not link to a specific episode, but rather mood in general. However, there is not a clear link to a negative attribute. Thinking that one is worse at handling things or that other people have fewer problems focuses on the perceived flaws. Thinking about feelings does not necessarily link to any flaws. Thus, this item does not necessarily have (3) a negative valence.

The content of repetitive thoughts is also important when differentiating depressive rumination from *worry*, and the purpose factor discussed above can be used to distinguish between them (Borkovec, Ray, & Stober, 1998; Smith & Alloy, 2009). Worry is more future oriented than depressive rumination which has a greater focus on past events. In addition, worriers tend to have greater confidence that worrying will help in problem solving than ruminators (Papageorgiou & Wells, 1999a, 1999b, 2004b). Depressive rumination has been found to relate more to a searching purpose compared with worry, which was more associated with attempts to solving possible future problems (Watkins, 2004).

Let us illustrate this further with Emma, the depressed woman described earlier. Here are some of the repetitive thoughts that plagued her:

1. "Why can't I get along with people?"
2. "Why do people dislike me?"
3. "What's wrong with me?"
4. "What should I do differently?"

Notice how the three first thoughts focus on a negative theme and are abstract. These are questions so vaguely framed that the answers to them hardly will help her solve a *particular* problem. She is not asking herself the specific question of "why did that episode end bad on that occasion?" She is asking abstract questions about why she has failed as a (social) person. She is not asking for a solution to her problem but rather a reason for or a meaning in the way she appraises herself. The first three ruminating thoughts are classified as brooding; they are abstract, have a searching purpose, and they have a negative valence.

The fourth thought does not ask about a meaning or a reason for why Emma is as Emma is; she is instead asking for alternative ways of behaving. Thus, the purpose with or motivation behind this question is problem solving rather than brooding. The question is neutral in valence but unfortunately for Emma the question is still too abstract to do her any good. There is no *general* way of behaving and before she makes her question more concretely linked with an actual episode it will likely leave her with few new solutions.

The Analytical Rumination Hypothesis

The analytical rumination hypothesis postulates that depression and rumination evolved to solve complex social problems. Andrews and Thomson (2009) structure this hypothesis under the four following claims: (1) complex problems trigger

depressed affect; (2) depression coordinates changes in body systems that promote sustained analysis of the triggering problem; (3) depressive rumination helps people solve the triggering problem; and (4) depression reduces performance on laboratory tasks because depressive rumination takes up limited processing resources.

The analytical rumination hypothesis is an adaptationist hypothesis. This means that the hypothesis postulates that ancestral individuals with an ability to respond to social stressors with depressive rumination experienced fitness benefits because rumination enhanced social problem solving more effectively than alternative designs. From this perspective, if humans still experience the evolutionarily relevant complex social problems, depressive rumination will still serve this evolutionary function in humans.

Let us illustrate the logic of the analytical rumination hypothesis by going back to our case Emma. From this perspective, the reason she loses interest in pleasurable activities, sleeps less, eat less, has a failing sexual desire, walks slowly, and has a hard time concentrating at the office is because these symptoms help Emma engage, without distraction, in depressive rumination that is designed to *solve her problems*. The presence of a complex social problem has activated an adaptation designed to increase the chance that her social problems will stay in her working memory so that she may effectively analyze and solve them. Her inability to focus at work is thus caused by complex adaptations that use up her cognitive resources, in order to solve her fitness-relevant problem, not because her failure to focus is simply a symptom of her mental illness. It is a feature, not a bug.

As mentioned in the introduction, mainstream clinical theories on depression and depressive rumination usually assume that depression and rumination are maladaptive, or at least unhelpful to the individual (i.e., they are clinical states in need of treatment). The analytical rumination hypothesis is therefore an important alternative view of the possible adaptive functions of depressive rumination. The analytical rumination hypothesis provides testable predictions regarding the functions rumination might serve, which is helpful when asking questions about why rumination exists and why depression and rumination are so common. Even though we think that the analytical rumination hypothesis has many problems (see below), these predictions can produce new knowledge on the evolution of rumination and repetitive thinking, more generally.

There is evidence that low-mood states have specific effects on decision making and interpersonal processes, suggesting that moods are evolutionary functional (Forgas, 2002, 2013). Broadly speaking, more negative moods might call for a focus on the details of the external world, whereas more positive moods rely on preexisting internal schematic knowledge and heuristics (Forgas, 2016). In studies in which people are induced into a sad mood, people produce more concrete, persuasive, and higher quality arguments (Forgas, 2007). The ability to accurately detect deceptive communication is also improved in sad people (Forgas & East, 2008). There are many other examples in the literature of how sadness can improve motivation, interpersonal behavior, memory, and judgment in predictable manners (for reviews see Forgas, 2013, 2016). Based on several lines of evidence on sad people and their detail-focus and cognitive perseverance, the analytical rumination hypothesis postulates that depressive disorders also

serve these functions. From this perspective, many people who engage in depressive rumination and have major depressive disorder experience the high intensity of symptoms because the social problems they face in their lives are highly complex: The greater the complexity of the problems, the more depressive symptoms and depressive rumination are necessary to solve (Andrews & Thomson, 2009).

Although sadness—and possibly the repetitive thinking typically involved—might serve specific cognitive functions such as problem solving, it is unclear how this relates to depressive disorders such as major depressive disorder as defined by the DSM-5 (American Psychiatric Association, 2013). When treating depression, the analytical rumination hypothesis predicts that psychotherapeutic techniques that interrupt or discourage problem-solving behavior in patients will be less effective than therapy that facilitates the ruminative processes in which depressed individuals engage (Andrews & Thomson, 2009). In other words, encouragement of rumination should be more effective in relieving patients' depression symptoms in the long term, compared to therapies that teach disengagement of depressive rumination. What does the evidence from the treatment literature tell us about these questions?

Metacognitive Therapy

Metacognitive therapy (Wells, 2009) is a relatively new approach to the treatment of mental disorder. Metacognitive therapy focuses on the maintaining mental processing, and seeks to discontinue these processes. The maintaining mental processes are considered inflexible and maladaptive (in the here and now) due to what is called cognitive attentional syndrome (CAS; Wells, 2000). This construct guides unhelpful coping behaviors, including worry and rumination, and threat-monitoring behavior. This persistent mental processing results in the symptoms of disorder, and low mood or other aversive emotional states. Cognitive attentional syndrome is monitored and maintained by metacognitions. There are two major types of metacognitions: positive metacognitions about the benefits of ruminating and negative metacognitions about the harm or uncontrollability of rumination. Rumination is initiated and maintained due to positive metacognitions. Conversely, negative metacognitions are a result of distress and unhelpful learning experiences about the depression. Many patients attempt to stop ruminating by attempting to force negative thoughts out of their mind, self-distract, or avoid triggers of negative mood or rumination. None of these coping behaviors are helpful or efficient. Fear of negative thoughts and negative metacognitions is therefore the result, and both are maintained and strengthened in the process.

The metacognitive therapist will therefore aim to reduce intuitive but unhelpful coping behaviors and challenge metacognitions, to deactivate cognitive attentional syndrome and thereby discontinue rumination behavior and reduce depressive symptoms. The metacognitive therapist, unlike cognitive behavior therapists, does not challenge the content of the patients' depressive rumination and brooding. Rather it is the idea that rumination is helpful that is challenged: metacognitions, both negative and positive, are

changed through verbal reattribution or behavioral experiments. The patient will learn how to gain attentional control (through the attentional training task) and, further, the patient is taught how to meet rumination triggers with observation not engagement (detached mindfulness). So when Emma experiences symptoms of depression she might engage in rumination, which her questions about "Why she can't sleep?" or "How her lack of concentration at work will affect relationships with her colleagues?" illustrate. Both lack of sleep and concentration challenges Emma in her daily activities like socializing at work, watching television, or feeling rested. However, the metacognitive therapist will point out that ruminating about why she has acquired those symptoms might make them worse. Instead the therapist will encourage her to leave her negative thoughts alone without attempting to force them out of consciousness. Forcing triggers out of consciousness is not possible. Further, engaging with them in rumination, brooding, worry, or other forms of repetitive mental processing maintains aversive emotion and unhelpful behavior. The metacognitive therapist will ask her how long she has ruminated. How did that make her feel? How many problems has rumination helped her solve? Would it be time to try a different strategy? This challenges both positive and negative metacognitions. By taking a detached mindfulness stance to triggers (discontinuing rumination by leaving the thoughts alone), Emma will notice that her mood does not drop. She will believe less in the uncontrollability of metacognitions, as a result.

Comparing the Analytical Rumination Hypothesis with Metacognitive Therapy

The major disagreement of these two approaches is in how they conceive rumination. Although the adaptive approach suggests that rumination is desirable and currently adaptive, a source of future well-being, the metacognitive approach considers rumination to be pathological, both here and now and in the long run. The most fascinating aspect of this disagreement is that most of the tenets of the analytical rumination hypothesis are the intuitive metacognitions shared by most depressive patients. Metacognitive therapy targets these metacognitions for intervention. In other words, from a metacognitive therapy perspective, depressive patients are intuitive analytical rumination hypothesis theorists, and treating depression will involve attempting to change their minds about how adaptive and uncontrollable rumination is. The analytical rumination hypothesis has yet to be applied in clinical trials. Treatment based on the tenets of the analytical rumination hypothesis has yet to be developed, but would include techniques that aim to facilitate and encourage the rumination process, such as expressive writing, wherein a spike in depressive symptomatology during treatment is considered a sign of progress (Hayes, Beevers, Feldman, Laurenceau, & Perlman, 2005). Many clinicians would be skeptical of such an approach, in which depression is the outcome of a functioning organism, not pathology, and in which the severity of symptoms gauges functioning not pathology.

A crucial point of the analytical rumination hypothesis is that rumination should be difficult to discontinue. Different symptoms of depression are interpreted as coordinated changes in body systems designed to maintain analytical rumination: increased activity in the left ventral prefrontal cortex has the function of maintaining problem-related information in working memory. Psychomotor changes such as appetite reduction, reduced sleep, and psychomotor retardation free up cognitive resources in order to maintain rumination. Further, anhedonia reduces motivation to engage in distracting activities (Andrews & Thomson, 2009). In short, according to the analytical rumination hypothesis, depression is designed to maintain rumination and make it difficult to stop. Although metacognitive therapy also considers rumination to be resilient to distraction and forced cessation, metacognitive therapy holds that this is only due to false assumptions, unhelpful strategies, and metacognitions maintaining the cognitive attentional syndrome.

Thus, whereas the analytical rumination hypothesis claims that it is difficult to stop ruminating, metacognitive therapy considers this a false-negative metacognition. The metacognitive therapist will teach the patient detached mindfulness. As we have seen, this consists of leaving negative thoughts and ruminative triggers alone, not engaging with them. As the thoughts are not dangerous, and one does not need to engage with them—something patients believe one does need to do due to both negative uncontrollability metacognitions and positive metacognitions about the benefits of ruminating—leaving them alone makes them fade, both in awareness and in relevance. Those metacognitions make detached mindfulness a nonintuitive stance to negative thoughts and rumination triggers. On the other hand, the intuitive coping behavior is to attempt to distract oneself or force the thoughts out of consciousness. However, these two approaches do not work. The thoughts and triggers appear more often in consciousness due to those strategies, which paradoxically reinforces ideas about depression being uncontrollable and harmful (negative metacognitions).

Findings from Clinical Trials Implementing Metacognitive Therapy

Wells et al. (2009) conducted a case study on four patients with major depressive disorder, where 6–8 weekly sessions of metacognitive therapy were administered as described by Wells and Papageorgiou (2004) and Wells (2008). Depression was measured with the Beck Depression Inventory (Beck, Ward, & Mendelson, 1961) and Hamilton's Rating Scale for Depression-17 (Hamilton, 1960). Meta-beliefs about rumination were measured with Positive Beliefs about Rumination Scale (Papageorgiou & Wells, 2002) and Negative Beliefs about Rumination Scale (Papageorgiou & Wells, 2003). Ruminating behavior was assessed with a weekly measure of rumination constructed for the study. All four patients showed a rapid decline in ruminating behavior once treatment

started, and showed dramatic improvements on all measures at 3- and 6-month follow-up compared to baseline (Wells et al., 2009). Likewise, Wells et al. (2012) found significant improvements in depressive symptoms, rumination, and metacognitive beliefs at 6- and 12-month follow-up in 12 patients with treatment-resistant depression after up to eight sessions of metacognitive therapy. An A-B design case study with 5–11 sessions of metacognitive therapy on four depressed participants found significant improvement in depressive symptoms, rumination, and metacognitive beliefs at 3- and 6-month follow-up (Callesen, Jensen, & Wells, 2014). A multiple baseline pilot study on six women with postpartum depression found significant reductions in depression and metacognitive beliefs in all participants, with large effect sizes that were maintained at 3- and 6-month follow-up (Bevan, Wittkowski, & Wells, 2013). A study of group metacognitive therapy found large significant improvements in ten patients at 1- and 2-year follow-up (Dammen, Papageorgiou, & Wells, 2015, 2016). In one of the first randomized controlled trials, Jordan et al. (2014) found moderate to large effect sizes for both cognitive behavioral therapy and metacognitive therapy in the treatment of 48 participants suffering from major depression and bipolar disorder. Groves et al. (2015) investigated differences in neuropsychological functioning after either cognitive behavioral therapy or metacognitive therapy treatment. Thirty-three depressed participants completed a battery of tests on executive functioning and attention before, during, and after treatment. Those randomly assigned to metacognitive therapy treatment showed significantly greater improvement in spatial working memory and executive function, compared to the cognitive behavioral therapy group. Considering that depression is a disorder with high comorbidity, Hjemdal et al. (2016) conducted a trial of metacognitive therapy with ten participants with major depression and comorbid psychiatric disorder. After 10 sessions of metacognitive therapy, all cases of major depression were successfully treated, and of 21 diagnoses at pretreatment, only 3 remained. Finally, in a recent randomized waitlist controlled trial (Hagen et al., 2017), 39 participants received 10 sessions of metacognitive therapy either immediately or following a 10-week waitlist condition. Hagen et al. (2017) report large controlled effect sizes both depressive ($d = 2.51$) and anxious symptoms ($d = 1.92$). Further, they classified 70–80% as recovered posttreatment and at 6-month follow-up following metacognitive therapy.

In summary, metacognitive therapy has been found to be an effective treatment for major depressive disorder both as group therapy and individual therapy and improvements are maintained at 6-, 12-, and 24-month follow-ups for the different studies, respectively. In addition, metacognitive therapy shows promise as a transdiagnostic treatment, and seems particularly effective against comorbid psychiatric disorder. The fundamental reason for this may be the transdiagnostic feature of maintaining mental processing; many disorders are maintained by worry, rumination, or other mental processing. Therefore, learning that one is in control of one's own thoughts and mental processing by changing metacognitions that suggest otherwise might reduce several symptoms and suffering across several disorders. Especially worry and rumination will be reduced, according to the current research.

Process Research: Is the Actual Change Due to the Specified Treatment Model?

Three important questions remain after reviewing findings from the efficacy studies. Although it seems that metacognitive therapy is associated with unusually efficient symptom reduction, this does not mean that change is caused by the processes specified by the treatment model. The claim from metacognitive therapy is that change and symptom reduction is due to reduced beliefs in both positive and negative metacognitions and reduced implementation of rumination as a result. Does metacognitive therapy target metacognitions? Does metacognitive therapy result in reduced rumination? Moreover, are changes to metacognitions and reduced rumination the reason for symptom reduction?

Basic experimental research in attention and psychopathology suggests that continuation or discontinuation of rumination is indeed causing or removing depressive symptoms (Wells & Matthews, 1994). For example, depressed subjects randomly assigned to attend to objects rather than to ruminate on emotions were significantly less depressed (Nolen-Hoeksema & Morrow, 1993). There is some relevant basic research that might shed some light on these issues, such as a study that found that, even for obsessive compulsive disorder patients treated with exposure and response prevention, change in metacognitions was the best predictor of improvement (Solem, Håland, Vogel, Hansen, & Wells, 2009). There is also some early basic research on depressive rumination and metacognitions, which metacognitive therapy rests on (Papageorgiou & Wells, 1999b, 2002, 2004b). Nevertheless, more treatment-specific process research is necessary to address what specific ingredients (and how they work) make metacognitive therapy effective in treating depression in the reported trials.

In addition to process research, one may consider elements of treatment, as the treatment is delivered, to identify what may or may not explain change. To be certain that the protocol therapists are delivering the treatment according to the manual, it is necessary to implement adherence measures. Few of the current studies have published such data. With this caveat, though, the current findings do suggest that there is something new being delivered in the metacognitive therapy treatment package.

As noted, metacognitive therapy does not engage with content as cognitive behavioral therapy would. There is therefore no focus on current problems, but on the rumination process whatever the content or nature of current problems. Thus, the patients' problems are not an issue for the treatment and no time is spent problem solving. Rather, rumination is deemed unable to solve problems, thus challenging positive metacognitions about rumination being an efficient problem-solving strategy, but then rather than suggesting how to solve problems the therapist will refocus the patient toward detached mindfulness and discontinuing rumination. Patients therefore do not improve or experience symptom reduction due to therapy solving problems.

Metacognitive therapy is counterintuitive. The analytical rumination hypothesis is intuitive to depressed patients; these patients share almost all features and tenets of the analytical rumination hypothesis. In order to treat depression, the analytical

rumination hypothesis recommends increased rumination to solve the problem that elicited the depressive episode and rumination. This is also exactly what most patients believe and do, although one of the major problems they grapple with is the question of why they became depressed in the first place. For the efficient treatment of depression with metacognitive therapy, one needs to socialize the patient to the opposite treatment model. The patient is not solving problems, just engaging in mental problem-focused spinning activity, which reduces mood and consolidates negative metacognitions. Therefore, the patients are taught how to change their intuitive attitudes and beliefs about rumination, and thus how to discontinue this activity. Given that metacognitive therapy shows unusually large treatment effect sizes, this suggests that engaging in depressive rumination is not only unnecessary and unhelpful, but also controllable, but not harmful, despite maintaining depressive symptoms in the long term.

Discussion: Is Rumination Adaptive?

The contrasting claims of the analytical rumination hypothesis and metacognitive therapy put depressed individuals like Emma in a precarious situation. If she consults a proponent of the analytical rumination hypothesis, she might be relieved to learn that her intuitions are true: She should embrace her rumination, because it helps her understand why she is depressed, and ultimately will solve any problem that triggered her depression. If, however, she consulted a metacognitive therapist, she would learn the opposite: Ruminating does not help her gain insight or solve problems, and she should undergo treatment to learn how to stop ruminating. This divide between clinical and evolutionary psychology is unfortunate, and should be resolved by careful examination of the data.

A characteristic of adaptations is that they are reliably triggered by the problems that they evolved to solve. As such, if depression evolved to solve complex problems, we would expect complex problems to reliably trigger depressive episodes. Depressed people do seem to suffer more complex problems than others, according to self-report measures (Lyubomirsky, Tucker, Caldwell, & Berg, 1999). As Andrews and Thomson (2009) note, social conflicts are associated with higher levels of depression (Hammen, 1992), especially if the conflict is with close friends (Antonucci, Akiyama, & Lansford, 1998). In addition, being unhappily married greatly increases the risk of depression (Weissman, 1987). Furthermore, Kendler, Karkowski, and Prescott (1999) found a causal relationship between stressful life events and onset of the first few episodes of major depression in a large-sample study on female twins. However, roughly one-third of the association between stressful life events and depression seems to be due to genetic factors that predispose one to both depression and the experience of stressful life events (Kendler et al., 1999), and among those that experience stressful life events only some will become depressed (Kessler, 1997). In addition, this association disappears after the third depressive episode (Kendler et al., 1999). To the degree that one assumes that social conflicts cause depression, there is still significant variability

in people's susceptibility to major depression. One key factor that predisposes people to depression is their ruminative response style (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema, 1987, 2000; Nolen-Hoeksema & Morrow, 1991). For example, Nolen-Hoeksema and Morrow (1991) measured emotional health and styles of responding to negative moods before and after a major earthquake. Those that typically responded to negative moods by ruminating before the earthquake also had a higher incidence of major depression 7 weeks later. This is the opposite of what one would expect from the perspective of the analytical rumination hypothesis: Because depressive rumination is posited to effectively solve the triggering problem, having a ruminative response style should lead to a quicker resolution of problems, and a shorter depressive episode (Varga, 2012). In addition, a history of depressive episodes is a major predictor of future depressive episodes, and any subsequent episode of depression increases the odds of recurrence (Solomon et al., 2000). From the analytical rumination hypothesis point of view, this would mean that those who experience complex problems for which depression is needed are increasingly more likely to experience such problems again in the future. The recurrent nature of major depressive disorder does not seem to fit with the analytical rumination hypothesis model, considering that Andrews and Thomson (2009) argue that avoiding future avoidable stressors is part of the function of depressive rumination (Kleppestø, 2014; Varga, 2012). Additionally, an even greater predictor is a family history of depression (Sullivan, Neale, & Kendler, 2000). This suggests a large heritable component, which speaks against an adaptationist explanation of major depression (Nettle, 2004). Why does this heritable variation continue to exist? An adaptationist perspective would suggest that depression is universal, and as such would fail to show heritable variation (because everyone shares the genetic variants). The other adaptive possibility is that evolutionary forces such as negative frequency-dependent selection maintain the genetic variants involved in depression. However, major depressive disorder is a highly heritable disorder and *increases* in prevalence as a function of degree of genetic inbreeding in populations (Rudan et al., 2003). This is consistent with major depression being maintained by the constant influx of genetic mutations with overall small effects on the phenotype, rather than being a complex adaptation maintained by frequency-dependent selection or any other force of balancing-selection (For discussion see Keller, 2008; Keller & Miller, 2006).

Further, the analytical rumination hypothesis does not directly explain an important aspect of depression, namely its mortality rate including complications associated with cardiovascular disorder and suicide: In a meta-analytic review Van der Kooy et al. (2007) found that clinically diagnosed major depression was a substantial risk factor for cardiovascular disease, rivaling that of smoking and diabetes. Although the mechanisms are unclear, it is plausible that behavioral factors such as reduced physical activity, smoking, and reduced treatment adherence, in combination with physiological changes associated with depression, can account for the increased mortality (Lett et al., 2004). Somewhere between 2.2 and 8.6% of people diagnosed with affective disorder commit suicide (Bostwick & Pankratz, 2000). This suggests serious pathology. If it is not, it must be argued either that the net fitness benefits of major depression outweigh this mortality, or one would need to

argue that suicide is also adaptive, as some have (e.g., de Catanzaro, 1995). Either way, a comprehensive adaptationist theory of depression needs to address the effects of self-harm and suicide.

The most important claim of the analytical rumination hypothesis is that prolonged rumination effectively solves complex fitness-relevant problems. It follows from this claim that treatments focused on discontinuing ruminating behavior should be ineffective. Reviewing results from clinical trials on metacognitive therapy indicate that the opposite is the case. There is no evidence that discontinuing rumination has any harmful effects, and patients treated with metacognitive therapy show remarkable improvements that are maintained at 6-, 12-, and 24-month follow-ups. This treatment is based on theoretical underpinnings that view depressive rumination to be wholly maladaptive (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008; Wells & Matthews, 1996) and its central goal is to discontinue rumination. The fact that people suffering from major depressive disorder are effectively treated with metacognitive therapy strongly suggests that depressive rumination, and indeed major depressive disorder, is maladaptive.

From an evolutionary perspective to psychopathology, the current findings suggest the following: It is important for evolutionary psychologists to stay abreast with developments in mainstream clinical psychology and of course vice versa. Fields that become too insular suffer from losing touch with broader scientific developments. For years, the field of evolutionary psychology has been arguing against accusations of panadaptationism and panselectionism (Kennair, 2002). Although adaptationist models are highly testable, and provide original hypotheses, a scientific field needs to be tempered by empirical findings. The current review of data from treatment trials and basic research within mainstream clinical psychology suggests that the adaptationist approach to rumination may be dovetailing with metacognitions many depression patients have about their cognitive processing: metacognitions that maintain their depressive symptoms, and prolong their suffering. Entertaining such metacognitions and arguing for exacerbating rumination and depressive symptoms would, in light of the current findings, be considered foolhardy. On the other hand, it could be that long-term effects of rumination are better matched to better social functioning, improved navigation of social hierarchies, healthier relationships, or similar evolutionarily relevant measures of well-being, condition, and coping in life.

We also run into the problem of recurring episodes. Depression may appear as a single episode, but many people have recurring periods of depression. Is this due to negative processing causing illness or is it because these people have several complex social problems in their lives that need solving? The latter would be more convincing if all the episodes had clear precipitating negative life events that most people would start ruminating after. Note that it is not ruminating, in general, but engaging in depressive brooding that maintains depression. It seems that most people do not ruminate after negative life events (Larsen, Kleppestø, Bendixen, Buss, & Kennair, 2017), and far from everyone gets depressed after negative life events (Bonanno, 2004; Caspi et al., 2003). Thus, ruminating after events might not be a human universal; and brooding certainly is not. In addition, one might point out that when searching for a solution there are many different forms of ruminative and problem-solving mental behaviors that are possible, from those that coldly and

without affect-activation solve the problem via action rumination to those that react with brooding. Nevertheless, among these many types of mental processing, brooding might not be a problem-solving type of processing. When we solve problems, we do not engage in procrastination and we do not merely consider the downsides, negative aspects, or sadness-inducing aspects of the problem; rather than focusing on the problem, we focus on the solution. The problem might be that, due to the maintaining aspects of the cognitive attentional syndrome, the incorrect metacognitions and unhelpful coping behaviors involved in depressive rumination are not exposed. Thus, one continuously returns to unhelpful and symptom-enhancing processing. Currently, more of the evidence supports the metacognitive therapy approach to the question of the adaptiveness of rumination.

Finally, why are there (at least in Western countries) sex differences in rumination? There is still work to be done from an evolutionary perspective into the evolution of rumination and sex-specific patterns of repetitive cognitive processing. Despite the current chapter being critical of a specific evolutionary approach to depression, we are far from negative about an evolutionary approach to mental disorder, in general (Kennair, 2003, 2007, 2011, 2014, 2017). On the contrary, we support and promote that level of analysis, and believe that a functional science of evolutionary psychology is crucial for a better understanding of psychopathology. This is especially the case given that one of the best definitions of mental disorder, Wakefield's (1992, 1999) harmful dysfunction, demands that we know when and how evolved mental mechanisms are functioning (Kennair, 2011).

Conclusions

Is prolonged depression healthy? No. There is increased likelihood for chronification, somatic complications, and neurocognitive problems in the wake of prolonged depressive episodes (Hammar & Ardal, 2009; Hammar, Lund, & Hugdahl, 2003). Is discontinuing rumination helpful? First, it is possible to teach patients how to discontinue rumination. Second, the findings above suggest that reducing rumination is associated with reduced symptoms of depressive disorder. However, if the analytical rumination hypothesis is that depressive symptoms are beneficial, the case is not closed by curing depression: May there still be long-term beneficial effects of rumination? Maybe. However, note that follow-up data suggest that patients improve or stay better over time. The analytical rumination framework therefore needs to predict and test specific negative long-term effects of treatment. Patients with prolonged and/or recurrent depressive episodes will need to show beneficial and predictable life trajectories on fitness-relevant aspects compared to patients that have been treated effectively. This would be relevant irrespective of the mode of treatment, but it would be especially interesting to test patients treated effectively with rumination discontinuing interventions—as these will probably curtail general rumination and should therefore show the most fitness-reduced long-term trajectories. In the meantime, the long-term data does not suggest that this is anything but speculation.

One complicating matter here is that depression is generally a recurrent disorder or state. It will reappear, and after the first few episodes, it seems that there no longer is the need for a precipitating negative life event. The first or second depressive episode often follows a negative life event, but most people do not get depressed due to similar stressors. This is again reason to question the universality of rumination and depression as solutions to social problems. On the other hand, as there are no studies that investigate whether depressed patients do come up with solutions to these life problems compared to patients who have received efficient therapy, it is too early to conclude with certainty.

One may not turn this argument around, though; absence of evidence either way is not an argument for a strong claim. The strongest arguments currently suggest that depression causes individual suffering and complications over time. Further, the societal costs are staggering worldwide. To boot, there is no evidence of long-term benefits. Given the results from treatment trials with metacognitive therapy, it would be ethically dubious to suggest that patients should increase rumination. There is no evidence that this would solve complex social problems, and no reason from any basic research on rumination and depression to suggest that increased or continued rumination will reduce depressive symptoms.

To conclude, current research on treatment suggests that discontinuing rumination is possible and brings about clear reduction in depressive symptoms. Metacognitive therapy may therefore currently be considered a promising treatment of depression. The analytical rumination hypothesis's holdout would therefore be for the long run: Future studies on depression would need to show that brooding and rumination increase fitness or at least condition and well-being in evolutionarily relevant ways, despite the negative emotional states and other consequences of depressive disorder.

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The Evolution of Social Anxiety

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"Understanding the evolutionary history of a trait, how it gives a selective advantage, and the costs it imposes can help to illuminate its design and regulation and can guide research into its mechanisms and control." (Nesse et al., 2007; p. 965).

Introduction

Social relationships constitute a highly rewarding context for most people, providing a source of support and nurturance, as well as protection against loneliness, depression, and even death (Cacioppo, Hawkley, & Thisted, 2010; Cohen, 2004; Steptoe, Shankar, Demakakos, & Wardle, 2013). Interpersonal relationships can also, however, be stressful. They are marked by periodic conflict with others and entail inherent risks of negative evaluation or criticism (Bertera, 2005; Rook, 1984). Further, social strains contribute to psychological and physical health problems (Seeman, Gruenewald, Cohen, Williams, & Matthews, 2014; Yang, Schorpp, &

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Harris, 2014). It is thus not surprising that interpersonal difficulties constitute one of the most common reasons that people seek psychological treatment (Bankoff, 1994; Benton, Robertson, Tseng, Newton, & Benton, 2003; Pledge, Lapan, Heppner, Kivlighan, & Roehlke, 1998). One particularly common manifestation of such difficulties is social anxiety (SA), defined as an excessive fear of negative evaluation that can lead people to avoid social engagement. Its associated behavior patterns may result, in the most severe cases, in a clinical diagnosis of social anxiety disorder[1] (SAD, formerly called social phobia; APA, 2013). Indeed, this disorder is common. Approximately 7–8% of adults meet the criteria for SAD in a given year (Kessler, Chiu, Demler, & Walters, 2005), and an additional 10–11% have at least some impairing symptoms (Fehm, Beesdo, Jacobi, & Fiedler, 2008).

Not surprisingly, given both its ubiquity and its negative effects, SA has been the focus of much research. Despite this work, it remains difficult both to predict who will develop the condition and, for many affected individuals, to treat SA successfully. Indeed, although researchers have identified a range of genetic, temperamental, and environmental risk factors (Bas-Hoogendam et al., 2016; Spence & Rapee, 2016), we do not fully understand how these interact to precipitate SA. Further, even when gold standard treatments, such as cognitive behavioral psychotherapy, are applied reliably, roughly half of the adults with SA fail to show a meaningful response (Loerinc et al., 2015).

In recent years, due at least in part to frustration with persistent limits to their ability to predict and treat SA, researchers have begun to explore alternate approaches to understanding this condition. Of these, the most relevant for our purposes is the hypothesis that, although SA certainly has detrimental effects that require treatment, it is an adaptive behavior pattern that has been selected for the benefits that it may have provided and that continue to be relevant. In other words, SA may be, at its core, a behavior pattern that was selected in our evolutionary history because it has useful properties, rather than a psychopathology stemming from novel cultural expectations and environmental factors in modern Western environments. If this is the case, then understanding why this suite of traits was under positive selection pressure not only would provide a better understanding of when to expect SA symptoms (i.e., those that match the conditions under which there was positive selective pressure), but also may help us decide when treatment is needed and, when indicated, to more successfully treat severe manifestations (i.e., by eliminating or modifying the core circumstances that elicit symptoms, as opposed to just treating the symptoms themselves). Indeed, this approach has coincided with a general appreciation for the idea that considering the evolutionary history of behavior helps us to better understand the trait in question. As is evident in this volume, there has been substantial interest in the evolutionary history of psychopathology, in general, and more specifically in the possibility that psychopathology may be adaptive in some circumstances (Brüne, 2015; McGuire & Troisi, 1998).

[1] Throughout the text, we refer to social anxiety, or SA, by which we mean the cluster of anxiety-related symptoms, but do not mean that there is a clinical diagnosis unless otherwise specified.

In this chapter, we first summarize the history of thought surrounding SA, and then outline the evolutionary frameworks that scholars have proposed. We next discuss the ways in which recent work on social behavior in animals, particularly other primates, affects our understanding of these models. In particular, although the original models focused on aggression and dominance as key issues in eliciting interpersonal difficulties, more recent work emphasizes the formation and maintenance of peaceful social relationships and highlights situations in which it may be to an individual's substantial advantage to hesitate in social circumstances. Indeed, new research demonstrates that there are times when appeasing or avoiding is highly beneficial. Building from this more recent work in primates, we outline an updated model. We include a series of open questions around SA, and its evolutionary history, that will need to be answered to better prediction and treatment of SA.

Just because it's unpleasant doesn't mean it's pathological: The Importance of an Evolutionary Approach to Psychopathology.

> *"The ubiquity of the illusion that defenses are abnormalities arises from several sources. First, defenses are often associated with some kind of suffering and therefore seem maladaptive. Unfortunately, however, discomfort is itself probably one aspect of a mechanism that makes it useful. Second, they are reliably associated with disadvantageous situations, so the association bias makes it seem as if they are the problem. Finally, it is possible to use drugs to block the expression of many defenses with very little harm, completing the illusion that defenses are useless." (Nesse & Young, 2000, p. 79).*

Psychopathologies, including SA, are not fun. They feel bad, and commonly occur in situations that we did not want to be in to begin with. People wish the symptoms to end, which is sometimes possible with easily obtained and well-tolerated medications. As a result, we tend to focus on the resulting *symptoms*, rather than their causes. We act as if the symptoms alone are the problem, rather than also recognizing them as *expressions* of the actual problem. But why is this an important distinction? If we can decrease symptoms by simply taking a pill, why not do it?

First of all, because it is not always so easy to relieve psychological problems. Despite substantial research into treatments (medication and other) for psychopathologies, and evidence that these treatments have lasting benefits for a substantial proportion of people, they do not work well, or at all, for many others. In a given year, roughly 60% of people with SA do not receive treatment at all. Of those who do get help, only about a third get treatment that has been shown in clinical trials to work well (Wang et al., 2005). Even medications and other first-line interventions, such as cognitive behavioral therapy, fail to relieve symptoms in at least half of the treated cases, and cure even fewer than that (Loerinc et al., 2015; Ravindran & Stein, 2010).

Second, without effective treatment, there is a high likelihood of developing more debilitating symptoms (Wittchen, 1998; Wittchen & Fehm, 2003). As an example in the physical realm, imagine that you are a marathon runner and you develop a stress fracture. You have qualified for a major race, so you choose to take painkillers and run through the pain. There are two likely outcomes. First, you probably will not do well in your race, due to the fact that your body is not functioning

at its full level of ability. Second, you will probably make the injury worse, possibly resulting in a long period while you recover in which running is impossible. The same is true in the psychological realm. Attempting to soothe symptoms with medication may provide temporary relief, but if the root causes of symptoms are not addressed, they will continue to cause problems, and may indeed cause even worse ones. This is perhaps the most devastating effect because, as Nesse indicates in the quote above, efforts to relieve distress can hide the real problem.

So how does considering SA from an evolutionary perspective address these issues? If one approaches SA with a focus on how it may be adaptive, one can reconceptualize the condition as serving both useful and impairing functions, rather than simply the latter. Clinicians can then work collaboratively with affected people to identify the sources of anxiety symptoms and to develop treatment plans that take these into account and, potentially, address them along with symptoms. Although attention to the contexts in which symptoms arise is common in psychotherapy, particularly CBT (Mennin, Ellard, Fresco, & Gross, 2013), an evolutionary approach is grounded in comparative data that can be used to generate a road map of likely causes, that is, situations and contexts that lead to an increase in anxiety-related symptoms in other species. An evolutionarily informed perspective regarding the social circumstances that trigger anxiety and why they might do so can assist the practitioner in predicting the onset of symptoms and determining if they reflect adaptive or maladaptive processes.

But how does one do this effectively? An evolutionary approach only works if it is evidence based. So-called Just So Stories—in which creative but unsupported causes are hypothesized for selective pressures behind traits—are ineffective and, because they misdirect research and treatment, possibly dangerous. Instead, any such hypotheses need to be empirically grounded and rigorously tested. This is challenging if one is looking only at people in a modern, Western world that may not particularly resemble the environment in which humans evolved (Henrich, Heine, & Norenzayan, 2010). Indeed, it is virtually impossible to come up with good hypotheses without having broad insight into environmental and social pressures across multiple groups. One solution is to look across cultures, to find underlying commonalities that may indicate shared causes (e.g., Dinnel, Kleinknecht, & Tanaka-Matsumi, 2002). Although useful, even this approach limits research to shared causes within humans, and may mask fruitful avenues that are shared more widely across the animal kingdom. Thus, the comparative approach, in which we look at other species for insight into our own behaviors, holds distinctive value.

Indeed, the comparative approach has been immensely useful in generating insight into the evolution of a wide variety of human social and cognitive behaviors (Brosnan & de Waal, 2014; DeAngelo & Brosnan, 2013; Kappeler & Van Schaik, 2005; Kornell, 2009; Wiley, 1983), as well as human physical health (Merlo, Pepper, Reid, & Maley, 2006; Nesse et al., 2010; Williams & Nesse, 1991) and mental health (Brüne, 2004, 2015; Crow, 1995; Grinde, 2005; McGuire & Troisi, 1998; Nesse, 1998; Nesse & Young, 2000). An evolutionary perspective offers a new way of thinking about behaviors that too often seem only problematic (i.e., morning sickness is unpleasant, but may protect the fetus; Flaxman & Sherman, 2000). Such

a view may not only help those with SA understand their suffering in a more nuanced and accurate way, but also may lead to novel interventions that, as we argued previously, can take into account and address the roots of symptoms rather than simply provide a "Band-Aid" to fix them. Finally, although much has changed in humans' evolutionary trajectory since our lineage split from the other great apes, we nonetheless share many of the same issues. Like us, other primates are highly social, highly reliant on their friendships for their health, well-being, and longevity, and suffer from a suite of anxiety behaviors that are ameliorated by changing circumstances and, in some situations, drugs, indicating the possibility of similar underlying mechanisms.

Below we expand on these ideas and argue that by considering the evolutionary angle we may be better able to thoroughly define the contexts in which SA symptoms arise and why they do so. Specifically, understanding the settings in which SA occurs, and the selective pressures that led to it, may help us to provide a more precise, evidence-based diagnostic criterion for when the experience of SA reaches a clinical threshold and when it is "normal." Critically, this means both better understanding of how to treat SA and insight into *when SA might be worth not treating, or even fostering*. This latter perspective is often underemphasized in approaches to psychiatric disorders, yet it could be an important one for appropriately targeting and tailoring interventions.

The Benefits of Studying SA in Nonhuman Species

Humans are not the only species that experience anxiety; indeed, if we are to explore the evolutionary correlates of SA comparatively it is critical to be able to identify SA-like behavior in other species. Much work demonstrates that other primates show anxiety-related symptoms, in situations that, from a human perspective, appear to be stressful. Further, these symptoms can be alleviated using the same medications that humans use (implying the same, or at least similar, underlying mechanisms). A review from 2002 identifies almost 70 studies on fear and/or anxiety in nonhuman primates (primarily rhesus macaques, *Macaca mulatta*, squirrel monkeys, *Saimiri spp.*, and common marmosets, *Callithrix jacchus*, although other model species are also mentioned; Barros & Tomaz, 2002). Tests ranged from traditional conflict tests to more ethologically appropriate models (social interaction, involuntary isolation) and found that in about half of these anxiolytic drugs decreased anxiety-related symptoms. This is a relatively widespread effect, and argues for the utility of studying anxiety in other primates. The effectiveness of the anxiolytic drugs also hints at a common underlying mechanism. Indeed, research on macaques shows that selective bilateral lesion of the amygdala led to a lack of both fear and social inhibition in rhesus macaques, just as it does in humans (Amaral, 2002), indicating a similar neurobiology.

Conveniently for such work, the key behavioral measure for primate anxiety comprises displacement activities (i.e., self-grooming, self-scratching, body-shaking,

yawning; Gabriele Schino, Perretta, Taglioni, Monaco, & Troisi, 1996). Such behaviors are easy to observe and therefore function as a noninvasive assay for anxiety. Indeed, numerous studies have used self-directed behaviors (SDB) to index distress in social contexts. For example, in wild olive baboons, females increase SDBs by approximately 40% when the nearest neighbor is dominant to them as compared to when the nearest neighbor is subordinate (Castles, Whiten, & Aureli, 1999). Rough self-scratching has been used to assess social anxiety in wild chimpanzees, and research indicates that there is a sex difference; among males, performance of this SDB was related to rank order (higher ranking males performed less scratching), but in females, social proximity, rather than low rank, increases SDBs. In neither sex, however, did the rank of individuals in social proximity influence SDB rate (Kutsukake, 2003). Chimpanzees also show SDBs during difficult tasks, although interestingly the increase occurs only when the task becomes harder, and no decrease is seen when subjects start with a hard version that then becomes easier (Leavens, Aureli, Hopkins, & Hyatt, 2001). However, care must be taken to disentangle anxiety from other environmental factors. A recent study of Japanese macaques indicates that one SDB, self-scratching, is most closely related to environmental factors (lice load), whereas another, self-grooming, may be more closely related to displacement behavior in the context of social uncertainty (Duboscq, Romano, Sueur, & MacIntosh, 2016). Although this balance will vary across contexts and species, it is important to consider other causes of these displacement behaviors. When possible, for example, researchers can correlate changes in SDBs with changes in stress-related hormones (i.e., cortisol).

Of course, the clear advantage of SDBs as a behavioral assay for studying SA in primates is that they appear to be a relatively consistent measure of anxiety in uncertain contexts (social and nonsocial) that can be assessed without interfering with ongoing activity. This means that an SDB assay is appropriate for ongoing social interactions, which it might not be desirable (or possible) to interrupt in order to obtain a physiological measure or a sample for hormonal assay. SDB assays are also good options for studying social behavior and distress in wild populations where more invasive interventions may not be possible or ethical. Indeed, these situations are exactly the ones that we need to know more about, if our interest is in understanding the evolution of SA. It is key to know the natural contexts in which SDBs occur, as well as the impact of the social environment, neither of which is amenable to studies of isolated individuals.

Aside from providing data about the evolutionary history of SA, these studies strongly support nonhuman primates as an excellent model for human anxiety-related behavior. There are multiple advantages to studying questions about SA in other species. Practically speaking, in an ideal circumstance we can use a model species to ask questions that are impractical or impossible, or are unethical, to ask with humans. For example, it is difficult and expensive to get high-quality, longitudinal data on humans. However, there are primate social groups that have been studied for decades, and whose entire social history, including the frequency of both positive and negative social interactions, personality, health, and kinship, has been documented for the whole social group (most of these social groups are closed,

meaning that we also do not have to contend with the influences of extraneous relationships). Such detailed long-term data about individuals and their social relationships are virtually impossible to obtain in humans, and yet may be key in unlocking the causes of SA. In studies of nonhuman primates, we can also manipulate social environments (e.g., by adding or removing individuals), which allows us to directly test hypotheses about the influence of various factors on anxiety and also to determine whether and why some individuals are more resilient in the face of social stress than others.

Additionally, although interviews with humans give insight into what they are thinking and feeling, such interviews may also be misleading, as people inadvertently or intentionally misreport motivations and behaviors (Liang, 1988). Studying animals by necessity focuses the inquiry to actual observed behaviors, which may make it easier to observe unexpected behaviors or contexts that can be used to develop predictions in humans (Brosnan, Newton-Fisher, & van Vugt, 2009). Although of course this can be done in humans, in animals there is no other way, which may lead to creative ways to "ask" questions through observational studies. Finally, in nonhuman primates, we can gain access to physiological data that may be difficult to obtain from humans. For example, cortisol or testosterone levels can be obtained simply and noninvasively from primate social groups, and both can be used to explore everything from the endocrinology of cooperative interaction to social stress related to dominance interactions (Trumble, Jaeggi, & Gurven, 2015; Wheeler, Tiddi, & Heistermann, 2014).

Building an Evolutionary Model of Social Anxiety

One of the primary motivating factors for human behavior is a need to belong, or to form relationships with others that are strong and enduring (e.g., Baumeister & Leary, 1995). As Baumeister and Leary (1995) specified, this need to belong is distinct from an indiscriminate need for social interchange; instead, it constitutes a "need for regular social contact with those to whom one feels connected" (p. 501). From an evolutionary perspective, such a powerful drive for consistent connection with valued others makes sense, given that individuals embedded in mutually supportive social networks should have preferential access to resources needed for survival and reproduction (Leary, 2001). Indeed, other primates also show such close ties, and new evidence shows unequivocally that, as with humans, relationship quantity and quality affect everything from longevity to offspring survival (Palombit, 2001; Seyfarth & Cheney, 2012; Silk, 2007; Silk, Alberts, & Altmann, 2003, 2006; Silk, Seyfarth, & Cheney, 2016; Silk et al., 2010; Smuts, 1985).

Not surprisingly, then, it is common for humans to experience social anxiety, or fears that others may evaluate them negatively and consequently ridicule or reject them—such experiences signal risk for exclusion from valued resources or for loss of rank or status (Leary, 2001). Consequently, when they feel socially anxious, people behave in ways that presumably help them to avoid such unpleasant outcomes

(Rinck et al., 2010). Such avoidant patterns commonly encompass deferential, withdrawn, and a broad range of other "safety" behaviors (Cuming et al., 2009).

The term "social anxiety" is commonly used in the psychological literature to refer to extreme manifestations of fear of negative evaluation and its associated behavior patterns. When SA leads to lasting distress across a broad spectrum of social settings, it can impede professional and academic advancement, prevent healthy relationship formation, and increase risk for substance abuse and depression (Ruscio et al., 2008; Schneier, Johnson, Hornig, Leibowitz, & Weissman, 1992; Stein & Kean, 2000). Such impairing symptoms cluster under the diagnosis of SAD, which is among the most common of all psychological disorders (Kessler et al., 2005). For individuals with severe or clinically significant SA, fears of rejection or negative evaluation are reinforced and perpetuated by cognitive biases to interpret ambiguous social cues through a negative lens and to assume that aversive social outcomes are highly probable and, when they occur, costly in terms of their likely effects on status and connection (Foa & Kozak, 1986; Stopa & Clark, 2000).

However, although the behaviors associated with SA can be problematic if selected in excess or in contexts in which alternate responses would lead to better outcomes, each is also a normal part of a healthy social repertoire. Deference and withdrawal, for instance, can confer advantages in some situations, by decreasing the risk that one will enact antisocial or otherwise inappropriate behaviors (Hermans & van Honk, 2006) or enabling one to escape from conflicts with dominant others (Weeks, Rodebaugh, Heimberg, Norton, & Jakatdar, 2009). Thus, transient and/or mild social fear, which most people experience to varying degrees in response to real social threats or challenges, likely has adaptive value.

Clinical Models of SA

Researchers have proposed numerous models to explain how and why SA develops and persists among humans (Clark & Wells, 1995; Heimberg, Brozovich, & Rapee, 2010; Hofmann, 2007; Moscovitch, 2009; Rapee & Heimberg, 1997; Schlenker & Leary, 1982). Schlenker and Leary (1982) were among the first to propose a conceptually integrated model, which centered on the idea that SA arises when people want to make good impressions on others, but lack confidence that they will be successful in doing so. This self-presentation model, which is rooted in social learning and interpersonal theories, shifted the focus from a traditional, psychodynamic view of "anxiety as drive" to a perspective in which anxiety is a constellation of emotional, cognitive, and behavioral responses that arise from a mismatch between perceived abilities and desired outcomes (Schlenker & Leary, 1982, 1985).

In the 1990s, researchers proposed two additional models, both of which revolved around the roles of attention and perception in the generation and maintenance of SA (Clark & Wells, 1995; Rapee & Heimberg, 1997). These distinct, but compatible, models both emphasize the role of attention to cues indicating social threat in both precipitating and perpetuating socially anxious behaviors and feelings,

particularly in people who assume that others will be critical and will hold them to unrealistically high behavioral expectations. Clark and Wells' (1995) model is built around the idea that signals of threat that are internal to the self (e.g., aroused autonomic responses in social settings) preferentially capture socially anxious people's attention. In other words, SA results when attention to the self is enhanced and processing of cues from the external world that might disconfirm social fears is diminished. In Rapee and Heimberg's model (Heimberg et al., 2010; Rapee & Heimberg, 1997), attention to both internal and external cues that social threats are present is considered important for the emergence and perpetuation of SA. Thus, socially anxious people persistently scan their internal and external environments for evidence that they are performing poorly and will be judged harshly or that they performed well, which could set the performance bar even higher. Further, this model proposes that a tendency to dwell ruminatively and vividly on negative aspects of behavior after social events increases the likelihood that socially anxious people will view themselves through a biased lens. Note that both self-presentational and cognitive models of SA are compatible with evolutionary perspectives.

Evolutionary Models of SA

In his presidential address to the Society for Psychophysiological Research in 1985, Öhman (1986) sketched out a model of SA that underscored the idea that this type of anxiety emerged as a function of specific biological pressures. In laying groundwork for his model, Öhman (1986) contrasted human responses to escaping an angry conspecific by running away with those to escaping a wild animal by running away. He argued that whereas the escape from a wild predator might lead to relief, the escape from an angry human might instead elicit shame or humiliation. An evolutionary perspective that takes into account the distinct behavioral systems that drive these two distinct responses to frightening stimuli provides one way to understand why fear of people might have evolved. In particular, Öhman (1986) contended that social fear constitutes the opposite pole of social dominance and that individuals within a group engage in complementary interactions distributed along this dimension in order to establish and maintain a stable social hierarchy that ensures social order. Within these interactions, social submissiveness serves to "avert attack by dominant conspecifics" (Öhman, 1986, p. 130).

In a more thoroughly defined evolutionary model of SA, Gilbert and colleagues (Gilbert, 2014; Trower & Gilbert, 1989) proposed, broadly, that a socially anxious pattern of emotional and behavioral response constitutes a vestigial response to social threat. Within this model, competition for social status engenders a particularly salient type of threat—status loss—and those with high levels of SA are considered to be acutely attuned to indicators that such loss is impending. Gilbert and colleagues' model is rooted in defense approaches that are adaptive in the context of social groups that revolve primarily around hierarchy, like those observed in some other species and much like those based in the dominance/submission system

around which Öhman's model is built. More specifically, Gilbert and colleagues argue that humans with SA operate interpersonally from a competitive stance that would be better suited to a purely hierarchical context. They thus fail to engage with others in friendly and affiliative ways that might actually better serve their goals of belonging, or being included and valued.

More recently, Gilboa-Schechtman, Shachar, and Helpman (2014) proposed an evolutionary model that resembles Gilbert and colleagues' model in that it encompasses both social rank and affiliative elements. Grounded in research on the expression and processing of emotional cues that signal warmth or dominance, this model draws upon Leary's (2001) idea that SA may alert individuals to threats to their integration within a valued social group. According to this model, fears of both exclusion and defeat are salient for the socially anxious person. Gilboa-Schechtman and colleagues contend that these fears may be adaptive in the context of unstable or volatile hierarchies, but in more stable and cohesive communities they may lead to problematic behavior.

Notably, these models are grounded largely in research on humans; although Gilbert draws upon the nonhuman primate research literature, he and his colleagues focus primarily on studies of dominant/submissive behavior in other species, with less attention devoted to research on affiliation and other types of positive interaction. If we aspire to develop a thorough and fully accurate evolutionary model of SA, it is important that we integrate up-to-date research on both humans and related species and take account of the ways that new findings shift our understanding.

Challenges to Existing Evolutionary SA Models

The Reality of Primate Conflict Behavior

One key problem with the current evolutionary models of SA is an incomplete view of primate behavior. As noted earlier, only Gilbert and colleagues' model draws heavily from research on nonhuman primates; however, their model was originally developed in the late 1980s. In the intervening 30 years, there has been a fundamental shift in how primate social behavior is perceived, which is not reflected in this model. At the time they developed it, research on nonhuman animals was primarily focused on aggression and striving for dominance, and little interest or thought was given to other aspects of social behavior. Around this time, researchers began to focus on more "positive" aspects of social behavior, demonstrating that animals maintain long-term, and apparently valuable, friendships; that these friendships enhance longevity and health; and that although conflicts certainly do occur, the animals work hard to repair and maintain their beneficial relationships.

One of the first well-known discussions of this shift in perspective came from Frans de Waal, an influential Dutch biologist. In the early 1980s, he published years' worth of observations of a large, multi-male, multi-female captive colony of chimpanzees at Berger's Zoo in Arnhem, the Netherlands. His report, entitled *Chimpanzee*

Politics, showed a markedly different side of these apes' behavior (1982). They were, indeed, focused on dominance, particularly the males, but dominance was not achieved by sheer force; instead, the apes developed a series of alliances that they carefully manipulated to gain and maintain power. In a dramatic example of this, when the leading males' coalition collapsed, within a day, the third male—who was individually stronger than either of the coalition partners—became the alpha.

As has gradually become clear, relationships are critical in nonhuman primates (Silk et al., 2006; Smuts, 1985). In most species, individuals begin life with a set of relationships derived from their mothers' social networks. These relationships are not static, however; individuals maintain these connections and develop new ones through grooming and other prosocial (i.e., positive) behaviors that strengthen social ties. Moreover, having such positive relationships is critical. For example, data from long-term field studies with baboons demonstrate that primates with more social ties are longer lived and have better infant survival than those with fewer such ties (Silk et al., 2003, 2010). Other evidence shows that nonhuman primates work hard to nurture their relationships; for example, baboons use vocalizations flexibly to build and maintain connections to others (Silk et al., 2016). This focus on building and maintaining *positive* relationships is different from the focus on aggression and dominance as the key to understanding primate social behavior that was predominant when Gilbert and Trower were developing their model.

Moreover, even the dominance hierarchy itself functions differently than research in the 1980s suggested. At that point, the hierarchy was conceived as a mechanism by which more dominant individuals were able to control access to resources (i.e., food, mates, sleeping sites) at the expense of more subordinate individuals. Of course, that is true to some extent. However, the degree to which it is true varies depending on the social structure of the species in question. Some species, like rhesus macaques, have strict, linear, despotic dominance hierarchies in which the more dominant can take anything that they wish. Others, however, such as chimpanzees, have much more relaxed hierarchies that both lack strict linearity (dominance relationships often depend on which other individuals are present) and are much more tolerant. In chimpanzees and capuchin monkeys, another tolerant species, subordinates can expect to maintain possession of resources. In chimpanzees, there is impartial support in conflicts, especially by high-ranking individuals breaking up fights among others (von Rohr et al., 2012). Indeed, even in the more despotic social systems we see policing, in which dominant individuals intervene impartially in fights to restore peace (Flack, de Waal, & Krakauer, 2005; Flack, Girvan, de Waal, & Krakauer, 2006).

In fact, the existence of the hierarchies themselves provides a stabilizing function as a mechanism for reducing conflict in day-to-day interactions among group members. Groups that have a strong, stable dominance hierarchy have few actual fights; incipient conflicts are ameliorated through a series of standardized signals and norms that resolve the conflict in ways that are predictable. These include, for example, unilateral, unambiguous submission signals that indicate that an individual is subordinate; a subordinate who gives such a signal is telling the dominant "I know you are dominant, we don't have to have a fight!" On the other hand, groups with

unstable dominance hierarchies endure repeated fights, lessened grooming, and substantially increased tension. Indeed, observers can predict when a dominance hierarchy is about to shift through increases in aggression and changes in the frequency and direction of these standardized signals (de Waal, 1982). A strong dominance hierarchy also reduces day-to-day aggression, because it obviates the need to fight each day to reestablish the hierarchy. Indeed, it is not *dominance* that leads to fights and conflict, but *uncertainty* about dominance that does so.

Moreover, once there has been conflict, animals work hard to repair relationships; again, it is not the relationships themselves—positive or negative—that appear to cause stress. Rather, it is uncertainty about where the relationship stands, or the risk that a positive relationship will not be repaired following a conflict (it is difficult to distinguish these two underlying motivations[2]). Reconciliation is defined as increased positive contact, as compared to baseline, between two former combatants. It is measured using an approach called the post-conflict/matched-control (PCMC) method (de Waal & van Roosmalen, 1979). In this, subjects are observed for some time period (typically 10–30 min) following a fight (post-conflict) and all instances of affiliation are recorded. Then subjects are recorded for another period of the same duration, at the same time of day, on a subsequent day when there was not a preceding fight in the group. Preferably, this second observation occurs the next day, but if these conditions are not met, it is done on the soonest subsequent day that meets those criteria (matched control). Subjects are considered to show reconciliation if they exhibit increased grooming in the period following conflict as compared to the matched control period. Using this methodology, reconciliation has been found to be widespread in primates (i.e., Cords, 1993; de Waal & Ren, 1988; Palagi, Antonacci, & Norscia, 2008; Pereira, Schill, & Charles, 2000) as well as other highly social nonprimate vertebrates (i.e., Schino, 1998; Wahaj, Guse, & Holekamp, 2001). Some have even argued for consolation, or positive contact with the victim by a third party (Romero, Castellanos, & de Waal, 2010; but see Koski & Sterck, 2007).

Overall, the updated view of primate social behavior focuses heavily on affiliation, not conflict, and on the role of positive relationships in everything from offspring survival to longevity. If anything, the new data emphasize the role of uncertainty in causing stress and anxiety; subjects appear to be most disturbed when

[2] There is an important distinction in evolutionary biology that deserves clarification here. When biologists talk about *evolutionary function* they mean the reasons why a given trait was selected (i.e., the evolutionary history and evolutionary function from Tinbergen's famous four questions; Tinbergen, 1963). An evolutionary mechanism, on the other hand, is the underlying architecture that enables the behavior (i.e., cognitive—Was it learned? Socially or individually?—mechanisms, neural architecture, hormonal underpinnings) and how the behavior developed. A related, but not entirely overlapping, point is that a mechanism does not imply any knowledge or intention. Therefore, if we say that an organism reconciles to reduce the uncertainty in the relationship, this simply mean that animals have been selected to perform certain behavior patterns that reestablish positive bonds, which is beneficial for their long-term fitness and is caused in the short term by the negative affect that accompanies the uncertainty. Note that the organism does not need to understand this or even have any goals; although this could certainly be a part of it, and is in some species, including humans, it need not be for the behavior to function as such.

the relationship is unknown rather than when the relationship is marked by a sharp difference in dominance. Moreover, the behaviors associated with anxiety may function not only to appease, but also to solicit positive contact. The good news is that this revised model of primate social behavior looks substantially more like human behavior than did the previous, despotic view. Further, evidence that primate social behavior more closely resembles that of humans raises the possibility that the evolutionary approach is even more relevant than previously believed. Nonetheless, this new information requires a rethinking of how we perceive the role of relationships and dominance in the evolution of SA.

Nuances of Social Behavior in Human SA

In addition to the deeper understanding that we have acquired regarding primate social behavior, new knowledge about SA has emerged over the past few decades that must be taken into account in evolutionary models. First, whereas early conceptualizations focused on fear of negative evaluation as the core cognitive component of SA, more recent research suggests that people with SA also fear positive evaluation (Weeks & Howell, 2012, 2014). Weeks and colleagues have suggested that those who see themselves as low ranking engage in a reflexive calculus aimed at ensuring not only that they avoid sinking to the bottom of the hierarchy, but also that they avoid *rising* to a status level that invites dominant peers to view them as threats (Weeks & Howell, 2012). Thus, SA may function to help preserve stability of the social hierarchy in both upward and downward directions.

This desire to "fly under the radar" and maintain the social status quo may relate to a second advance in our understanding of SA: as recent studies demonstrate, people with this condition call on a varied repertoire of behaviors to avoid losing or gaining social status. Early research on interpersonal behavior in SA focused on identifying behavioral deficits (Arkowitz et al., 1975), such as difficulty reading or producing social cues (Beatty & Beatty, 2001; Schroeder, 1995), and submissive "safety" behaviors (Daly, 1978; Fromme & Schmidt, 1972; Fugita, 1974). More recently, however, researchers have shifted attention to interpersonal behavior patterns and styles that characterize those with SA, and have used their data to paint a more complex and nuanced picture.

Findings suggest, first and foremost, that a strict focus on submissive actions or social ineptitude fails to capture the full range of social behavior associated with SA. Instead, there appear to be striking variations in interpersonal styles both across and within individuals with SA. Studies show that people with SA cluster into broad stylistic categories, with some interacting in a cold/quarrelsome way (e.g., demanding reassurance with increasing hostility) and others displaying a more friendly/submissive style (Cain, Pincus, & Grosse Holtforth, 2010; Kachin et al., 2001). Failure to show warmth to others appears to be particularly problematic. Rodebaugh and colleagues have found evidence, for example, that such interpersonal constraint, especially when combined with a tendency toward vindictiveness, leads people with SA to show progressively less generosity toward friends over a series of interactions

(Rodebaugh, Heimberg, Taylor, & Lenze, 2015; Rodebaugh et al., 2013). Further, people with SA who exhibit a friendly submissive style appear to respond better to psychotherapy, showing fewer symptoms and better adjustment after treatment than peers with a cold interpersonal demeanor (Cain et al., 2010).

An intriguing line of research into the degree to which individual people's social behavior vacillates across time and settings suggests that anxious people not only show elevations in both quarrelsomeness and submission, but also shift markedly—and more than depressed peers—among agreeable, dominant, and submissive behavior both during the course of a single event and across events over time (Rappaport, Moskowitz, & D'Antono, 2014). These data, although not specific to SA per se, suggest that at least some socially anxious people do engage in affiliative and cooperative ways. However, they may do so indiscriminately or unpredictably, potentially shifting away from approaches that might have been useful for deepening or enhancing relationships had they been used consistently.

Several areas of research into social behavior thus converge to suggest that people with SA engage in a broad range of behaviors that function in the short term to circumvent evaluation by others, but in the long term to keep the individual predictably situated in the social hierarchy, which is not always desirable. These behaviors vary among avoidant, hostile, and affiliative gestures; indeed, some evidence suggests that a fluid and variable interpersonal style may serve as a characteristic marker of anxious individuals. The degree to which context shapes both an anxious person's choice of behaviors and the regularity with which that individual engages in any given type of behavior remains unclear; clarifying the ways in which different people with SA respond to varying pressures in different social environments and community structures will be an important next step.

An Updated Model of SA

Clearly, an evolutionary perspective is critical for helping us to both understand the causes of SA and make better decisions about how to manage it. By highlighting the adaptive, as well as the maladaptive, aspects of SA, an evolutionary perspective could also help shift perceptions of this condition and decrease the stigma that affected individuals often face or fear. In this section, we update existing evolutionary models, taking into account the previously discussed changes in our understanding of primate social life, particularly the importance of developing and maintaining a strong set of positive relationships, and of human SA and how it relates to distinct patterns of interpersonal behavior. But before we outline the model, we address one area that we have alluded to, but not addressed in detail, the critical role that we hypothesize is played by the degree of uncertainty within the given social context.

The Important Role of Uncertainty

Clearly any updated evolutionary model needs to take into account new knowledge about primate relationships. In addition, however, it must also incorporate the best available evidence about what environmental challenges might engender SA or related emotional states. This evidence, for primates, points to uncertainty (i.e., about the social hierarchy or one's place in it) as a key challenge that warrants attention, consistent with existing evolutionary models of SA (Gilboa-Schechtman et al., 2014; Trower & Gilbert, 1989). If animals (including humans) cannot make reasonable guesses about where they stand and what will happen, how can they make reasonable guesses as to what to do next? Such uncertainty is distressing. Indeed, a growing literature identifies "intolerance of uncertainty" as an important predictor or correlate of SA in humans (Carleton, Collimore, & Asmundson, 2010; Teale Sapach, Carleton, Mulvogue, Weeks, & Heimberg, 2015).

Notably, as Grupe and Nitschke (2013) point out in their cogent model of the neural basis for anxious responses to uncertainty, the anticipatory cognitive and emotional processes that we engage when we are trying to ward off or lessen the impact of possible threats are typically adaptive. In other words, picking up on a real threat before it happens seems likely to enhance fitness and survival. Similar reactions to improbable threats, however, are less useful and may instead impede healthy functioning if they occur with excessive frequency or intensity. Thus, assuming that every instance of uncertainty will have negative consequences is, under most circumstances, going to be maladaptive. Grupe and Nitschke (2013) also note that uncertainty combined with uncontrollability is a particularly strong trigger for anxious responses. Indeed, this pairing has been linked with anxiety and distress across decades of research (Havranek et al., 2016; Miller, 1979; Mineka & Kihlstrom, 1978). The degree to which individuals see themselves as capable of managing, or at least responding effectively to, uncertain social contexts may thus also help determine how distressing those contexts are.

When Do We Expect Increases in the Expression of Anxiety?

Overall, then, we argue, consistent with existing evolutionary models of SA, that the core feature that elicits anxiety (in any situation) is uncertainty. When animals or people do not know where they stand, what is happening, or what the future will hold, they become anxious. In the social realm, this sequence may manifest in several ways.

First, we expect anxiety to be the most common when formerly stable relationships become unstable. This may occur as a consequence of conflict, but can also simply reflect the introduction of new and unfamiliar individuals to a group or the departure of familiar conspecifics. Note that we expect some level of anxiety in the face of instability in any relationships, not simply good ones or bad ones. Knowing that one has a poor relationship with another individual is better than not being sure whether the other individual will groom you or bite you; however, we would

nonetheless expect higher levels of anxiety when valuable relationships are unstable, regardless of the valence of interactions among the individuals involved.

Second, we expect that anxiety should increase in the context of aggression primarily when aggression is unexpected or when it is not properly reconciled. Unfortunately, however, it is difficult to measure this, as reconciliation reduces not only uncertainty about the relationship, but also anxiety, through grooming and other affiliative behaviors. Indeed, it is hypothesized that both uncertainty and inadequate reconciliation necessarily contribute to anxiety (see Fraser, Stahl, & Aureli, 2008; Kutsukake & Castles, 2001, for evidence that both of these factors are key, the so-called integrated hypothesis). However, there is some evidence that uncertainty plays a role distinct from the anxiolytic effects of positive contact. Even the aggressor seems to find the uncertainty of conflict stressful, and to show larger reductions in SBD after reconciled conflict than unreconciled ones; in this case, though, body contact with other individuals did not reduce SBDs, indicating that the effect is specific to the individual with whom there was a conflict (Das, Penke, & Van Hooff, 1998). This supports a primary role of uncertainty reduction.

Finally, we expect anxiety in all individuals, dominant or subordinate, in situations of social uncertainty. Very low rank is certainly stressful, but even high-ranking individuals may suffer from extremely high levels of stress (Sapolsky, 2004). Susceptibility to stress for individuals of high rank may be greatest when their rank is unstable (i.e., they are ascending, being challenged, or are young; Knight & Mehta, 2017). Further, at these inflection points, individuals of all ranks may be especially doubtful that their social context is within their control, because shifting circumstances may call for them to use new or unpracticed social skills.

When Does Anxiety Become Pathological?

Of course anxiety in these contexts is not pathological; it's completely typical. And that is a key point—some anxiety is not only expected, but also beneficial. Much like pain lets you know to pull your hand away from the fire, anxiety lets you know it is time to seek out the object of your anxiety and see if you can (re)establish, and potentially improve, the relationship's parameters.

But when does such anxiety become pathological? We argue that pathological SA occurs when this system goes into overdrive, with subjects failing to establish stable relationships, perceiving uncertainty in relationships that are not actually there, or overreacting to even subtle cues of instability. When these missteps occur repeatedly, which may be especially likely to happen in uncertain contexts, individuals may become increasingly prone to read others' cues through a biased lens, which results, in turn, in recurrent failure to recognize opportunities to form relationships or instances when relationships are not stable. These failures are likely to have negative repercussions that provoke distress. In such cases, we would expect high levels of anxiety-related behaviors (SBDs such as self-scratching, self-grooming, yawning, or body shaking) or hormones (i.e., cortisol), atypically large spikes following moderately uncertain situations, or spikes in situations to which

other individuals (or the same individual, in secure contexts) do not react. These are all empirically testable questions, allowing us the opportunity to test and refine this hypothesis.

One additional thing to remember—following the tradition of behavioral phylogeny, what we are outlining is the early stages of the evolution of SA, with the ultimate goal of understanding how this developed into the suite of emotional and behavioral patterns that we see in humans with SA today (i.e., Brosnan & de Waal, 2014; Brosnan et al., 2009). Humans, with our substantially more complex cognition and social environments will have developed additional symptoms, situations that elicit SA, and so forth. What is important, though, is to break SA into its component parts in order to determine the original function of (non-pathological) SA, to delineate how it was originally expressed pathologically, and then to figure out how human clinical SA developed from this. Mel Konner (2002) has likened this process of discovery to considering how a bird caught in a thicket could escape; in principle, the bird has limitless options, but in reality, the ways in which its wings are tangled constrain it to a few realistic choices. Much as with the bird's wing, we need to figure out not the limitless ways SA *could* have evolved, but the constraints that forced it into the current path (Konner, 2002). Only in this way can we understand the root causes of the condition and its associated behaviors and then trace the key aspects that developed along the way.

What Makes Humans Stand Apart?

Humans express SA differently than other primates, so one important key will be determining which aspects of distinctively human behavior and cognition were critical in reshaping SA. For example, although some primates, and in particular apes, appear to have a limited theory of mind (Boesch, 1992; Hare, Call, Agnetta, & Tomasello, 2000), most lack the sophisticated sense of self that humans possess. Nonhuman primates are thus not likely to be as vulnerable as are humans to distress associated with insult to one's self. One key question when considering human SA is the degree to which this uniquely human vulnerability to insults to the self combines with discomfort with uncertainty to underpin SA.

In addition, humans have evolved two more distinctive capacities that add nuanced layers to the basic foundation of SA. First, we have a complex sense of "other" that engenders an advanced type of empathy (Watt & Panksepp, 2016). Although empathy is, in many contexts, helpful for strengthening social ties, a growing body of research suggests that it can also be problematic by leading to distress (Tone & Tully, 2014). How this ability to place ourselves vividly in others' shoes interacts with the tendency to experience distress in contexts of social uncertainty remains unclear and warrants further investigation. For example, highly empathic people may overread others' subtle cues (Chikovani, Babuadze, Iashvili, Gvalia, & Surguladze, 2015), thus potentially picking up on evaluative signals that were not intended to be public and consequently responding in unwanted ways. These people may also, due to their sensitivity to others' distress, respond

preferentially to others' evaluative cues in a friendly submissive way that has its own distinctive set of risks and rewards from that associated with a cold/quarrelsome style. Empirical work examining how individual differences in empathy relate differentially to SA and related behavior across species and social contexts is a potentially useful next step.

Humans' ability to look forward in time and anticipate how a behavior in the moment might affect future outcomes is particularly sophisticated (Roberts, 2002). This ability to project oneself forward in an abstract and hypothetical way may make humans more acutely aware of the uncertainty inherent in social life—unlike members of other species, we must grapple not only with today's instability, but also the instability that might arise tomorrow and the next day. Although being able to imagine that the future has clear advantages, it also sets humans up to brace for negative outcomes that may never occur. Members of more present-oriented species thus play on a necessarily simpler field, with only immediate social cues to take into account when evaluating where one stands in the hierarchy.

Not only do humans have some characteristics that vary markedly from those of other species, but we also live in social structures that differ in key ways from other primates'. In particular, we relate to strangers in a much more complicated way than do members of most other species. In primates, interactions with strangers are basically consistent—you generally want to keep them away or get away from them. In humans, in contrast, interactions with strangers are more of a gamble. They can be advantageous, opening one up, for example, to new resources and avenues of support, or may be dangerous. Humans thus have additional decisions to make about when social environments are safe and they also face a constant stream of threats to the stability of their social hierarchies, given the fluidity with which strangers enter social groups.

Conclusion

Humans have developed what may be the most flexible social system of any animal, but with this flexibility come costs; we also suffer greater uncertainty as to our place in that system, and how we influence or are influenced by other humans. When considering this, it is not surprising that socially mediated anxiety is so common. Our goal in this chapter has been to emphasize ways in which taking an empirically informed evolutionary perspective can help us to better understand SA, better predict the contexts and individuals in which it will emerge, and better determine when it is appropriate to treat the symptoms, versus the cause, versus neither.

Of course, we cannot yet answer that question. Thus, our secondary goal was to provide a hypothesized framework that could be used as a starting point to test specific predictions that will further refine our understanding. Some of these studies will need to be done with nonhuman species, to better understand how our reactions fit with the animal kingdom or to utilize a model systems approach to study questions that are not empirically tractable in humans, and some will need to be done

with humans. Each will provide a better way of understanding the ways in which SA is derived from what we believe were (and are) rational and beneficial reactions.

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Jealousy, Infidelity, and the Difficulty of Diagnosing Pathology: A CBT Approach to Coping with Sexual Betrayal and the Green-Eyed Monster

David M. Buss and Mike Abrams

Intense jealousy can be emotional acid that corrodes marriages, undermines self-esteem, triggers battering, and is a key motive in the murder of mates and ex-mates (Buss, 2000a, 2000b; Buss & Duntley, 2011; Daly & Wilson, 1988; Daly, Wilson, & Weghorst, 1982). Extreme jealousy has been given many names in the clinical and psychiatric literature—*The Othello Syndrome*, *Morbid Jealousy*, *Psychotic Jealousy*, *Pathological Jealousy*, *Conjugal Paranoia*, and *Erotic Jealousy Syndrome*. Jealousy, of course, can be pathological. It can destroy previously harmonious relationships, rendering them hellish nightmares of daily existence. Trust slowly built from years of mutual reliance can be torn asunder. Jealousy causes more women to flee in terror to shelters for battered women than any other cause (Wilson & Daly, 1996).

A full 13% of all homicides are spousal murders, and jealousy is overwhelmingly the leading cause (Buss, 2005; Daly & Wilson, 1988). When an adult woman is murdered, the odds are between 50% and 70% that the perpetrator is a husband, boyfriend, ex-husband, or ex-boyfriend. A common sentiment expressed by these killers is *"If I can't have her, no one can."* Jealousy is a dangerous emotion that has driven lovers to such violent extremes that many cultures have laws specifically tailored to it—crimes of passion.

One pathological aspect of extreme jealousy, according to traditional psychiatric thinking, is not the jealousy itself. It is the delusion that a loved one has committed

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an infidelity when none has occurred. The rage itself upon the actual discovery of an infidelity is something people everywhere intuitively understand. In Texas until 1974, a husband who killed a wife and her lover when he caught them *in* flagrante delicto was not judged a murderer. In fact, the law held that a "reasonable man" would respond to such extreme provocation with acts of violence. Similar laws have been on the books worldwide. In France, Italy, the United Kingdom, Brazil, and Uruguay, for example, killing in this context typically resulted in reduced criminal charges, such as from murder to manslaughter, and reduced sentences if convicted. Extreme rage upon discovering a wife naked in the arms of another man is something that people everywhere find intuitively comprehensible. Criminal acts that would normally receive harsh prison sentences routinely get reduced when the victim's infidelity is the extenuating circumstance. Why do people intuit that a "reasonable man" would be driven to such extremes? And are diagnoses of pathological jealousy destruction always warranted?

A professional couples' therapist related the following story. A young couple we will call Joan and Richard came to her with a presenting complaint of irrational jealousy. Without provocation, Richard would burst into jealous tirades and accuse Joan of sleeping with another man. His uncontrollable jealousy was destroying their marriage. Richard and Joan both agreed on this point. Could the therapist help cure Richard of irrational jealousy?

A common practice in therapy for couples is to have at least one session with each member of the couple individually. The first question the therapist posed to Joan during this individual interview was: Are you having an affair? She burst into tears and confessed that, indeed, she had been carrying on an affair for the past 6 months. Having confessed to the therapist, Joan felt obligated to reveal this information to her husband. They ended up divorcing. Richard's jealousy, it turned out, had not been irrational after all. Presumably, he had been picking up on subtle cues that triggered his jealousy. Since he trusted Joan and she had assured him of her fidelity, however, he became convinced, with Joan's help, that his jealousy had been irrational. In a sense, Richard had failed to listen to his internal emotional wisdom.

In scientific surveys of jealousy, nearly all men and women report having experienced at least one episode of intense jealousy (Buss, 2000a, 2000b). Thirty-one percent say that their personal jealousy has sometimes been difficult to control. And among those who admit to being jealous, 38% say that their jealousy has led them to want to hurt someone. This intense emotion, in short, is not limited to spouse killers.

The Evolution of Jealousy

Despite its dangerous manifestations, jealousy helped to solve critical reproductive quandaries for ancestral men and women. Consider first a fundamental sex difference in our reproductive biology—the fact that fertilization takes place inside women's bodies and not men's. Internal female fertilization is not universal in the biological world. In some species, such as the Mormon crickets, fertilization occurs

internally within the male. The female takes her egg and literally implants it within the male, who then incubates it until birth. In other species, fertilization occurs externally to both sexes. The female salmon, for example, drops her collection of eggs after swimming upstream, the male follows and deposits his sperm on top, and then they die, having fulfilled the only mission in life that evolution gave them. But humans are not like salmon. Nor are we like Mormon crickets. In all 5,416 species of mammals, of which we are one, and in all 350 species of primates, of which we are also one, fertilization occurs internally within the female, not the male. This posed a serious problem for ancestral men—the problem of uncertainty in paternity.

From an ancestral man's perspective, the single most damaging form of infidelity his partner could commit, in the currency of reproduction, would have been a sexual infidelity. A woman's sexual infidelity jeopardizes a man's confidence that he is the genetic father of her children. A cuckolded man risks investing years or even decades in another man's children. Lost would be all the effort he expended in selecting and attracting his partner. Moreover, he would lose his partner's labors, now channeled to a rival's children rather than his own.

Women, on the other hand, have always been 100% sure that they are the mothers of their children—internal fertilization guarantees that their children are genetically their own. No woman ever gave birth and, watching the child emerge from her womb, wondered whether the child was really hers. One African culture captures this sex difference with a phrase more telling than any technical summary: *"Mama's baby, papa's maybe."* Biology has granted women a confidence in genetic parenthood that no man can share with absolute certainty.

Our ancestral mothers confronted a different problem—the loss of a partner's commitment to a rival woman and her children. Because emotional involvement is the most reliable signal of this disastrous loss, women key in on cues to a partner's feelings for other women. A husband's one-night sexual stand is agonizing, of course, but most women want to know: "Do you love her?" Most women find a singular lapse in fidelity without emotional involvement easier to forgive than the nightmare of another woman capturing her partner's tenderness, time, and attention (Shackelford, Buss, & Bennett, 2002). We evolved from ancestral mothers whose jealousy erupted at signals of the loss of love—mothers who acted to ensure the man's commitment.

But who cares who fathers a child or where a man's commitments get channeled? Shouldn't we love all children equally? Perhaps in some utopian future, we might, but that is not how the human mind is designed. Husbands in our evolutionary past who failed to care whether a wife succumbed to sex with other men and wives who remained stoic when confronted with their husband's emotional infidelity may be admirable in a certain light. Perhaps these self-possessed men and women were more mature. Some theories, in fact, propose that jealousy is an immature emotion, a sign of insecurity, neurosis, or flawed character. Non-jealous men and women, however, are not our ancestors, having been left in the evolutionary dust by rivals with different passionate sensibilities. We all come from a long lineage of ancestors who possessed the dangerous passion.

Jealousy, according to this theory, is an adaptation. An adaptation, in the parlance of evolutionary psychology, is an evolved solution to a recurrent problem of survival or reproduction. Humans, for example, have evolved food preferences for sugar, fat, and protein that are adaptive solutions to the survival problem of food selection. We have evolved specialized fears of snakes, spiders, and strangers that are adaptive solutions to ancestral problems inflicted by dangerous species, including ourselves. We have evolved specialized preferences for certain qualities in potential mates, which helped to solve the problems posed by reproduction. Adaptations, in short, exist in modern humans today because they helped our ancestors to combat all of the many "hostile forces of nature," enabling them to better survive and reproduce. Adaptations are coping devices passed down over millennia because they worked—not perfectly, of course, but they helped ancestral humans to struggle through the evolutionary bottlenecks of survival and reproduction.

Many expressions of jealousy, according to this perspective, are not signs of immaturity, but rather important passions that helped out ancestors, and most likely continues to help us today, to cope with a host of real relationship and reproductive threats. Jealousy, for example, motivates us to ward off rivals with verbal threats and cold primate stares (Buss & Shackelford, 1997a, 1997b). It drives us to keep partners from straying with tactics such as escalating vigilance or showering a partner with affection. And it communicates commitment to a partner who may be wavering, serving an important purpose in the maintenance of love. Sexual jealousy is often a successful, although sometimes dangerous, solution to persistent predicaments that each one of our ancestors was forced to confront.

We are typically not conscious of these reproductive quandaries. Nor are we usually aware of the evolutionary logic that led to this dangerous passion. Men do not think: "*Oh, if my wife has sex with someone else, the certainty that I'm the genetic father is jeopardized, thereby endangering my genetic legacy …I'm really mad*!" Nor does a man whose partner uses birth control think, *"Well, because Joan is taking the pill, it doesn't really matter whether she has sex with other men; after all paternity is not an issue."* Nor does a woman think: "*It's really upsetting that Dennis is in love with that shrew instead of me; this jeopardizes my hold on his emotional commitments to me and my children, and hence hurts my overall reproductive success."* Instead, jealousy is an essential passion, just as our hunger for sweets and our craving for companionship are evolved adaptations. Jealousy can be considered a type of nonconscious emotional wisdom passed down to us over millions of years by our successful forebears.

Jealous men were more likely to reserve the cost of parental obligation for their biological children, rather than squandering them on the children of rivals. As descendants of a long line of men who acted to ensure their paternity, modern men carry with them the dangerous passion that led to their forebear's reproductive success.

According to this hypothesis, jealousy represents a form of ancestral wisdom that can have useful as well as destructive consequences. The view of extreme jealousy as inevitably pathological ignores a profound fact about an important defense designed to combat three real threats to intimate relationships—infidelity,

potential mate poachers, and a partner's outright defection from the relationship. Jealousy is not always a reaction to an infidelity that has already been discovered. It can be an anticipatory response to adaptive problems such as a mate value discrepancy or to the sudden presence of potential mate poachers (Schmitt & Buss, 2001). So it can be a preemptive strike to prevent an infidelity or defection that might occur. Labeling jealousy as delusional or pathological simply because a spouse has not yet strayed ignores the fact that jealousy can head off an infidelity that might be lurking on the horizon of a relationship.

The Difficulty of Diagnosing When Jealousy Is a Pathological Disorder

Some expressions of jealousy clearly qualify as psychologically disordered. The DSM notes one form—Delusional Disorder-Jealous Type (Easton, Shackelford, & Schipper, 2008). This requires clear evidence of delusions of a partner's infidelity when no infidelity has occurred. Consider this case. On Christmas Eve, a man looked out of his living room window across the street and noticed his neighbor's Christmas tree lights blinking. When he compared them to the analogous lights his wife had set on their tree, he noticed that they were blinking in synchrony with those of the neighbor. He concluded that his wife was having an affair. His wife insisted that he see a psychiatrist, who diagnosed him with delusional jealousy. As it turned out, his wife was indeed having an affair. Moreover, she was having an affair with that specific neighbor. So is delusional jealousy the proper diagnosis? Clearly, there was a delusional component; it is extremely improbable that there existed Christmas tree light synchrony intentionally created by his wife and his neighbor. But his inference of his wife's infidelity was perfectly on target and not delusional.

Some have offered criteria for distinguishing normal from pathological jealousy. For example, Marazziti et al. (2003) identify these key criteria:

- Time taken up by jealous concerns.
- Difficulty in putting the concerns out of the mind.
- Impairment of the relationship.
- Limitation of the partner's freedom.
- Checking on the partner's behavior.

The difficulty with the application of these criteria is that they are overly broad. If a partner is indeed having an affair, or perhaps even considering having an affair, these expressions of jealousy may signal the normal operation of the adaptation of jealousy. Limiting the partner's freedom and even extremes of checking on the partner's behavior to the point of stalking are common manifestations of mate guarding (Buss, 1988; Buss & Shackelford, 1997a, 1997b).

Kingham and Gordon (2004) offer these common symptoms of pathological jealousy:

- Accusing partner of looking or giving attention to other people.
- Questioning of the partner's behavior.
- Interrogation of phone calls, including wrong numbers or accidental phone calls, and all other forms of communication.
- Going through the partner's belongings.
- Always asking where the partner is and whom they are with.
- Isolating partner from their family and friends.
- Not letting the partner have personal interests or hobbies outside the house.
- Controlling the partner's social circle.
- Claiming the partner is having an affair when they withdraw or try to escape abuse.
- Accusing the partner of holding affairs when the marriage's sexual activity stops because of the abuse.
- Lack of trust.
- Verbal and/or physical violence toward the partner, the individual whom is considered to be the rival, or both.
- Blaming the partner and establishing an excuse for jealous behavior.
- Denying the jealous behavior unless cornered.

Again, however, all of these behaviors have been documented as common mate guarding and retention tactics whose frequency is increased when someone faces one of the adaptive problems of partner infidelity, threat of mate defection, presence of mate poachers, or all the three (Buss, 2000a, 2000b). Even threats of harm to self if a partner threatens to leave the relationship and verbal or physical violence directed a partner are common expressions of mate guarding across cultures (Buss, 1988; Buss, 2000a, 2000b; Buss & Duntley, 2011). A threat of suicide if a partner leaves sometimes solves an adaptive problem of mate retention and the partner stays. And often, violence and threats of violence cause a woman to stay in a relationship, even if she wants to get out, again solving the problem of mate retention. Although physical violence toward a spouse is illegal in some cultures, but by no means all, laws against wife-beating and spousal rape are relatively recent and have not characterized most of the centuries in which humans have had written laws.

Four additional problems render a diagnosis of pathology problematic—the signal detection problem, the on-average effectiveness problem, error management logic, and sensitivity to predictors of infidelity even when none has occurred.

The signal detection problem. Although infidelity is often morally condemned and seen as a sign of dysfunction, a good case can be made that affairs evolved to solve adaptive problems. For men, the historical reproductive benefits of infidelity were fairly straightforward—increased sexual access to fertile women translated into more offspring and greater reproductive success (Symons, 1979). For women, infidelity is more puzzling, since rarely could it have translated into higher reproductive output (the exception being married to a man who was impotent or infertile). The two leading evolutionary hypotheses for female infidelity are (1) securing good genes from an affair partner while securing investment from a regular partner (Gangestad & Haselton, 2015), and (2) the mate-switching function, by which

affairs secure a backup mate, pave the way for exiting a bad relationship, trading up to a higher mate value partner, or all the three (Buss, Goetz, Asao, Conroy-Beam, & Duntley, 2017).

Because it has been advantageous for one individual to have an affair, and the affair comes at a potentially steep cost to the partner, defenses evolved to prevent its occurrence. The psychological complex of jealousy and its behavioral output in mate guarding and retention, as discussed above, are the primary coevolved defenses. As defenses against a partner's infidelity evolved, more sophisticated strategies for conducting affairs evolved. Chief among these were secrecy. As jealousy evolved and became more elaborate in design specificity, infidelity got driven underground, cloaked in great secrecy. As one sex became more and more sensitive to subtle cues of infidelity, such as unexplained absences, strange scents, changes in sexual interactions, and many others (Shackelford & Buss, 1997), the other became more adept at concealing these cues. The resulting coevolutionary arms race created a signal detection problem—how could a calamitous infidelity be detected when cues to its occurrence were so skillfully concealed? Consequently, motivated monitoring, seemingly paranoid suspicions, cutting off a spouse's social contacts, isolating a partner, snooping through their belongings—all seen by some as signs of pathology—may instead be the normal behavioral output of an adaptation working effectively to detect intentionally concealed subtle signals.

The on-average effectiveness of the jealousy adaptation. Another problem is that solutions to adaptive problems evolve because, on average across the sample space of instances, they solve or ameliorate the problem better than alternative designs extant in the population at the time of its evolution. Callus-producing adaptations are designed to protect the anatomical and physiological structures beneath the skin, but those structures sometimes still get damaged despite the presence of calluses. Adaptations for coalitional warfare can evolve, even if these result in the death of the attackers some of the time or even a lot of the time (Tooby & Cosmides, 2010). In other words, there are many "instance failures" of adaptations, despite their on-average effectiveness (Cosmides & Tooby, 1999).

Jealousy, an adaptation designed to defend against a partner's infidelity and potential defection, also produced many instance failures. Some partners still cheat despite jealous mate guarding. Some partners still defect, despite the deployment of the most effective mate retention tactics at a person's disposal. These instance failures do not falsify the hypothesis that jealousy is a well-designed adaptation, since all adaptations work based on their on-average success, not based on their success in each and every case in which the relevant problem is confronted.

Jealousy embodies error management logic. When faced with conditions of uncertainty, there are two ways to err—failing to detect a problem that exists and falsely detecting a problem when none exists. A rustling in the leaves may signal a poisonous snake or a harmless sound stemming from a gust of wind. The costs of inferential errors differ in this case. Inferring a snake's existence when there is no snake produces relatively trivial caution and avoidance. Failing to infer a snake's existence when there is one could result in death. In short, there is often a cost asymmetry in inferential errors under conditions of uncertainty. According to Error

Management Theory, recurrent cost asymmetries of this sort result in the evolution of cognitive biases to err in the direction of avoiding the more costly error (Haselton & Buss, 2000). A smoke alarm is set sensitively by design to produce many false positives because the cost of missing an actual fire is far steeper than the cost of dealing with annoying alarm sounds when there is no actual fire.

Error management theory logic applies with equal force to the evolved design of jealousy (Buss, 2000a, 2000b). Failing to detect an actual infidelity is generally costlier than falsely suspecting one that has not occurred. Jealousy-motivated vigilance or suspicion is generally less costly than being oblivious to an infidelity. Falsely suspecting a spouse of infidelity, of course, can have costs from small to large. It could produce relationship conflict, wasting valuable effort on a problem that does not exist. Persistent jealousy also sometimes drives a partner out of a relationship or into the arms of others. But if the on-average cost of erring by falsely suspecting exceeds the average cost of missing an infidelity or defection, jealousy thresholds will evolve to avoid the more costly error.

Jealousy is triggered by predictors of infidelity when none has occurred. The difficulty of diagnosing when jealousy is pathological becomes further compounded by the fact that jealousy is designed to be activated by predictors of infidelity, but also to statistically recurrent predictors of infidelity when no infidelity has actually occurred. Consider mate value. People generally couple based on overall mate value; the 8s tend to pair up with other 8s, the 6s with other 6s (Buss, 2003). Over time, however, mate value discrepancies can emerge. A man or woman might receive a large promotion at work or large status boost from a career breakthrough, dramatically improving their mate value. A man or a woman could become ill, suffer a debilitating injury, or suffer a status loss, dramatically debilitating their mate value. Because the components that contribute to mate value are never static and always change over time, mate value discrepancies inevitably emerge. If they get large enough, they predict infidelity, defection, and mate switching (Buss & Shackelford, 1997b). If jealousy is designed to be triggered by a mate value discrepancy, even if no infidelity or defection has occurred, it can seem pathological when it is not.

Mate value discrepancies are not the only statistical predictors. Other candidates include erectile dysfunction, orgasmic difficulty, sexual dissatisfaction, decline in sexual desire or drive, the sudden introduction of new sexual positions, abrupt changes in clothing style, innocuous but unexplained absences, and many others (Buss, 2000a, 2000b; Shackelford & Buss, 1997). A man who experiences erectile dysfunction or whose wife becomes sexually dissatisfied may suspect that she will seek sexual gratification elsewhere. Abrupt changes in clothing or sexual positions may signal infidelity, but may simply be innocuous attempts to spice up a life of quiet desperation. Since jealousy is designed to become activated to statistical predictors of infidelity, even if it has not occurred and might never occur, but rather just increases the odds, it can seem pathological when in fact it is functioning precisely as it was designed to function.

From an evolutionary perspective, a diagnosis of disorder requires that an evolved mechanism not function as it was designed to function (Wakefield, 2005). In the

case of jealousy, if it gets activated in contexts it was not designed to get activated, is triggered by drugs or alcohol that produce delusions or lower thresholds for suspicion for example, it is not functioning as it was designed to function and so can become pathological or disordered. But the signal detection problem, the on-average success of adaptations that produce many instance failures, the adaptive error management biases designed to avoid the more costly errors even at the expense of more frequent but less costly errors, and the fact that jealousy is activated by statistical predictors of events that have not occurred, render a diagnosis of jealousy as pathological diabolically difficult.

Cognitive behavior therapy, however, can produce insights into these difficulties and help patients with presenting problems of jealousy.

Rational-Emotive/Cognitive Behavior Therapy (RE/CBT) Applied to Jealousy or Infidelity

Although evolutionary theory compellingly depicts jealousy as an adaptation, it is one that can be perceived as maladaptive in many modern social settings. Until very recently, there were few normative values against violence, murder, or any other socially disagreeable manifestations of jealousy. Until not long ago, the jealous male could violently assault or kill any perceived competitor without formal consequences. Of course the victim's family, tribe, or clan might seek retribution, but such reprisals were far less assured than those confronted by a violently jealous man today. From a purely adaptive point of view, it was advantageous for a male to use any effective means to remove a competitor, at least if one could implement this removal in a manner carried out to minimize the costs of doing so (e.g., victims fight back or even kill to prevent being killed). If a potential competitor were killed without consequence, the risk of being cuckolded or losing a mate plummeted. Moreover, women had little recourse when severely restricted or even battered by a mate. Indeed, if women in some present cultures face restrictions in dress, social behavior, and sexual expression, one can only imagine what a social order exclusively dominated by the strongest males would impose on women.

Sex-differentiated mating strategies have evolved in humans. These include sex-differentiated mate preferences, with men prioritizing cues to fertility such as physical appearance and youth, and women prioritizing a man's willingness and ability to channel resources to herself and her children (Buss, 1989). Both women and men share preferences for long-term mates who are healthy, kind, and intelligent. Given the large gender asymmetry in minimum obligatory parental investment, men have evolved stronger motivations to seek short-term sex, including a desire for partner variety, letting little time elapse before seeking sexual intercourse, a high sex drive, minimizing entangling commitments, and many others (e.g., Buss, 2015; Jonason & Buss, 2012).

The male in his quest to gain sexual access to females had to compete with other males with the same agenda. This led males to be competitive, protective of their mates, and aggressive with competing males. A male who provided material support

in the early stages of infant development would improve the survival chances of his offspring. So a strategy that included controlling, protecting, and providing some care for both the mother and his offspring was used (Buss, 1988; Buss & Shackelford, 1997a, 1997b; Fisher, 1992, 2004).

Of course, women evolved in parallel to men and also possess inclinations that are adaptations to the environments in which they evolved. Female evolution appears to include several strategies that increased their reproductive success. One of these included bonding with a male and fending off other females by actively seeking the male's attention. And in early ancestral settings, females would use verbal aggression to diminish the competing female's standing in the social order—a strategy still present in modern times (Buss & Dedden, 1990; Campbell, 1999). Female jealousy evolved to take a more defensive and less physically risky style. Female jealousy evolved as a mate retention adaptation, functioning to protect against the hazard of committing reproductive resources in a male, nurturing his children (and genes), only to have him divert his resources to other females. Women who lost a man's commitment to another woman would have faced the loss of protection and provisioning, putting themselves and their young at risk.

Women prone to jealousy are less likely to bear children from unfaithful. In other words, they will detect them men who are not committed to caring for them and their children during their most vulnerable time—from pregnancy until the offspring is approximately 4 years old and weaned from the mother (Fisher, 1992, 2004). The jealous woman not only wards off female competitors for the male's affections, but also continues to assess his commitment to the partnership. In effect, jealousy is a kind of vigilance to identify a deceptive male's feigned commitment, developed to prevent the male from impregnating another female, leaving the partnership, and devoting his resources to another family.

Adaptations for infidelity under certain circumstances have evolved in both men and women (Buss, 2015). The male who impregnated more females would have more descendants. Females whose reproductive potential is limited by our long gestation and weaning period, still may still benefit from infidelity if it leads to procuring genes from males whose appearance suggests good health (Gangestad & Haselton, 2015) or from using affairs to switch mates—to leave one mating relationship and trade up to a better or less cost-inflicting one (Buss et al., 2017). For example, women are more attracted to men with greater physical symmetry, larger body size, superior physical strength (Puts, 2010), and men who are successfully polygynous. This latter criterion may seem to be counterintuitive, but may be explained by the "sexy son hypothesis" (Weatherhead & Robertson, 1979). Specifically, women who seek males who are highly polygynous, and otherwise desirable, will tend to produce a larger number of comparable sons. These polygynous sons will experience higher reproductive success, thereby increasing the reproductive success of the mothers who produced them.

The evolutionary perspective discussed here is critical in appropriately applying Rational-Emotive/Cognitive Behavior Therapy (RE/CBT) in cases of jealousy and infidelity. If the clinician applies social dogma that interprets jealousy as pathological and infidelity as immoral, the client will not be adequately aided. Clinicians who have not studied evolutionary theory often intuit that infidelity cannot be pathological simply because it is so common. The same is true for jealousy. The jealous person viscerally feels that he or she is protecting him or herself from a perceived danger (Buss & Shackelford, 1997a, 1997b; Buunk, Angleitner, Oubaid, & Buss, 1996; Clanton, 1996). Telling this person that they are pathological for being jealous, is not helpful, nor accurate. If a clinician endorses these desires and behavior as part of the proper functioning of human mating adaptations, then the client will be helped with attaining self-acceptance. A client who has self-acceptance is more able to objectively evaluate their actions and change to more adaptive behaviors in the current environment. Acceptance that a client's perspective is natural does not necessarily endorse it. Indeed, there are great many natural phenomena that are pernicious and harmful that are rejected by societal mores and laws (Curry, 2006).

Jealously, and the infidelity it guards against, developed with concomitant strategies. The deceptive partner has evolved abilities to furtively deceive the partner, and the jealous suitor has evolved mechanisms to detect the deception. These strategies may be considered culturally immoral, but they are not pathological using Wakefield's (1995) evolutionary definition of psychological disorder. Rather, both infidelity and jealousy in modern humans are ancestral reproductive strategies that may or may not be adaptive in the modern environment. Thus, jealousy and infidelity are closely related problems for the therapist. The former refers to the emotions and behaviors related to defending an intimate relationship. The latter involves the emotional distress that results when those defenses fail. Jealousy is not a pleasant emotion; it is perceived by most people as a type of urgent vigilance (Maner, Miller, Rouby, & Gailliot, 2009; Maner & Shackelford, 2008), and certainly produces much subjective distress (Buss, 2000a, 2000b). The perceived necessity and non-agentic aspect of jealousy needs to be appreciated by clinicians. It needs to be viewed as an evolved emotion that feels necessary for the affected individual. The jealous person typically does not feel neurotic or foolish, despite the distress the emotion is evoking in him or her. The jealous person believes that he or she is inferring a risk of a great loss, and jealousy is the consequential emotion that is apprehended as necessary to defend against the potential loss.

RE/CBT for Individuals or Couples

In the mid-1950s Albert Ellis observed that the preponderance of clients seeking his help for sexual or relationship problems suffered from distortions of thinking. Despite his psychoanalytic education, he did not find repressions, libidinal cathexes, ill-formed psychic objects, or any of the other Freudian pathologies. Instead, he found a consistent pattern of people distressing themselves with their own rigid, demanding, or inflexible beliefs (Ellis, 1957). His work led to the first cognitive behavior therapy that he would ultimately refer to as Rational-Emotive Behavior Therapy. By the mid-1960s Aaron Beck independently came to similar conclusions about psychopathology when working with depressed people. Over the next few decades their work, along with others like Arnold Lazarus, Donald Meichenbaum, and Michael Mahoney, led to the clinical approach now referred to as cognitive behavior therapy (CBT). In this chapter we will use the term RE/CBT to refer to these therapies, including elements of Ellis's original approach, combined with more recent protocols.

The RE/CBT approach to jealousy and infidelity in couple therapy (Abrams, 2012) seeks to uncover and modify each partner's distinctive cognitions that contribute to the struggles that brought them to counseling. When RE/CBT is used to help people with jealousy, it is usually after it has become a significant impediment to the relationship (De Silva, 1997). The person seeking help often recognizes that jealousy is problematic, or may seek help because the partner is rebelling against the jealousy and insists on the mate getting help. In either case the treatment would be similar.

A different approach is taken for couples seeking help. Therapy for two people in conflict requires that all interventions consider the often competing interests of the participants. As the evolutionary perspective makes clear, there are usually evolved psychological adaptations operating behind the stated motives of each participant. Even if infidelity is viewed as offering an evolutionary advantage to one member, it needs to be addressed quite sensitively when both members are present. Evolutionarily endowed inclinations are explanations, but not moral justifications. Understanding evolved drives is frequently a starting point in the effort to control or redirect them.

After an initial session to obtain background information, the couple is instructed in the principles of RE/CBT, so each partner can recognize and help correct the irrational thinking or cognitive distortions in himself or herself, as well as the partner. However, RE/CBT has features that are invoked regardless of the specific problem. Unlike the purported "depth" or "insight" therapies, RE/CBT seeks to illuminate and change the cognitions in the form of beliefs, attitudes, philosophies, or personal values that underlie all mental anguish responsive to talk therapy. These irrational cognitions typically take two forms: beliefs that are inflexible or absolutistic (A) and beliefs that are demanding (D) (e.g., Ellis, 1997). They can take forms like:

- "It would be completely humiliating if my lover cheated on me." (A)
- "I could not stand it, if I were lied to by my significant other." (A)

- "If I love someone, they must never do anything inconsiderate." (D)
- "My lover must absolutely be completely faithful to me." (D)
- "A significant other must treat me the way I want." (D)
- "If someone repays my fidelity with infidelity, he or she absolutely must be severely punished." (D and A)
- "I find it absolutely unbearable that someone is thinking they have made a fool of me." (A)

The innate nature of these jealous beliefs is supported by the intensity by which they are commonly held (Ellis, 1987). The degree of rage and alienation felt by the jealous companion is directly proportional to their confidence in the truth of the kinds of beliefs stated above. That is, the more strongly one holds a distorted or irrational cognition, the more intense the emotion when that belief is violated. And when a member of a couple feels provoked to jealousy it may be precipitated without a cognitive appraisal, by means of automatic circuit-logic reactions. However, the only way the individual can consciously assess feelings is verbally. Therefore, it is the initial goal of the RE/CBT process to guide clients to express these jealous emotions verbally. It is through this process that the jealous individual will begin to apprehend that the intensity of their emotion may not be in proportion to any objective threat to his/her immediate well-being—even if his partner is actually cheating. RE/CBT interventions will help the client see that even if betrayed, responding with intense negative emotions will only make the situation worse. Thus, RE/CBT helps one see that while having an unfaithful partner is clearly undesirable, it does not have to be perceived as devastating. Evolution has selected us to be jealous, but the contemporary interests of the individual do not always correspond with the interest of his or her genes. Stated differently, adaptations that historically led to reproductive success may currently conflict with personal happiness (Buss, 2000b).

Many jealous people will have irrational or distorted beliefs that arise due to the evolutionary threats posed by infidelity. However, the evolutionary imperative not to be cuckolded or to avoid loss of parental investment does not pose as great a danger to current reproductive success as it did for our distant ancestors. There is a mismatch between ancestral and modern environments in this respect. It is this paradox—that jealousy was once critical to reproductive success, yet may no longer be essential—that must be addressed by the therapist. It is quite reasonable for the jealous person to feel hurt, disappointed, sad, or alienated. But when the jealous person's narrative makes real or imagined concerns intolerable or disruptive to everyday life, then the person's concerns can be assumed to be based on cognitions that were once supremely functional, but may no longer be so in the modern world.

These beliefs are often accompanied by cognitive biases in which the person feeling jealous focuses only on those aspects of their environments that validate their disturbing beliefs. The jealous person will tend to reject alternative hypotheses for suspicious behavior, focus exclusively on behaviors deemed deceptive, exaggerate signs of disaffection on the part of the lover, and so on. So the disturbing beliefs will lead to confirmatory perceptions that will in turn intensify the irrational or

distorted cognitions. The therapist must recognize and dissect the components of this cognitive feedback loop as experienced among individual clients to determine whether jealousy is or is not pathological.

For example, in our discussion of the error management and the signal detection models of jealousy, it is evident that in all cases except for the correct detection or incorrect rejection of infidelity (ruling it out when it is occurring), the individual may appear pathological. In these cases, jealous individuals will believe that they have correctly discovered a basis for jealousy, even when it has not occurred. An example might be the discovery of a partner having a friendly email or text message exchange, leading to the conclusion that their partner is having an affair. There are four possible outcomes for the potentially jealous person:

1. Correctly detecting that the partner is cheating.
2. Correctly concluding that a faithful partner is indeed faithful.
3. Incorrectly concluding that the partner is unfaithful.
4. Incorrectly ruling that the cheating partner is in fact cheating.

People with the greatest level of jealousy have made being correct on the first and not being wrong on the third so overwhelmingly important, that they are willing to perennially be wrong on the second and fourth. The intensely jealous person has irrationally made being cheated on so dreadful, that they are willing to perennially torment themselves and their partners with false alarms and false accusations.

The more catastrophizing an individual conceptualizes a negative outcome, the less likely the person is to rule out its possibility. Let's compare those who apprehensively dread infidelity to individuals suffering from phobias. This may be best illustrated in individuals with aerophobia. The person afraid of flying is generally well acquainted with the vanishingly small probability of the flight crashing. Despite this, the person will persist in being afraid because he or she tends to pre-emptively experience the most appalling disaster imaginable. In short, if a person obsessively imagines a terrible outcome, the miniscule probability of its occurrence does little or nothing to offset the trepidation of the improbable. This can be seen in the client whose jealousy requires treatment. Over human evolutionary history, it would indeed pose potentially catastrophic risks to a man's reproductive success if jealousy did not exist. However, these risks are far lower today with environmental changes like effective birth control and genetic testing. And even when infidelity does lead to extra-pair reproduction, the cost in reproductive success may matter less than to his or her ability to enjoy life. As Pinker once noted, he has chosen not to reproduce at all, so his genes can go jump in a lake. We do not need to be slaves to emotions that may have been supremely functional in ancestral environments, but that currently impede modern-day well-being.

This leads a key therapeutic intervention to address the separate goals of the individual's selfish genes and that of the individual's current well-being. Consequently, even if the jealous person is not distorting the probability of a partner being unfaithful, the risks to the individual in the moment can be parsed from the historical risks to reproductive success. These are the essential loci of treatment for the RE/CBT therapist treating a client who has problems with jealousy.

RE/CBT for Individuals Troubled with Jealousy

Jealousy evolved as an adaptation in males, as a defense against being genetically cuckolded or abandoned entirely. It minimized the risk of losing fitness due to parental investment wasted on a non-related offspring. Among females, jealousy evolved as a means of limiting the risk of a partner diverting his resources to another woman and children. Because jealousy is an evolved emotion, it will tend to feel logical and protective to the individual experiencing it. Consequently, the jealous man will typically react as though the therapist is attempting to get him to lower his guard. This is true in the case of other genetically prepared fears such as the phobias associated with prepared classical conditioning (Seligman, 1971). These fears and phobias tend to arise with minimal consciously articulated cognition.

For example, people with phobic reactions to heights, insects, animals, and other innately feared things will often suffer these fears without the irrational or distorted beliefs that underlie social or self-worth fears. Thus, people with phobic fear of dogs or spiders will commonly display great fear without requiring a complicated cognitive appraisal of the danger. In contrast, an excessive fear of professional failure, loss of social status, or rejection in love tends to require a cognitive appraisal because they involve more complex problem solving. Importantly, both kinds of fears will generate cognitions that are addressable through RE/CBT. Once a comfortable therapeutic relationship is established, individuals typically become more open to the possibility of having irrational or distorted cognitions; they are often motivated because this type of thinking makes people feel distressed and wretched. Indeed, addressing personal misery is a prime reason for seeking therapeutic help.

RE/CBT for Couples Troubled with Jealousy

Since jealousy is almost always a problem between couples, couple's treatment often will be the focus. Individuals rarely seek help for jealousy for the reasons related to jealousy's evolutionary history detailed earlier. That is, when judging oneself, jealous people rarely feel that jealousy is a problem. Rather, they feel that the world, and those in it, are not to be trusted. More often, jealousy becomes a problem when it interferes with a couple's union. Like other universal human qualities like aggression, anger, or social pride, jealousy is normally distributed. There are those at one end of the distribution who feel little jealousy and those at the other who are consumed by it.

It is important for the clinician to be aware that wherever the individual falls on the jealousy spectrum it will feel rational to that individual. And that sudden outburst of jealousy might be resulting from changes in a partner's behavior that had been previously suppressed. Despite these overall stable individual differences, it is also true that jealousy is sometimes relationship-specific or context-specific within

relationships. A man involved with a flirtatious sexually provocative woman might be jealous with her due to the frequent male sexual attention she garners, but not when shifting to a relationship with a more introverted, less flirtatious woman. A woman whose husband received a dramatic job promotion might experience a sudden surge of jealousy, but the emotion might dissipate entirely when he loses his job.

When a couple seeks help, it is always best to conduct the first session seeing each member separately. All couples ultimately seek help because of some failure of communication, and psychotherapy at its essential core serves to facilitate communication (Abrams, 2012). Indeed, if both members of a couple were able to perfectly communicate their perspectives and articulate a means to change or improve the relationship, a therapist would not be necessary. In addition to communication per se, couples also seek for barriers to communication, such as disputes over the accuracy of events and actions (Loftus, 2007). Partners confabulate, spin, distort, advocate, and even lie when communicating with one another. These distortions often become deeply ingrained and are an essential topic of counseling. With the safety of confidentiality each member can more comfortably reveal concerns or actions that may have been withheld from the partner.

Among the concerns that the therapist is evaluating is the legitimacy of the jealousy. Humans have evolved means to detect cheating or deception in others. So the therapist needs to ascertain whether the jealous partner is overzealously protective or whether he/she is sensing behaviors that overlie diminishing commitment in their partner. If this is the case, the therapy needs to shift from jealousy as a primary problem to jealousy as a symptom of other problems with the relationship. However, if the relationship is being impeded by a partner whose suspicions are not based on changes to relationship or actual deceptions, the goal is to illuminate source of the distortions or exaggerations of the jealous partner.

The paradoxical aspect of problematic jealousy is that jealous partners sometimes are undermining the relationship they feel compelled to protect. A little jealousy can be beneficial, but extreme jealousy wreaks havoc on relationships. The joint session will have both partners taking time to discuss what they see as the problem in the relationship. In most cases, the jealous partner will complain about the inappropriate actions that are inciting their suspicions. And the partner under suspicion will complain of the distrust, accusations, and restrictions coming from the jealous partner. It is generally best to start with the accused partner to clarify that, despite the averseness of living under suspicion, it is not unbearable. They need to be shown that they are free to ignore the accusations, curtail commitment, or even leave the relationship. They will tend to believe that it is grossly unfair to be falsely accused or that it is deep violation of the relationship not to be trusted. In response, they can be guided to see that their jealous partner has elevated them to an exalted status by making their potential loss an obsessive fear. In justifying the jealousy this way, the jealous partner is also being made aware of their own jealousy.

RE/CBT for Infidelity

The anguish induced by infidelity is not assuaged with the knowledge of its high prevalence (Barash & Lipton, 2001) or its evolutionarily nature. As with jealousy, the victim of infidelity can seek help individually or as part of a couple. Working with people who have suffered infidelity differs in kind from those troubled solely by jealousy. Those in a jealous relationship will be troubled by emotions that anticipate a dreaded event, while the victim of infidelity will suffer passions that ensue after the aversive event has actually occurred. Consequently, RE/CBT for these occurrences will require different strategies, and will be addressed separately below.

RE/CBT for Couples with an Unfaithful Partner

It is not unheard of for the clinician to treat a couple in which both partners were unfaithful, but this is unusual. The modal case is a couple in which one partner has been unfaithful, so we will focus on this more typical case. It is important to note that there are differences in treating infidelity in Lesbian, Gay Male, and Heterosexual couples (e.g., Kleinplatz, 2012). The cultures that these dyads arise from tend to have divergent perspectives on infidelity that will impact the response and subsequent treatment of people from the cultures. However, the RE/CBT approach can be applied, with minor modifications, to all groups.

Each member of the couple is seen separately to assess for level of anger, alienation, allegiance to the relationship, the individual's willingness to move on or change their unfaithful behaviors, and commitment to the counseling process. If the therapist determines that the relationship remains viable then the treatment process can continue. In contrast, if it is made clear that the factors that led to the infidelity are intractable or either partner makes clear that the alienation is too great for the relationship to continue, then the therapist must directly address this in the next session. During this subsequent session, the therapist must explicitly enumerate the reasons why the couple's relationship is no longer viable, and make the case that it seems that the purpose of seeking counseling by one or both partners was to facilitate an exit from the relationship. If the therapist is correct, one or both partners will readily accept the judgment. Conversely, if the therapist is not correct, then one or both partners will advocate for the continuation of therapy. In this case, the therapist is obliged to continue the treatment process until he or she, or the demurring couple, is proven wrong.

In the event that both partners support the continuation of the relationship, the next step is to address the negative emotions that invariably persist after the infidelity. In general, men are generally less willing to pardon sexual infidelity and women are less willing to pardon emotional infidelity (Shackelford et al., 2002). These evolved inclinations underlie cognitions similar to the following:

- "If he loves someone else, he cannot ever really love me," or "he completely lied about ever loving me."
- "If I stay with him/her, I am making a horrible mistake as I am endorsing terrible behavior."
- "If she slept with another man, I cannot ever trust her again."
- "If my partner was involved with another person, I am forever at risk of being a complete fool."
- "I cannot bear that my partner cheated with is still around to make it happen again."
- "It is terrible that I must forever be vigilant against my partner cheating again."

These distorted cognitions are not the unique pathology of the individual but, instead, are a modern evolutionary expression of millennia of development. Clients receiving RE/CBT should be made aware of the unnecessary dread that our adaptions evoke in us. The man no longer has to fear losing resources by unknowingly raising another man's offspring, since modern birth control and paternity testing technology all but rules this possibility out. Most modern women no longer have to fear desperate impoverishment for her and her offspring if her partner leaves for another woman. In most contemporary societies around the world women can function without male support; they can work and, in dire situations, their children can receive societal subsidies. These realities that belie the historical dangers of infidelity need to be discussed with the clients.

Of course, the partners in the relationship will not immediately renounce their despair or anger, but acknowledging the realities of contemporary human life versus those of their ancestors, will force them to examine the basis of their distress. The therapist will help the clients to verbalize the negative emotions that they are feeling. In doing so, the individuals will begin to understand the cognitive narratives to which they may be clinging—narratives that likely made more adaptive sense in ancestral than in modern environments.

RE/CBT for an Individual Who Suffered Infidelity

Recently, individuals who have discovered that their significant other has been unfaithful are likely to have done so as a result of the increased ways to uncover deception of a partner (Abrams, 2016). The Internet provides both greater access to extra-pair relationships, such as through internet dating sites, and many more ways to discover them, such as through cyberstalking. In addition, the vast number of sexual connection websites and social media like Facebook, Twitter, and LinkedIn provide many means to find connections that can ultimately become sexual. Text messages, browser histories, social media communications, and emails all leave traces for a deceived partner to discover that their fears are realized (Mitchell, 2007).

When a person discovers that a partner has been unfaithful, their distress tends to be proportional to the trust and love that has been devoted to the offender. As a

result, counseling is most often sought by the individual who was deeply committed to the relationship and strongly believed that their partner was similarly committed. Victims of a partner's infidelity commonly suffer both grief and rage, sometimes alternating between the two. The goal of therapy is to elicit the basis of the client's feeling of damage to their self-worth, and the feelings of loss regarding the offending partner.

Aggrieved individuals tend to lament their own past failures or current diminished worth connected with the infidelity. If the relationship is irreparable, the individual will commonly mourn it as an irretrievable loss. A client afflicted by infidelity will often be simultaneously enraged at, and desirous of, their partner—splitting (or black and white thinking) is not the exclusive domain of the borderline personality and commonly occurs in distressed individuals (van Rijsbergen, Kok, Elgersma, Hollon, & Bockting, 2015).

Many people in extreme interpersonal distress will tend to alternate from idealization to rebuke in their attempts to fathom the behavior of their lovers. The individual sufferer of infidelity commonly feels shamed, angry, and even depressed. As with anyone suffering a great personal loss, the distress is generally in direct proportion to both the perceived importance of the loss, and with the perceived unfairness of their infidelity. A person strongly committed to, and deeply in love with unfaithful partner will be far more distraught than one with a more casual relationship. Such strong negative emotions are often associated with cognitions that generalize the event to all aspects of the person's life, including their future and self-worth. The evolutionary aversion to infidelity plays a major role in the common tendency to catastrophize the event.

The therapist needs to make every effort to acknowledge the client's anguish, but must then help them view it as circumscribed loss. One way to do so is help the client see the loss of a fidelity as a loss akin to any other loss—one that is sad, but not completely destructive. That client can be directed to take an economic view of the event, such that infidelity can be likened to stealing from a relationship. Trust, sexual resources, and intimacy were purloined from the deceived partner. If it is likened to any other pilfering in another kind of trusted relationship—such a commercial partner stealing from a business, it will be easier to discern the cognitive distortions that are arising from evolutionary inclinations. The intensity and range of negative emotions with sexual infidelity are far greater than if they were deceived by even the most trusted business partner.

The client is then guided to explore negative emotions that are painful or dysfunctional with the goal of finding the values, demands, or beliefs that underlie them. The individual who was a victim of the infidelity may express that it feels wrong or risky to trust a partner who has strayed. This aspect needs to be openly discussed by both parties with the goal of explaining to both parties that the infidelity, although wrong, was not a maximally bad action. Rather, it is our psychological adaptations feel catastrophic. This will become apparent with probing or Socratic inquiries about the viewpoints that underlie their most painful emotions. For example, in the case of the client who experiences anxiety about infidelity, exploratory

questions will help clients clarify that the anxiety overlies judgments about their situation.

Inquiries such as the following will open up lines of discussion that will allow the RE/CBT therapist find and challenge to client's beliefs that are exacerbating or prolonging his/her misery.

- "It seems that it will be impossible for you to even trust anyone again"
- "Do you think that if this relationship ends, you will be alone forever?"
- "If a person is deceived by someone he loves it means he can never be completely loved."
- "If your partner cheated it must mean that you were an inadequate human being."
- "Apparently, you will never be able to function in life, without getting retribution for being deceived."
- "Your lover's dedication to you is the only basis for determining you value as a human being."

In most cases, even the most distraught clients will not overly affirm the beliefs and attitudes that underlie their heartache. They will be both distraught and angry, and in more emotional states will tend to vacillate between wanting retribution and wanting their lover back. In the discussions that such inquiries will provoke the client can be shown that their lover did something bad, but is not an atrocious human being. Their overt or tacit belief that "my partner absolutely should have been faithful," can be guided to "I would have deeply preferred by my partner's fidelity." Similarly, the belief that "I cannot stand that this happened to me," can be shifted to "I am deeply hurt and disappointed by this disloyalty, but I am fully equipped to move on in life."

The RE/CBT therapist can also use imagery techniques to have the client see themselves in a better situation, and in a time when they are not distraught. Their anxiety and anger can be attenuated with relaxation techniques that guide them to focus on the current moment. This is particularly import to clients who are ruminative about their lover's behavior. They believe that they have been irrevocably damaged by the infidelity and will act on the delusory belief that recapitulation will somehow change the past. The client will also be helped by performing assignments in which they keep a log in which they challenge in writing any thoughts they have that support that their being betrayed by a loved one represents an irretrievable loss.

Summary of Evolutionary RE/CBT for Jealousy or Infidelity

The inclusion of an evolutionary perspective adds clarity and focus to cognitive behavioral interventions for both jealousy and infidelity. The evolutionary view removes much of the pathologizing and moralizing associated with both as seen in many clinical publications that treat jealousy as a pathology (e.g., Mullen, 1996; Stockdale et al., 2015). When seen as evolutionary inclinations that are most adaptive for a different epoch or setting, the therapist can change the focus from treating

an aberrant behavior to helping the client see its self-defeating nature. Telling concerned lovers that they should abandon their neurotic jealousy is as effective as telling someone that fearing a war zone is foolish. The jealous person feels their jealousy is protective and judicious, and they will not relinquish it easily. Excessively jealous clients so fear the loss of the relationship that they will destroy it with hypervigilance. It is this self-defeating aspect of jealousy that RE/CBT most effectively target.

The therapist faces conceptually similar problems in dissuading infidelity. The unfaithful client trades short-term sexual pleasures for the benefits of an enduring relationship, although sometimes infidelity functions as a mate-switching tactic (Buss et al., 2017). And in risking the enduring relationship, there is attendant emotional harm that regularly ensues. Rather than moralize or invoke cultural mores, the RE/CBT therapist educates the client to the evolutionary logic of evolved emotions and desires and their possible irrationality in the modern environment. It is irrational because the overall costs of maintaining a disingenuous relationship is greater than the costs of the two alternatives: leave the relationship and seek novel partners or stay in the relationship that offers benefits greater than sexual variety.

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Evolved Vulnerability to Addiction: The Problem of Opiates

Elizabeth M. Hill, Lindsey Hunt, and Daniel G. Duryea

According to the Centers for Disease Control and Prevention (CDC), 2015 marked the record for drug overdoses in the United States as more than 33,000 individuals died from an overdose of heroin or synthetic opioids, with nearly half of these deaths attributed to prescription opioids. CDC estimates indicate that 60% of all drug overdose deaths involve opioids. Since 1999, opioid overdose has nearly quadrupled with more than half a million deaths documented between 2000 and 2015. Current estimates suggest that 91 citizens perish daily from opioid overdose (CDC, 2016).

Increased availability of both licit and illicit forms of opioids has been attributed to the escalation in both use and deaths (Friedman, 2014). The prescribing of and misuse of opioid drugs has increased significantly in the United States. For example, 174.1 million prescription opioids were prescribed in 2002; this number increased to 256.9 million by 2009. In 2014, 4.5 million people in the United States aged 12 and older indicated that they used a prescription pain medication for a nonmedical issue in the past month, and 289 thousand people indicated they used heroin in the last month (SAMHSA, 2014). As the cost of procuring nonmedical prescription drugs has increased, the availability of less expensive street heroin has increased, thus expanding numbers of addicted users and potential overdoses (Center for Substance Abuse Treatment [CSAT], 2005). To combat the dangers of prescription opioids and overdose the Food and Drug Administration (FDA, 2016) now requires "boxed-warnings" emphasizing risks for accidental overdose and addiction potential when taking opioids and benzodiazepines.

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These efforts by the FDA to combat the dangers of prescription opioids and the risk of overdose promise to be effective eventually, and new intervention and treatment methods are promising. However, risk remains high. This state of affairs places significant emphasis on the importance of available treatments to save lives and prevention initiatives to reduce the significant demand for opioids in the population.

Given the current demand for opioids and risks of addiction, it is imperative to understand the etiology of addiction and psychopharmacological function of opiates. A broad perspective may enable better understanding and eventually lead to better treatment and prevention approaches. We need to marshal all areas of knowledge about addiction from all possible sources in order to help understand and intervene. This chapter takes an evolutionary approach to augment traditional perspectives.

An evolutionary perspective could add to our understanding of addiction, but it must be stated that addiction has no adaptive value; rather, humans have evolved processes and mechanisms for other purposes, which allow vulnerability to addiction (Nesse & Berridge, 1997). An evolutionary perspective helps explain how we are neurologically and behaviorally susceptible to addiction. Brain substrates for normal motivation and emotion use chemical neurotransmitters, which enable other nonnatural chemicals to engage these pathways. Nesse (2016) summarizes:

> Vulnerability to substance abuse results from our novel environment. The reliable availability of pure chemicals and clever new routes of administration increase the rate of drug taking. Tobacco administered via the technological advance of cigarettes is the most widespread and harmful addiction, with alcohol a close second. The so-called hard drugs of abuse, such as amphetamines and cocaine, act even more directly on ascending dopamine tracts to establish addiction. Substance abuse is a universal human vulnerability to drugs that hijack reward mechanisms. (p. 1017).

Here we take an evolutionary perspective to understand the current epidemic of opiate addiction. Understanding opiate addiction involves understanding many aspects of motivation, emotion, brain pathways, and neurotransmitters, which will be described. One area of research that has led to greater understanding of opiate addiction is research on the neuroscience of distress calls of neonatal rodents. When rodent pups are separated from the home nest, they emit ultrasonic distress calls. These are above 35 kHz (Branchi, Santucci, & Alleva, 2001), not audible to human ears. Upon hearing these calls, a mother rat or mouse will investigate the sound location, pick up the pup in her mouth, and retrieve it to the nest (Noirot, 1972; Sewell, 1970). Rodent pups reliably make these calls when they become cold (Okon, 1970). They cannot thermoregulate until about 2 weeks old and thus depend upon warmth from the mother and littermates in the nest. Separation from the nest can be fatal quickly. These calls are labeled “separation distress calls.” Understanding the neuroscience of distress calls of rodents and other animals has led to a greater understanding of opiate addiction in humans (Panksepp, 1998). The common link is the endogenous opioid system in the mammalian brain, which subserves response to pain and also motivates distress calls. The evolution of these brain systems is critical to survival, but the system is vulnerable to drugs of addiction that mimic natural opioids and provide stronger relief of both physical and emotional pain than endogenous opioid activity.

This chapter will propose that the involvement of the opioid system in social attachment and physical pain contributes to the current prevalence of addiction to opiate drugs. The process and course of addiction will be described. Then we will briefly review the brain systems involved in opiate addiction and note the common opioid drugs of abuse. Next, we will describe factors that may explain why opiate addiction has become such a problem today. An evolutionary perspective is helpful in understanding addiction (Nesse & Berridge, 1997), as this perspective led to the current understanding that physical and social pain use the same brain pathways (Panksepp, 1998). We will present recent evidence for the role of opioid brain systems in social and physical pain. These vulnerabilities are shared by all human beings. However, research has shown that individual differences exist in the opioid systems in the brain that contribute to both types of pain. Some of the most important research will be reviewed. Lastly, we will describe promising avenues of treatment, intervention, and prevention of opiate addiction.

What Is Addiction?

Addiction is considered to be a medical disease involving changes in brain pathways once addiction has taken hold (the "hijacked brain"). Considered a treatable medical disorder, drug use disorder is considered a brain disease instead of a moral failing or lack of willpower (CSAT, 2005).

Substance Use Disorders

According to the American Psychiatric Association (APA), Substance Use Disorders (formerly referred to as addictions) are disorders related to an individual continuing to use a specific substance despite experiencing significant problems related to this substance (APA, 2013). These problems fit into four areas: impaired control, social impairment, risky use, and pharmacological criteria. The severity of a given Substance Use Disorder is rated either mild, moderate, or severe based on the number of symptoms the individual is experiencing. *Impaired control* refers to the influence that the substance exerts on an individual's life. Examples include: time devoted to activities related to obtaining, using, and recovering from the substance; more substance being used than the individual intended; or the individual's inability to stop or reduce the amount of the substance despite efforts to do so. *Social impairment* refers to negative changes in behavior that are related to substance use. This includes the individual failing to meet obligations at work, home, or school; the individual withdrawing from activities that were previously important or pleasurable for the individual; or the individual continuing to use the substance despite use continually resulting in interpersonal problems. *Risky use* refers to the use of the substance in dangerous situations (e.g., driving while intoxicated), or the individual

continuing to use the substance despite having knowledge that the substance will have a negative effect on their physical or psychological problems. Finally, the area of *Physiological criteria* refers to the individual experiencing tolerance (i.e., needing to consume more of the substance to have the same physiological effect) and/or withdrawal (i.e., symptoms related to a reduction in the concentration of the substance in the individual's body). Substance Use Disorder cannot be diagnosed based on physiological criteria alone if the substance is being used appropriately to treat a diagnosed medical condition and the individual has a valid prescription for the substance. However, if an individual being treated for a medical condition begins to meet other criteria for Substance Use Disorder, then they will meet the diagnostic criteria despite having a valid prescription for the substance (APA, 2013).

Opioid Use Disorder (OUD)

Considered a treatable medical disorder, OUD is considered a brain disease instead of a moral failing or lack of willpower (CSAT, 2005). By virtue of opioids' pharmacodynamic and pharmacokinetic properties, opioids are extraordinarily reinforcing (i.e., addictive) both psychologically and physiologically over a relatively short period of regular use. The initial "rush" following opioid ingestion is experienced by the user as an intense euphoria and, therefore, becomes promptly positively reinforced. Typically, abused street and nonmedical prescription drugs are short acting (i.e., 4–6 h) and lead to increasing levels of discomfort as they are metabolized and diminish, characteristic of opioids' withdrawal syndrome. Withdrawal severity is moderated by tolerance and frequency of administration of the drug. Opioids become rapidly negatively reinforced through a conditioned avoidance of withdrawal as use progresses. Opioid use is further complicated by a simultaneous increasing tolerance to the initial euphoric effects of the drug. In combination, these factors make intentional behavior change (i.e., cessation) of opioids considerably more difficult when compared to other Substance Use Disorders (SUDs).

OUD is distinctively characterized from other SUDs in several respects. Opioids are available as licit substances in the form of prescription analgesics (e.g., oxycodone, hydrocodone, Percocet) or illicitly (e.g., heroin). In addition, nonmedical use of prescription drugs, especially for purposes of abuse, is deemed illegal. Opioids, unlike other commonly abused substances (e.g., alcohol, tobacco, cannabis, and cocaine), can be consumed through multiple routes of administration (e.g., intranasal, inhalation, rectal [mucosal], subcutaneous, transdermal, intravenous). Changes in routes of administration often indicate an intensification of addiction, for example, when intranasal use is substituted by methods of inhalation or intravenous administration.

Unlike alcohol and other common drugs of abuse, tolerance effects for opioids typically occur rapidly necessitating ever-increasing amounts of the substance. This is naturally followed by a characteristic withdrawal syndrome with severity predicated upon frequency and quantity of opioids used. The characteristic withdrawal

syndrome includes three or more of the following: dysphoria, nausea, tearing, runny nose, sweating, gooseflesh, dilated pupils, diarrhea, yawning, fever, and insomnia (APA, 2013). It is common for tolerant users avoiding withdrawal to experience financial problems maintaining a steady supply, in addition to engaging in protracted drug-seeking behavior.

The characteristics of opiate addiction are illuminated by understanding the brain pathways that are involved in this type of addiction. Brain pathways that are hijacked by opiate drugs are normally responsible for inhibiting pain.

Opioid System in the Brain

Addiction involves multiple pathways and brain areas. The dopaminergic mesolimbic reward pathway is best known and is implicated in most addictions. It travels from the midbrain ventral tegmental area (VTA) to the nucleus accumbens (NAcc). Stimulant drugs directly affect this pathway, while other drugs of abuse such as opiates increase dopaminergic transmission in this pathway more indirectly (reviewed in Nestler, Hyman, & Malenka, 2009). Here we focus on opioid systems and their interconnections with other systems. In one conceptualization, the DA reward pathway mediates approach to rewards (appetitive motivation), while consumption (which would terminate approach) is mediated by opioid pathways (Berridge, 1996; Robinson & Berridge, 1993). Stimulation of the DA reward pathway/median forebrain bundle is intrinsically reinforcing, as demonstrated by studies of rodents taught to bar press for brain stimulation (Olds 1977). Consumption of a reward (e.g., food or sex) never occurs, but rather compulsive self-administration is never-ending (as described by Panksepp, Knutson, & Burgdorf, 2002).

A major job of the opioid system in the brain is to inhibit pain (Meyer & Quenzer, 2013). At the spinal cord level, when pain occurs small inhibitory spinal interneurons release endorphins that reduce the likelihood of the pain signal being sent up to the brain by spinal projection neurons (Meyer & Quenzer, 2013). Pain is also regulated by descending modulatory pathways that are predominately found in the periaqueductal gray (PAG) area in the brain. Through these pathways, pain can be regulated in three different ways: (1) by inhibiting the spinal projection neuron, (2) by inhibiting excitatory interneurons that synapse on the spinal projection neuron, and (3) by exciting the small inhibitory spinal interneurons that inhibit the spinal projection neuron (Meyer & Quenzer, 2013). Pain can further be modulated by higher brain structures, including the cerebral cortex, medial thalamus, and the hypothalamus, which send neuronal projections to the PAG (Apkarian, Bushnell, Treede, & Zubieta, 2005; Meyer & Quenzer, 2013). Specific regions of the cerebral cortex that are considered to be part of the perception of acute pain include: primary and secondary somatosensory, insular, anterior cingulate, and prefrontal cortices (Apkarian et al., 2005). Thus, one of the functions of the opiate system is to lessen or stop pain signals transmitted to those areas.

Secondary pain affect is a term for how an individual imagines prolonged pain will interfere with his or her life (Price, 2000, 2002). It involves planning for how the pain will affect an individual's life, but may lead to rumination. Thus, individuals with particular personality disorders have been found to have greater secondary pain affect (Price, 2002). The anterior cingulate cortex (ACC) is believed to be the brain area where there is coordination between the immediate threat of pain in the parietal cortex with secondary pain affect in the prefrontal cortex (PFC; Price, 2002).

However, when it comes to opioids, pain is not where the story ends because opioids also promote feelings of euphoria and well-being (Meyer & Quenzer, 2013). These euphoric effects explain why individuals take opioids recreationally (Mestek, Chen, & Yu, 1996). Another issue with opioids is that their use is highly reinforced, which easily leads to the development of Opioid Use Disorders, even in individuals who are prescribed an opioid for a valid medical reason who originally only intend to take the drug as prescribed. The reward and reinforcement pathway in the brain was mentioned earlier, the dopaminergic mesolimbic pathway (Meyer & Quenzer, 2013). Evidence supports that opioids achieve reinforcement in this pathway by inhibiting neurons that prevent/reduce the firing of the VTA neurons (Meyer & Quenzer, 2013). Thus, by inhibiting the neurons that inhibit the VTA neurons, the VTA neurons that project to the NAcc are able to fire more often, and release more dopamine on the receptors of neurons in the NAcc, which results in positive reinforcement (Meyer & Quenzer, 2013).

Thus, opiate drugs affect the same reward pathways as other addictive drugs, but opiates have other effects that add to the addiction potential of these drugs. These effects will be described further below. First, the main types of opioid receptors will be described.

Opioid Receptors

Four opioid receptors have been discovered. These include the mu (μ), delta (δ), kappa (κ), and nociceptin/orphanin FQ (NOP-R) opioid receptors (Meyer & Quenzer, 2013). These receptors evolved to respond to different classes of ligands that the body makes: endomorphins, endorphins, enkephalin, dynorphins, and nociceptin/orphanin FQ (Meyer & Quenzer, 2013). Each of the opioid receptors has a class of ligands that binds to it more strongly than it does to the other opioid receptors, but there is overlap between which ligands activate each receptor (Goldstein, 1987). For example, the μ-opioid receptor has the highest binding affinity for endomorphins and endorphins, but studies have found that enkephalins and dynorphins can also activate these receptors at physiological levels, each of which more strongly binds to delta opioid receptors and kappa opioid receptors, respectively (Mestek et al., 1996; Meyer & Quenzer, 2013). However, despite strong genetic similarity between the NOP-R receptor and the other opioid receptors, NOP-R neither binds traditional opioid ligands, nor does its ligand, nociceptin/orphanin FQ, bind to the three other opioid receptors, suggesting a unique role for this receptor and its ligand

(Meyer & Quenzer, 2013). Of the receptors, the μ-opioid receptor has been studied the most in connection with opioid use disorders, as it has the strongest affinity for opioid drugs (Mestek et al., 1996). However, it is important to keep in mind that the opioid system as a whole is affected by use of opioids.

Based on receptor distribution in the brain and genetic knockout studies, evidence suggests that μ-opioid receptors have many different roles, such as in analgesia, respiratory/cardiovascular depression, nausea/vomiting, and sensorimotor integration (Meyer & Quenzer, 2013). Additionally, μ-opioid receptors have a complicated role in mood regulation, as the activation of μ-opioid receptors is associated with euphoria and relief from depression, whereas loss of these receptors in rodents is associated with reduced anxious and depressive symptoms (Filliol et al., 2000; Lutz & Kieffer, 2013a; Yoo, Lee, Loh, Ho, & Jang, 2004). Similar to μ-opioid receptors, δ-opioid receptors are associated with analgesia and positive reinforcement, but δ receptors are also found in higher brain structures and have additional roles in olfaction and cognition (Meyer & Quenzer, 2013). κ-Opioid receptors have distinctive roles from μ and δ receptors, as they regulate homeostatic mechanisms, such as eating, drinking, temperature control, and also produce hallucinations and dysphoria (Meyer & Quenzer, 2013). Studies suggest that the reinforcing properties of opioids are caused by μ- and δ-opioid receptors, whereas κ-opioid receptors were found to not be reinforcing, and to be possibly aversive (Shippenberg, 1993).

Drugs that Are Opioid Agonists

The endogenous ligands of the opiate system include: β-endorphin, enkephalins, and dynorphins (Lutz & Kieffer, 2013b). However, there are many natural and synthetic drugs that have been discovered or created to activate this system, which cause an array of different desired effects and symptoms. These effects include: analgesia, slowing of the gastrointestinal system, cardiovascular depression, nausea/vomiting, euphoria, calming, anti-depressant functions, suppressing coughing, dysregulation of homeostatic mechanisms, and reduced sex drive (Lutz & Kieffer, 2013b; Meyer & Quenzer, 2013). Opium was the first opiate used for medicinal, recreational, and ceremonial purposes. Natural components found in opium have been extracted and purified to be used as drugs that have more potent analgesic properties and/or fewer side effects than opium. The naturally derived opiates include morphine and codeine, of which codeine is less effective at reducing pain, but has fewer side effects than morphine (Meyer & Quenzer, 2013). Synthetic modification of the components in opium, as well as the synthetic production of molecules that bear structural similarity to these compounds, has given rise to a whole host of different medically available opioids that have an even greater number of street names (Table 1). Additionally, opioid partial agonists and opioid antagonists have been developed as less potent drugs and used in opioid replacement therapy for Opioid Use Disorder (buprenorphine and methadone), and to reverse the effects of opioid overdoses (e.g., naloxone) in order to save lives, respectively (Meyer & Quenzer, 2013).

Table 1 Opioid agonist drugs

Drug name	Street names	Source
Codeine (many brands)	Captain Cody, Cody, Lean, Schoolboy, Sizzurp, Purple Drank*With glutethimide:* Doors & Fours, Loads, Pancakes and Syrup	Prescription
Fentanyl (Actiq®, Duragesic®, Sublimaze®)	Apache, China Girl, China White, Dance Fever, Friend, Goodfella, Jackpot, Murder 8, Tango and Cash, TNT	Prescription
Heroin	Brown sugar, China White, Dope, H, Horse, Junk, Skag, Skunk, Smack, White Horse *With OTC cold medicine and antihistamine:* Cheese	Recreational drug
Hydrocodone or dihydrocodeinone (Vicodin®, Lortab®, Lorcet® etc.)	Vike, Watson-387	Prescription
Hydromorphone (Dilaudid®)	D, Dillies, Footballs, Juice, Smack	Prescription
Meperidine (Demerol®)	Demmies, Pain Killer	Prescription
Methadone (Dolophine®, Methadose®)	Amidone, Fizzies *With MDMA*: Chocolate Chip Cookies	Prescription
Morphine (Duramorph®, Roxanol®)	M, Miss Emma, Monkey, White Stuff	Prescription
Opium	Ah-pen-yen, Buddha, Chillum Chinese Molasses, Chinese Tobacco, Fi-do-nie, Gee, Goric, Pen yan, Pin gon, Pin yen, When-shee, Yen Shee Suey, Ze	Recreational drug
Oxycodone (OxyContin®, Percodan®, Percocet®, and others)	O.C., Oxycet, Oxycotton, Oxy, Hillbilly Heroin, Percs	Prescription
Oxymorphone (Opana®)	Biscuits, Blue Heaven, Blues, Mrs. O, O Bomb, Octagons, Stop Signs	Prescription

Reference:
http://www.opium.org/opium-street-names.html
https://www.drugabuse.gov/drugs-abuse/commonly-abused-drugs-charts

Why Are Opiates an Epidemic Problem Currently?

Opiate addiction and overdose are an epidemic and increasing problem for several reasons. First, medical prescriptions lead to nonmedical use, which leads to use of illicit drugs. Secondly, opiates are strongly addictive due to sensitization of the brain pathway subserving natural reward. Third, and less well known, is that opiates impact our brain systems related to social attachment in addition to impacting the brain reward system common to most addictive substances. It is the many effects on social attachment that may give opiate drugs a special appeal that is difficult to replace.

Addiction to substances involves progressively stronger craving of them, in addition to the phenomenon of tolerance and withdrawal symptoms. Systems that respond to addictive drugs appear to become sensitized after repeated exposure, where the desire for a drug increases rather than decreases (Robinson & Berridge, 1993). The mechanism

involves repeated activation of the mesolimbic pathway, which causes a process of long-term potentiation of glutamine synapses on VTA dopamine neurons (Nestler et al., 2009). Sensitization appears to make drugs and associated stimuli more attractive, a state that can persist for a long time and contribute to relapse after a period of abstinence.

As noted earlier, there is widespread exposure to opiates through prescription painkillers; as prescriptions become unavailable or too expensive individuals often begin to use heroin. The director of the National Institute of Health recently asserted several reasons for the growing epidemic of opioid use—the number of prescriptions being written by doctors has increased dramatically over the past two decades, the increasing acceptability of using substances recreationally in the United States, and aggressive marketing by pharmaceutical companies (Volkow, 2014).

A recent survey of American high school seniors in the Monitoring the Future study 2009–2013 found that the frequency of nonmedical opioid use in this sample was estimated to be 12.4% (Palomar, Shearston, Dawson, Mateu-Gelabert, & Ompad, 2016). Similar lifetime prevalence was reported (13.5%) for a European Union sample aged 12–49 (Novak et al., 2016). It has been suggested that opioid misuse results from unused pain medications being stored in the family home, thus giving access of these drugs to young people who live there (Dodrill, Helmer, & Kosten, 2011). In a study by Green, Black, Serrano, Budman, and Butler (2011), 45.8% of people who developed Opioid Use Disorder had been given a prescription for a valid medical reason.

The recent increase in heroin use is related to the surge in prescription opioid use. In the 1960s, 80% of heroin users were not exposed to prescription opiates first, but in the past 20 years, 75% of heroin users were first exposed to prescription opioids (Cicero, Ellis, Surratt, & Kurtz, 2014). A recent survey of American high school seniors, mentioned above, found that recent nonmedical use of opioids strongly increased the risk of heroin use (Palomar et al., 2016). Additionally, the demographic characteristics of heroin users have changed. Over the past 20 years, the people who have begun using heroin have been older (mid-twenties), predominantly white, and living in less urban areas than previous generations of heroin users (Cicero et al., 2014). Proposed reasons for this shift are that heroin is cheaper to obtain on the street and easier to obtain than prescription opioids, despite the facts that heroin is more likely to lead to an overdose and be less pure than prescription opioids (Cicero et al., 2014).

Wright et al. (2014) conducted a study to find what county-level features drive opioid prescription misuse. They found that access to healthcare, in particular dentists and pharmacists, increased access to prescription opioids (Wright et al., 2014). Other studies have listed systemic problems in the healthcare system as helping to fuel this national health epidemic, in that prescribers were not coordinating care for painful conditions and thus unknowingly overprescribing opiates to the same individuals (Dodrill et al., 2011). A recent analysis of data from the Veterans Health Administration determined that risk of overdose death increases in a dose–response fashion as prescription doses of opioids go from 0 to >100 mg per day or higher of morphine equivalent medication (Bohnert et al., 2011). Maximum daily dose over 50 mg was associated with elevated risk compared to dosage below 20 mg.

Third, and less well known, is that opiates impact our brain systems related to social attachment in addition to impacting the brain reward system common to most addictive substances. It is the effects on social attachment that may give opiate drugs a special appeal that is difficult to replace. We will focus on this aspect next.

Brain Pathways for Social Pain Overlap with Those for Physical Pain

The proposition that animal separation distress calls helps to understand human emotion, specifically social pain, may seem to be a stretch. Extrapolating animal research to humans requires the assumption that the neural pathways underlying behaviors are homologous in animals and humans. Experts in brain evolution have concluded that older brain structures have been modified and integrated with new structures, as more complex brains evolved. Interested readers are referred to a recent series of papers by O'Connell and Hoffman (O'Connell & Hofmann, 2011a, 2011b) that describe two ancient brain circuits present in vertebrates, the mesolimbic reward system and the social behavior network. These two circuits form part of a large social-decision-making network that is homologous among vertebrates (mammals, birds, reptiles, amphibians, and teleost fish; O'Connell and Hoffman, O'Connell & Hofmann, 2011a, O'Connell & Hofmann, 2011b).

Panksepp (1998) analyzed the brain substrates of separation distress, providing evidence that the emotion of separation distress uses the same brain substrates as physical pain, involving the opioid receptor system. Animal research supporting this conclusion dates to an early study by Panksepp, Herman, Conner, Bishop, and Scott (1978) showing that low doses of morphine reduced separation distress vocalizations in 6–8 week old puppies in a dose-dependent fashion.

Panksepp (1998) describes four primary emotion systems, one of which is called "PANIC." This basic emotion is associated with social loss and separation, subserved by neural pathways originally processing thermoregulation and pain. Nelson and Panksepp (1998) proposed that social pain messages are sent via pathways for physical pain, a neurological arrangement that facilitated social responses important for survival, such as making distress calls when separated from one's mother. Panksepp (1998) proposed that the PANIC system arises from the midbrain PAG, close to the area where electrical stimulation results in physical pain (p. 267). Separation distress calls can be obtained by stimulation of the PAG, and in the ventral septal area, the dorsal preoptic area, the dorsomedial thalamus, the bed nucleus of the stria terminalis, and the anterior cingulate cortex (Panksepp, 1998, 2003). There is evidence from a variety of species that morphine reduces distress calls and naloxone increases these (from chicks [Panksepp, Bean, Bishop, Vilberg, & Sahley, 1980] to sheep [Shayit, Nowak, Keller, & Weller, 2003]). Other neurochemicals are also involved in social bonds. Panksepp (1998) states that, in addition to opioid systems, the prime substrates of social bonds in mammals are oxytocin and AVP (p. 259). The brain pathways important for physical pain and social attachment will now be described.

Research on social pain in humans has been conducted, complementing research with animal separation distress vocalizations. Figure 1 shows the brain pathways relevant to pain, with three types of cross-hatching to distinguish those areas that are considered to process physical pain, pain affect, and social pain. This diagram is adapted with permission from Apkarian et al. (2005) and Price (2000).

Areas that subserve physical pain include the PAG, the somatosensory cortex (S1), the thalamus (medial), and the hypothalamus, along with spinal neurons (Price, 2002). As stated earlier, pain pathways involving the PAG can be modulated by higher brain structures, including the cerebral cortex, medial thalamus, and the hypothalamus, through neuronal projections to the PAG (Apkarian et al., 2005; Meyer & Quenzer, 2013).

The affective dimension of pain was described by Price (2000) as being processed in the amygdala, the anterior cingulate cortex (ACC), and the insula. These areas are shown with a different type of shading in Fig. 1. Intense physical pain that causes distress activates areas in the brain associated with pain, as well as the areas associated with the affective dimension of pain. Price (2002) viewed the ACC as associated with pain unpleasantness, as it receives multiple inputs and has connections with the prefrontal cortex, enabling cognitive evaluation of pain and related emotion.

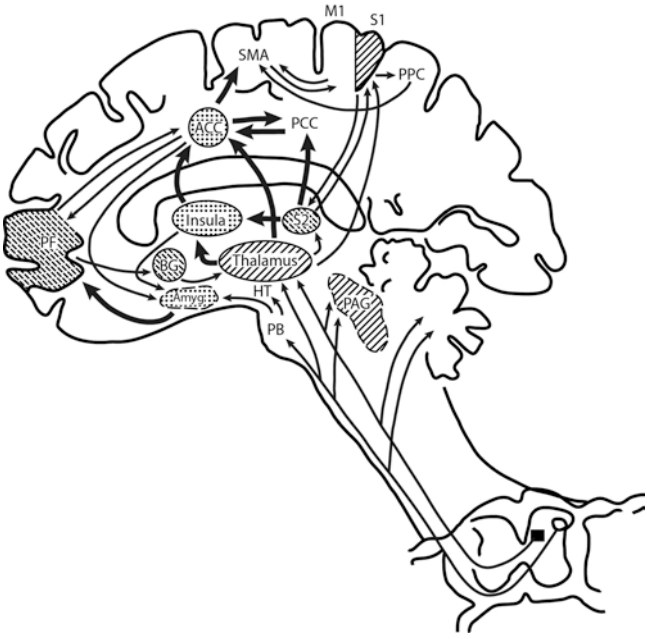

Fig. 1 Diagram of brain with pain-processing pathways indicated. Three types of shading indicate pathways related to physical pain (*diagonal hatching*), the affective dimension of physical pain (*stippling*), and social pain (*dashed hatching*). Names of brain structures are abbreviated: *PAG* periaqueductal gray area, *PB* parabrachial nuclei, *HT* hypothalamus, *AMYG* amygdala, *ACC* anterior cingulate cortex, *PCC* posterior cingulate cortex, *PPC* posterior parietal cortex, *S1* somatosensory area, *S2* secondary somatosensory area, *M1* motor cortex, *SMA* supplementary motor area, *PF* prefrontal cortex. Redrawn with permission from Apkarian et al. (2005) and Price (2000)

The proposition that animal separation distress will help us understand human emotion requires evidence that human social bonding and attachment is similar to that of animals, and the assumption that separation distress/social pain in adult humans can be induced and measured in a laboratory setting. The third type of shading in Fig. 1 shows areas related to social pain, assessed in the laboratory as pain from social rejection.

Researchers have recently begun studying social pain in laboratory experiments with humans (reviewed by Eisenberger, 2012, 2015). In the first study of its kind, Eisenberger, Lieberman, and Williams (2003) used fMRI to determine that the brain regions activated by social pain are similar to those activated by physical pain. Heightened activity in the ACC was shown when participants were exposed to social rejection (exclusion during a video game), and the amount of activity correlated strongly with self-reported distress ($r = .88$). In this study, participants initially thought they were playing the internet computer game with two others, but then the others stopped throwing them the ball. Eisenberger (2012) contends that social pain uses the same brain pathways as does the affective component of physical pain. Recent research in this and other laboratories have found similar results using various manipulations to induce social pain, such as reliving a romantic rejection (Kross, Berman, Mischel, Smith, & Wager, 2011). Indeed, with this type of intense social pain, neural substrates for the sensory component of pain were also activated (Kross et al., 2011). These authors concluded that: "brain systems that underlie social rejection developed by co-opting brain circuits that support the affective component of physical pain" (p. 6273; Kross et al., 2011).

The brain areas recruited for processing social pain are shown in Fig. 1: Secondary somatosensory cortex (S2), prefrontal cortex (PF), and BG (Basal ganglia, ventral striatum). During the experience of social pain, these areas are activated in addition to the areas responsive to pain affect (ACC, insula, and thalamus). Eisenberger (2012) described the two types of pain well:

> Physical pain is a deeply psychological phenomenon that can be altered by expectation, mood, and attention. Likewise, social pain is a deeply biological phenomenon that has been built into our brains and bodies over millions of years of mammalian evolution because of the crucial part it plays in our survival (p. 431–432).

Social Pain Pathways Involve Opioids

Brain pathways and areas responsive to pain utilize opioid neurotransmitters. Panksepp (1998) proposed that endogenous brain opioid systems regulate distress associated with separation and the pleasure that comes with social connection. Eisenberger (2012) hypothesized that brain areas related to the affective component of physical pain were coopted to "warn against and prevent the dangers of social harm" (p. 423).

The information about pain pathways and μ-opioid receptor distribution comes from various lines of research. One line of research employs brain scans to measure changes during laboratory manipulation of pain. Other research has used indirect

methods, such as testing pain thresholds. Early studies measured opioid receptor distribution anatomically. Table 2 shows distribution of three receptor types in the brain areas depicted in Fig. 1. This table is based upon in situ hybridization histochemistry using ^{33}P–labeled RNA probes in postmortem human brains (Peckys & Landwehrmeyer, 1999). Additional information is included for rat brain from Mansour et al. (1994) where human studies are lacking. It is clear that opioid receptors are widely distributed in the brain. The areas with highest concentration of μ and κ receptors appear to be the thalamus and amygdala. The δ receptor appears to be highly prevalent in many areas, including the parietal cortex, cingulate cortex, insula, and amygdala. It is beyond the scope of this chapter to describe the pain pathways and their neurochemistry in fine detail; rather we focus on the basic areas where there is a consensus about their involvement. For example, the diagram shows the ACC as one area, but we do not separate the dorsal ACC from other anterior cingulate areas, nor do we separate the lateral from the medial pain system. Research about the various sections of the ACC continues. In another example, the ACC was reported to show presence of μ-opioid receptors (MOR) at a low level with in situ hybridization (Table 2), rather than a highly dense concentration. Despite not being the highest in MOR distribution in anatomical studies (Table 2), the ACC has high opioid receptor binding potential (Baumgärtner et al., 2006). Baumgärtner et al. (2006) measured binding potential with the subtype unselective radio-ligand [^{18}F]flouroethyl-diprenorphine (which binds with MOR, KOR, and DOR with equal affinity). The fMRI studies reviewed by Eisenberger (2012) detected activity in these areas, blood-oxygenation level-dependent (BOLD) responses. Further research needs to integrate BOLD studies with those employing PET scanning methods.

Several experiments have investigated real-time changes in opioid receptor binding using PET scans in regard to emotional/social pain. Zubieta et al. (2003) conducted a study looking at changes in μ-opioid receptor binding in various brain regions when subjects were recalling a personal story that was sad versus when subjects were in a "neutral" state where they were merely asked to be aware of their physical sensations. This study found that in the sadness state there was a significant reduction of μ-opioid receptor binding compared to the neutral state in: the rostral anterior cingulate, ventral pallidum, amygdala, and inferior temporal cortex (Zubieta et al., 2003). In a similarly designed study, women with Major Depressive Disorder (MDD) and age and educational level matched control women were asked to think of a sad story during one scan, and be in a "neutral" state during another PET scan (Kennedy, Koeppe, Young, & Zubieta, 2006). This study demonstrated that women with MDD had significantly less endogenous μ-opioid receptor binding in the neutral state, and also had significantly less μ-opioid receptor binding in the left inferior temporal cortex. Matched control women had significantly more binding in the rostral ACC (Kennedy et al., 2006). More recently, a study demonstrated that social touch decreased endogenous μ-opioid receptor binding in the thalamus, striatum, cingulate cortex, insular cortex, and frontal cortex in men (Nummenmaa et al., 2016). The PET studies presented here provide a few examples of the burgeoning research investigating emotional aspects of pain. For more information on imaging and opioid receptors, see Henriksen and Willoch (2008).

Table 2 Distribution of opioid receptors in brain, based on anatomical studies of rat and human

Location	Receptor type		
	Mu	Kappa	Delta
Primary somatosensory cortex	No studies found—but +− +++ found in parietal lobe (rat)[a]	No studies found—but not found in dorsal parietal lobe (rat)[a]	No studies found—but ++++ in parietal lobe (rat)[a]
Secondary somatosensory cortex	No studies found—but + − +++ found in parietal lobe (rat)[a]	No studies found—but ++ − +++ in ventral parietal cortex (rat)[a]	No studies found—but ++++ in parietal lobe and nearby insula (rat)[a]
Prefrontal cortex	+++ Layer V (human)[b]	+++ Layer IV–V (human)[b]	+++ Layers II–IV (human)[b]
Posterior parietal cortex	+ Layers II–III, +++ layer VI (rat)[a]	++ − +++ Ventral expression layers V-VI (rat)[a]	++++ Bilaminar distribution (rat)[a]
Supplementary motor area	No studies found—part of frontal lobe	No studies found—but frontal lobe has high expression in humans[b]	No studies found—part of frontal lobe
Anterior cingulate cortex	++ (rat)[a]	None (rat)[a]	++++ Likely bilaminar distribution (rat)[a]
Posterior cingulate cortex	++ (rat)[a]	None (rat)[a]	++++ Likely bilaminar distribution (rat)[a]
Insula	+ Agranular insular cortex (rat)[a]	++ Agranular insular cortex (rat)[a]	++++ Agranular insular cortex (rat)[a]
Basal ganglia	+++ Most of basal ganglia (human)[b]	+++ Most of basal ganglia (human)[b]	+++ Only in large neurons of nucleus accumbens (human)[b]
Thalamus	++++ Most areas (human)[b]	+++ − ++++ (human)[b]	None (human)[b]
Hypothalamus	++ − +++ All areas (human)[b]	+++ Most areas (human)[b]	None (human)[b]
Amygdala	++ − ++++ In different nuclei (rat)[a]	++++ Somewhat varying in different nuclei (rat)[a]	+++ − ++++ In different nuclei(rat)[a]
Periaqueductal gray	+++ (human)[b]	++ − +++ (human)[b]	None (human)[b]
Parabrachial nuclei	+++ (human)[b]	None (human)[b]	None (human)[b]

+, scattered cells; ++, low level of cells; +++, moderate level of cells; ++++, high level of cells
[a]Mansour et al. (Mansour et al., 1994)
[b]Peckys and Landwehrmeyer (Peckys & Landwehrmeyer, 1999)

Another laboratory has examined human response to social rejection while visualizing brain activity using PET scanning with [^{11}C]carfentanil, a ligand that has high affinity for μ-opioid receptors (MOR; Hsu et al., 2013). The rejection

manipulation was false feedback on personal dating profiles being considered for possible intimate relationships. Fake feedback given during the PET scan about his or her own profile was designed to generate rejection or acceptance. There were areas of the brain that were significantly activated during rejection compared to baseline, specifically the left and right amygdala, right ventral striatum in the area of the nucleus accumbens, midline thalamus, and PAG. The authors note that this pattern is similar to the response to physical pain. During the social acceptance phase, compared to baseline, higher activation was seen in the amygdala and anterior insula, and lower MOR activation in the midline thalamus and subgenual anterior cingulate cortex. There was higher activation during rejection blocks compared to acceptance blocks in the right ventral striatum, bilateral amygdala, midline thalamus in the area of the nucleus accumbens, subgenual ACC, and dorsal ACC. Hsu et al. (2013) caution against interpreting MOR and BOLD studies together because the relationship between MOR activation has not been precisely correlated with BOLD signal in fMRI studies.

To reiterate, sufficient research supports the involvement of opioid systems in social pain, not just physical pain. Controversies remain because of differing methods and interpretations among numerous studies. In addressing these controversies, Eisenberger (2015) concludes that the dACC may function as a type of neural alarm system, with its role in pain unpleasantness being primary (as a role in responding to threatening situations) and with a role in discrepancy detection/conflict monitoring being more recently developed evolutionarily. Further research with various paradigms is necessary to delineate pathways that are active during different emotional states. However, enough evidence has accumulated to reveal the potential impact of opiate drugs on the brain pathways subserving social attachment that it is not surprising that the addiction potential of such drugs is so strong. The social attachment system is important for humans and other primates.

Social Attachment in Primates

A new theory has been proposed about the importance of opioid transmission in social attachment in primates, including humans. Machin and Dunbar (2011) review comparative work on primate social grooming and mother–infant behavior. Studies with naloxone discovered that it increased grooming behavior in monkeys (Fabre-Nys, Meller, & Keverne, 1982; Meller, Keverne, & Herbert, 1980). Naloxone also led to an increase in distress calls in nonhuman primate infants (Kalin, Shelton, & Barksdale, 1988; Martel, Nevison, Simpson, & Keverne, 1995). These findings led Dunbar (2010) to theorize that social touch stimulates opioid release for anthropoid primates, including humans. Social touch has been shown to increase serum beta-endorphin in dogs—which are highly social—and people (Odendaal & Meintjes, 2003). Dunbar (2010) suggests that touch and group activities such as music, dancing, storytelling, and laughter may increase social bonding via endorphin release. Dunbar et al. (2016) refer to social activities in large groups as “grooming at a distance” (p. 10). The

critical importance of social bonds to human survival (and reproduction) creates vulnerability to addiction when drugs mimic the natural neurotransmitters involved in brain pathways.

Could Humans Evolve a Brain that Is Less Prone to Opiate Addiction?

It is clear that the physical and social pain systems are essential for survival, and thus could never be eliminated by natural selection against addiction. The survival value of the physical pain system is obvious, but it is also clear that evolution could not act to remove our brain pathways related to reward and social attachment, the pathways that are hijacked by addictive substances. O'Connell and Hoffman (O'Connell & Hofmann, 2011a, 2011b) describe two ancient brain circuits present in vertebrates, the mesolimbic reward system and the social behavior network. These two circuits form part of a large social-decision-making network that is homologous among vertebrates (mammals, birds, reptiles, amphibians, and teleost fish; O'Connell & Hoffman, O'Connell & Hofmann, 2011b). These circuits subserve naturally rewarding behaviors such as sexual activity (O'Connell & Hofmann, 2011a). MacLean (1985) suggested that the evolution of vocalizations that maintain contact between mothers and offspring may be a key development in the evolution of mammals and that this capability may depend on pathways connecting the thalamus and the cingulate cortex.

Anthropoid primates, including humans, have extended long-term bonds beyond monogamous mating situations (Schultz & Dunbar, 2007). Social attachment has a unique role in human evolution in that social groups include nonrelatives with whom long-term cooperative reciprocal relationships are maintained (Machin & Dunbar, 2011). Because of this unique social system, Machin and Dunbar (2011) asserted that the "opioid system may play a more central role in sociality in primates (including humans) than in other mammalian taxa" (p. 985). They suggest that endogenous opioids are involved in maintaining stable long-term relationships (while relationship onset may be subserved by dopamine, serotonin, oxytocin, and vasopressin). Thus, our human complex social bonds are "emancipated" from hormonal control and are rather supported by higher cognitive abilities and may be maintained by brain opioid systems (reviewed by Machin & Dunbar, 2011). As stated by Machin and Dunbar (2011),

> … the evidence seems to suggest that while non-primate mammals may utilize the endorphin system to maintain infant/mother and sexual pair bonds, primates (and hence humans) may rely to a much greater extent on this system to maintain the complex, diverse and enduring social networks that are uniquely characteristic of this order (p. 1014).

The human brain has an evolved vulnerability to addiction. The dopaminergic reward system and the social and physical pain pathways are essential for survival. Because these pathways operate using neurotransmitters, vulnerability to the

effects of external chemicals is inherent. These susceptibilities are shared by all human beings. However, research has shown individual differences in risk for addiction. Some of the most important individual differences in risk to addiction will be reviewed.

Individual Differences in Vulnerability to Addiction

An evolutionary approach typically focuses on characteristics that are common to human beings as a species, *Homo sapiens*. However, an evolutionary perspective can also help illuminate some differences among humans. For example, demographic differences in patterns of alcohol problems follow similar gender and age patterns as do many risky behaviors (reviewed by Hill & Chow, 2002). Alcohol problems are most common for young men, whose rates of alcohol disorders are 3–4 times as high as for women. Onset of alcohol problems peaks during the ages 15–29. Hill and Chow (2002) examined risky drinking patterns using life-history theory, which is a subset of evolutionary theory. Life-history theory explains higher risk-taking propensity of young males as based in more intense competition for mates, where success in competition requires taking risks (Wilson & Daly, 1985).

Individual differences in risk-taking would have many sources. The risky behavior involved in alcohol intoxication or use of illegal substances is complex, and the motivational basis is multidimensional. Various areas of addiction research have used psychological concepts such as impulsivity and future orientation. Impulsivity has been separated statistically into two main components, cognitive and behavioral impulsivity (White et al., 1994).

Future discounting would correlate with risk-taking because of the way present versus future benefits and costs of a risky act are evaluated. When the future is devalued, both benefits and costs are given lesser weight when delayed, in an unconscious calculation underlying behavior. The present benefits and costs have more weight in a decision. This construct of future discounting fits into cognitive impulsivity or decision-making impulsivity noted above. Life history acceleration has been used to help explain health-related risk-taking versus health-promoting behaviors, a relationship that persists even after accounting for demographic factors (Kruger & Kruger, 2016).

Future discounting has been measured using monetary choices after delays. According to this assessment, steeper discounting of the future has been found among people with a history of addiction (e.g., Kirby, Petry, & Bickel, 1999; Vuchinich & Simpson, 1998). A lucid description of the methods and results of monetary discounting measurement is given in Kirby et al. (1999). Recently, researchers have proposed that delayed-reward discounting is a central feature of addiction (Bickel & Johnson, 2003; MacKillop et al., 2011). One prominent symptom of addictive behavior is the use of drugs (immediate temporary benefit) despite future costs due to such use and the loss of potential future benefits gained by remaining free of addiction. MacKillop et al. (2011) conducted a meta-analysis of 46 published

studies with the purpose of comparing groups with addictive behavior to those without. They found a medium effect size (d = .58) for this comparison, which indicates that groups with addiction differ from those without addiction by about a half of a standard deviation in delay-discounting.

Specific to the topic of this chapter, Kirby and Petry (2004) compared individuals with addictions who were abstinent to those who were actively using substances. Lower discount rate was shown by a group previously addicted to heroin but currently abstinent, compared to current users. This study included only seven people in the abstinent group, however. This finding was replicated in a recent study wherein individuals in treatment for opiate addiction were tested for delay-discounting at baseline and 12 weeks later at the end of treatment (Landes, Christensen, & Bickel, 2012). The patients' average delay-discounting became less steep over the course of treatment (which was buprenorphine).

A recent study with a sample of patients suffering from chronic pain assessed impulsivity as a risk factor for prescription opioid misuse (Vest, Reynolds, & Tragesser, 2016). They reported that two aspects of impulsivity did correlate with opioid misuse, which were the dimensions of urgency and sensation-seeking (but not lack of premeditation or lack of perseverance). Only urgency was a significant risk factor for future misuse (Vest et al., 2016). More research on urgency is warranted, because many existing studies focused on other aspects of impulsivity.

Individual differences may also occur through geographic variation in human populations. For some substance use disorders, such as Alcohol Use Disorder, differences in vulnerability have been found to exist based on the genetic predisposition of specific ethnic groups. An example of this is found in individuals of East Asian descent, where protective gene variants that affect alcohol metabolism cause flushing in response to alcohol consumption, and thus prevent binge drinking (Edenberg, 2007). Another example is found in specific Native American populations who have an increased prevalence of Alcohol Use Disorder, which is associated with unique gene variants that are also involved in alcohol metabolism (Mulligan et al., 2003). However, ethnicity-based vulnerabilities or protective effects have yet to be demonstrated in regard to Opiate Use Disorder. This is despite the fact that the use of opium-containing substances has been long standing in some parts of the world and relatively new in other parts of the world.

Opium is thought to have been originally used in cultural practices in the Middle East beginning between 3000–2000 B.C. (Brownstein, 1993). In contrast, it was not brought to China and India until around 700 A.D., and not to Europe until around 900–1200 A.D. (Brownstein, 1993). This geographic variation presents the possibility that populations that have used opiate derivatives for a long period of time could have evolved traits that are protective against addiction and/or other complications of opiate use, such as overdose death. An article on global epidemiology (Degenhardt et al., 2014) suggests that there are geographic differences in the consequences related to opioid use. Most startling are the years of life lost due to opioid use disorder. Of those regions that had a rate of years of life lost that was greater than 75 years per 100,000 people, the following had the highest, in order: South Sub-Saharan Africa, North America, Eastern Europe, and Australia (Degenhardt et al., 2014). This is in contrast to predominately Asian regions, where there were still high rates of disability due to

Opioid Use Disorder, but years of life lost per 100,000 people was under 10 (Degenhardt et al., 2014). This pattern generally suggests that regions with large populations of Caucasians had more death due to opioid use. It would be interesting in future work to see if there are different ethnic vulnerabilities to mortality from opioids.

Geographic and ethnic variation in substance use point to potential genetic differences. It is beyond the scope of the current article to review genetic susceptibility to opiate addiction, however. Research is being conducted on a polymorphism in the μ-opioid receptor gene, OPRM1. One allele (G) has been associated with beta-endorphin sensitivity (Bond et al., 1998). Individuals with this allele showed high levels of rejection sensitivity in a laboratory social exclusion experiment, with greater dACC and AI activation (Way, Taylor, & Eisenberger, 2009). Recent studies indicate possible associations of opioid addiction with OPRMI and also markers related to the δ-opioid receptor (OPRD1), galanin (GAL), and one related to ATP (ABCB1) (reviewed in Beer et al., 2013). Nevertheless, there is as yet no consensus on various candidate genes in vulnerability to addiction, due to inconsistent results between studies (Beer et al., 2013).

Based upon our previous discussion of the importance to survival of the brain pathways relevant to opiate addiction, human vulnerability to addiction cannot be eliminated by evolution nor by human invention. The impact of substance use on sufferers and society could be reduced. Efforts to stem the flood of prescription opioids were mentioned earlier. Effective approaches to treating addiction have been developed. Current treatment approaches combine medication with psychotherapy. It appears that successful treatment is associated with changes in future discounting, described above as a risk factor and correlate of substance use. A brief review of current treatment approaches will be given next.

Current Treatment Approaches for Opioid Use Disorder

Although human vulnerability to addiction cannot be eliminated, its impact could be reduced. Current treatment approaches combine medication with psychotherapy. Medication-Assisted Treatment replacement therapies are demonstrated as more efficacious than psychotherapy alone when treating OUD (e.g., Mattick, Breen, Kimber, & Davoli, 2009; Mattick et al., 2013).

OUD treatment options include methods of *harm reduction* (Seiger, 2014), *tapered withdrawal*, and *medication-assisted treatment* (MAT; CSAT, 2005). Optimal treatment options are established based upon a thorough biopsychosocial assessment of multiple patient-specific factors: (a) one's readiness for change (DiClemente, 2003); (b) medical issues (e.g., HIV status, hepatitis A, B, or C, and liver cirrhosis); (c) other substance use disorders; (d) psychological factors (e.g., co-occurring and/or substance induced disorders) characteristically depressive, anxiety, and personality disorders; (e) vocational status (i.e., employment history and educational status); (e) legal status (e.g., incarcerated, probation, or parole); and (f) status of family (e.g., child custody) and other significant relationships (e.g., living with others with OUD; CSAT, 2005).

Harm Reduction

Harm reduction (HR) is a treatment approach that recognizes a number of individuals with OUD have no desire to stop, are ambivalent, or view themselves as incapable of discontinuing use (DiClemente, 2003). HR approaches seek to minimize harm to the opioid user and the community at large. Examples of HR include needle exchange programs, healthcare outreach, education about cleaning used needles with bleach, strategies to avoid a drug overdose, and access and knowledge about naloxone (Narcan) and its ability to reverse acute opioid overdose (CSAT, 2005). The philosophy is to meet patients where "they're at" with the long-term goal of providing treatment when patients are ready to change their drug-using behavior (Seiger, 2014).

Medically Supervised Withdrawal

Those not eligible for MAT (e.g., OUD < 1 year; a minor i.e., <17 years old) or who prefer remaining medication free can opt for medically assisted withdrawal, which detoxifies the patient gradually with a process referred to as "tapering" (CSAT, 2005). Tapering is performed with either methadone (an opioid agonist) or buprenorphine (an opioid partial-agonist) short-term (i.e., ≤30 days) or long-term (i.e., ≤180 days). Federal regulations stipulate that two unsuccessful detoxification attempts in 1 year necessitate consideration of an alternative treatment option (CSAT, 2005).

Tapering

Tapering is a process whereby a gradual reduction in the dosage of methadone. Patients are informed about the salience and effects of individual differences and other variables in the tapering process, such as body weight, drug absorption, and individual metabolism and acquired opioid tolerance, which can affect their course of treatment (CSAT, 2005). Methadone doses are typically reduced in 5–10% increments every 1–2 weeks. Patients tapering from methadone typically experience reduction of opioid withdrawal symptoms; however, the final stages of tapering require an inevitable experience of discomfort. This may increase patient risk for opioid relapse and/or use of other drugs such as benzodiazepines, alcohol, and cocaine to ameliorate their remaining symptoms. Given that 80% of tapered patients return to opioid abuse, it is imperative that patients develop robust relapse prevention skills in addition to extensive sober and social supports (CSAT, 2005).

Outpatient Treatment Programs (OTPs)

OTPs provide a range of treatment options for a diverse range of individuals with OUD onsite and in collaboration with other community agencies. Patients deemed appropriate for OTPs should meet Federal and State requirements for opioid treatment and in addition, have previously failed a medically supervised withdrawal; or participate in a residential treatment setting; or require a long-term MAT stabilization. Hospital-based OTPs typically provide access to both medical and psychosocial services, which increase the prospects of patient compliance and successful treatment outcome (CSAT, 2005).

Residential Programs

MAT residential programs are recommended for patients who can benefit from structured stabilization and robust sober supports that address both their physical and psychological well-being (CSAT, 2005).

Community Self-Help

Self-help fellowships otherwise known as 12-step programs provide community-wide support for individuals with OUD. *Narcotics Anonymous* (NA) was fashioned after *Alcoholics Anonymous* in the late 1940s (NA, 2016) and supported individuals with alcohol and other drug problems. However, *Bulletin 29#* (NA, 2016) published in 1996 established that individuals on replacement therapy (i.e., MAT) were welcome to attend NA meetings but not to actively participate. *Methadone Anonymous* is a reported alternative that supports individuals in attempting or in sustained remission for OUD through MAT or maintenance (CSAT, 2005).

Therapeutic Communities

Historically, therapeutic communities (TC) such as *Synanon* in California were long-term residential treatment centers facilitated by staff typically in recovery from OUD themselves. The aim of TCs is to return individuals to successful drug free lifestyles that lead either to an effective return to the outside community or a transition within the Synanon community (Friedman, 2014).

Medication-Assisted Treatment (MAT)

OUD treatment is a dynamic, intentional, time-dependent process with characteristic fits and starts punctuated by substantial levels of patient ambivalence. This is the norm. It is often difficult for outsiders (i.e., those non-addicted) to appreciate, especially when a patient's life is in total shambles, that there is any reluctance at all in escaping the grip of OUD. Although an in-depth discussion of recovery is beyond the scope of this chapter, we provide a brief overview of the phases of Medication-Assisted Treatment (MAT) to give the reader a sense of the complexity of both the physiological and psychological aspects of OUD experienced by individuals in search of OUD recovery. Federal and state regulations must be followed in the United States to offer MAT, which inopportunely limit the number of opioid treatment programs (OTP) and qualified physicians available to prescribe methadone and buprenorphine for medication replacement purposes (see CSAT, 2005).

Stages of Comprehensive Medication-Assisted Treatment (MAT)

Successful MAT (i.e., with methadone or buprenorphine) is blended with evidence-based therapies, such as cognitive behavioral therapy (Beck, Wright, Newman, & Liese, 1993), motivational interviewing (Miller & Rollnick, 2002), and the trans-theoretical model (i.e., stages of change; DiClemente, 2003) in combination with social supports (e.g., community self-help, family, church), which afford individuals the optimal success in cessation of OUD.

Clinicians recognize six phases of MAT—acute, rehabilitative, supportive-care, medical maintenance, tapering and readjustment, and continuing care (CSAT, 2005). The acute phase can range from several days to months, and entails the cessation of all opioid use, including any other drugs abused by the patient. Therapeutic maintenance medication (i.e., methadone or buprenorphine) is initiated to suppress patient symptoms of the withdrawal syndrome and incessant drug craving.

In the rehabilitative phase referrals are made to appropriate supportive services determined at assessment to enhance and address unsuccessful functioning in patients' other major life areas (e.g., co-occurring disorders, activities of daily living, medical, social, employment, legal, family). This phase is essential to reducing patient anxiety, depression, and other salient impediments that allow establishing a firm footing in early recovery. Furthermore, the rehabilitative phase advances opportunities to engage patients in community-based recovery (e.g., 12-step programs) and faith-based organizations. In conjunction with formal treatment, these organizations can provide social support, teach patients to identify personal high-risk situations and emotional states, and develop a manner of daily living that promotes substance free living (CSAT, 2005).

Supportive care and medical maintenance phases involve progressively longer periods where patients may take medication at home rather than at a clinical site, progressing to a 30-day supply of medication. Progression in treatment is verified with routine drug tests (CSAT, 2005). The tapering and readjustment phase involves reducing one's medication dosage over time with the goal of forgoing medication altogether. Tapering and continued medication maintenance are both considered appropriate treatment goals; however, the risk for relapse increases significantly for those who cease medication (CSAT, 2005). The continuing care phase follows a patient's successful tapering and readjustment to living medication free. Ongoing treatment is highly recommended owing to the chronicity of OUD and perpetual risk of relapse for some patients.

Given the evidence that physical and emotional pain share brain substrates, it is logical to assume that successful OUD treatment necessarily consists of medication, psychological, and social supportive interventions. Unfortunately, a disproportionate focus is placed on medication to the exclusion of other psychological and social services. Emotional stress is a well-known and documented relapse trigger in nearly all SUDs, and research on opioids and social pain suggests that attention to treatment of emotional cues be as important, if not more important, especially for those whose treatment goal is eventual tapering to a non-medicated recovery state.

Conclusion

Panksepp et al. (2002) noted that social interactions and drug addictions utilize common brain pathways. They suggested that drugs trick animals into associating stimulation of the social brain pathways with drug stimuli. Understanding the social pain aspect can help us understand why opiates are so addicting. Reducing social pain as well as physical pain feels better than pain patients might expect. Treatment programs need to replace the reinforcing good feelings given by the drug, mimicking social connection. The stereotypical idea is that opiates are for physical pain, but we now know that much of the power of opiate addiction is about emotional pain.

Only recently have researchers started to understand the common basis of social and physical pain as a risk factor for drug addiction (LeBlanc, McGinn, Itoga, & Edwards, 2015). LeBlanc et al. (2015) proposed that pain pathway sensitization by stress may create greater risk for addiction through increasing pain unpleasantness (i.e., the affective dimension of pain). In one study of former opioid-dependent individuals who had attained abstinence, variation in sensitivity to physical pain correlated with the level of craving they reported to opioid cues (Ren, Shi, Epstein, & Wang, 2009). Pain-induced distress was the critical factor, not simply level of pain (Ren et al., 2009). Edwards et al. (2011) found that individual differences in pain sensitivity predicted opioid misuse among chronic pain patients. LeBlanc et al. (2015) reviewed preclinical animal studies showing pain-induced sensitization in the ACC and the central amygdala, which they speculate might underlie addiction liability to pain relief medication. More research is needed on affective dimensions of pain and pain relief.

Treatment programs need to focus more on ameliorating social pain during recovery from opiate addiction. Social attachment is a human universal. Heilig, Epstein, Nader, and Shaham (2016) acknowledge that neuroscience research on addiction has had insufficient impact on clinical treatment. Social integration has rarely been included as a measured or manipulated variable in animal studies of addiction neurobiology. Social stressors that trigger relapse for people are difficult to model in laboratory settings, such as family conflicts or social ostracism (Heilig et al., 2016). Heilig et al. (2016) discuss the role of social exclusion in addiction and recovery, "Improving the social integration of drug users through opportunities for housing, jobs and meaningful relationships is therefore not merely a nonspecific intervention but rather a neurobiologically specific and critically important way to decrease drug use" (p. 4).

This review has mentioned research that indicates risk factors for addiction, and some of these are potential targets of future research. One is future discounting and impulsivity. Another is gender differences. There is evidence from brain imaging that there are gender differences in the opioid system of the brain, from a study with women tested twice at two menstrual phases and with a manipulation of estrogen level by an estrogen patch (Smith et al., 2006). During estrogen administration, mu-receptor binding increased 15–32% in the target brain regions. MRI results correlated with individual subjective ratings of pain (Smith et al., 2006). This finding calls for further research in gender differences in all subjective and physical aspects of pain.

Another potential source of individual differences in vulnerability is trauma. A study of military veterans who had experienced trauma detected changes in opioid receptor binding under PET scanning (Liberson et al., 2007). Those with posttraumatic stress disorder had lower binding specifically in the ACC (Liberson et al., 2007). Lower binding might indicate a lower or depleted ability to process social connection, which might enhance vulnerability to opiates. More research on social losses and vulnerability to addiction is needed.

Variations in sensitivity to loss and activity in the "PANIC" emotional system may be viable indicators of vulnerability to opiate addiction. Eisenberger (2012) describes various individual differences that correlate with increased propensity to feel social pain, all of which might be researchable risk factors for opiate addiction. These included low self-esteem, anxious attachment, and interpersonal sensitivity (reviewed by Eisenberger, 2012).

Panksepp et al. (2002) speculated that, "If adequate social bonds fail to develop, an individual may show an altered future tendency to engage emotional brain systems through other (e.g., pharmacological) means …" (p. 461). It would behoove social scientists to assess patterns in contemporary society that appear to reflect general weakening of social bonds (Putnam, 2001). Kinship and community networks are less tightly enveloping of people than in past decades, leading to fewer routine social gatherings, joint volunteering efforts, regular group meetings, or other activities that automatically reinforce social connection. Putnam (2001) referred to these experiences as building a person's social capital, a storehouse of social resources. Instead, we now spend less time engaged in face-to-face socialization and communication, and more in

long-distance internet communication. Virtual communities on the internet, while attractive to people, do not foster the same sense of community commitment and social support given by an in-person network of relatives and close friends (Song, 2009). If a progressive widespread weakening of social bonds continues, it will present an increasing risk for vulnerability to the psychological effects of opiates.

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Criminology's Modern Synthesis: Remaking the Science of Crime with Darwinian Insight

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A Discipline Adrift

For over a century and half now, the biological sciences have been moored to a unifying set of principles—that life on earth is ancient; that all life is descended from a common ancestor; that the diversity of species on the planet is the product of random genetic mutations and a combination of random genetic drift and nonrandom selection favoring alleles promoting survival and reproduction; and that these processes apply to ***every living organism*** (Buss, 2015; Darwin, 1859; Dennett, 1995; Goetz & Shackelford, 2006; Pinker, 1997; Stearns, 2000; Wright, 1994). Yet, for decades (and decades) in the social sciences, scholars have conducted their work as if humans, for all practical purposes, were exempted from these universal evolutionary processes (Horowitz, Yaworsky, & Kickham, 2014; Maynard, Boutwell, Vaughn, Naeger, & Dell, 2015; Pinker, 2002). At most, these scholars allowed for the fact that our bodies may have been historically sculpted by natural selection, but the instant we invented culture we were freed from the laws of nature and exempted from the pressures of selection forces (Cochran & Harpending, 2009). Consequently,

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the role of biology in some disciplines has come a distant second to the role of culture and socialization in exploring and explaining human nature and human differences (Pinker, 2002; Winegard, Winegard, & Boutwell, 2017).

There are, of course, exceptions to this trend, as a host of important thinkers have stood firm in the theory that evolutionary processes—most importantly evolution by natural selection—provide valuable hypotheses and explanations for the outcomes that social scientists have often inadequately explained (Buss, 2009, 2015; Ellis, 1988; Rushton, 2000). And, slowly, more scholars across fields outside of criminology have abandoned the beliefs that human culture exists beyond the reach of biology and that human behavior is exempt from any moorings to Darwinian insight (Buss, 2015; Wright, 1994). For scholars of crime, however, this insight has been especially delayed, and too often these evolutionary thinkers have been shouted down, belittled, or ignored (Pinker, 2002; Wright et al., 2008). Criminology as a field has been hesitant to incorporate basic biological insights, much less to openly entertain the possibility that antisocial human tendencies might owe their origins to evolution.

Hesitancy toward biological theorizing about human behavior has a variety of sources, yet much of the concern seems to originate from misunderstandings about the most basic of biological concepts and a lack of exposure to the natural sciences, in particular to genetics and the processes of evolution by natural selection (Pinker, 2002; Wright et al., 2008). It is perhaps less surprising, then, that within the relatively brief history of criminology as a discipline, most theories of crime causation (with few exceptions) implicate *social factors* as the chief causes of crime, with little reference to the role that biological forces might play in helping to create a predisposition toward antisocial and violent behavior (Beaver, Barnes, & Boutwell, 2014; Ellis, 1988).

More recently, there has been a refreshing effort to unify criminological knowledge using evolutionary concepts, with a particular focus on life history theory (see also, Ellis, 1988). Boutwell & Barnes, et al. (2015) outlined a perspective in which a host of crime correlates (some of which seem quite disparate, at first blush) might be integrated using insights from evolutionary biology. Below, we briefly outline the basics of life history theory by drawing on examples rooted in decades of research throughout the animal kingdom, and discuss why these same principles are relevant to the study of human behavior. We conclude by discussing some of the ideas proposed by Boutwell and colleagues, and highlight the additional research that is likely to be fruitful in this area. Our hope is to draw attention to evolutionary approaches to understanding human behavior, and encourage others to empirically test the hypothesis that criminal behavior does, in fact, have a biological basis and relevant evolutionary explanations (Del Giudice, 2014; Ellis, 1988). In fact, criminology will *need* to test evolutionary hypotheses to remain a relevant behavioral science in a world in which evolutionary theory provides critical insight into the functioning of all living organisms.

Life History Theory: A Very Brief Primer

As Darwin (1859) noted, the central challenges facing all organisms are to survive and to reproduce. His theory of evolution by natural selection posits (in modern terms) that the genetic variants underlying traits that promote greater reproductive success (because of successful survival and reproduction) will become more common in subsequent generations, and accumulate in a population (Dennett, 1995). However, the best strategies for maximizing reproduction are not straightforward, because organisms must balance the trade-off between allocating their investment in survival (at a cost of reproducing, but such that they can continue to live and reproduce later) and reproduction (at a possible cost to survival; Stearns, 2000). The particular strategy an organism takes to balance this trade-off determines the length and timing of basic "life history" traits: growth, maintenance, reproduction, and senescence (i.e., aging and death; Stearns, 2000). In nature, we observe a great deal of variation among organisms in their life history traits (within mammals, imagine the "fast" life history of rabbits versus the "slow" life history of elephants) because the timing of each of these stages is closely related to the specific ecological challenges faced by a given population of organisms (Roff, 1992; Stearns, 1992, 2000).

Life history theory was formulated to explain how natural selection shapes life history strategies, such as the timing and rates of growth, maturation, reproductive investment, and senescence, as well as the age, size, and other traits of an organism at each stage of the life cycle (Stearns, 1992). In recent decades, a rich corpus of research has provided increasing clarity regarding the diversity in the reproductive strategies of organisms—within and across species and populations—by explaining these as adaptations to local environments (Bårdsen, Næss, Tveraa, Langeland, & Fauchald, 2014; Chisholm et al., 1993; Draper & Harpending, 1988; Harvey et al., 2017; Hua, Sieving, Fletcher, & Wright, 2014; Skrzynecka & Radwan, 2016).

MacArthur and Wilson (1967) described a spectrum of variation in life histories. In populations occupying ecological niches in which the environment is unpredictable and the risk of early mortality is high, rapid maturation and early reproduction might be favored by natural selection (Pianka, 1970). In ecological niches marked by stability and fewer external risks of early death, slower maturation and increased parental investment in relatively fewer offspring might constitute the favored strategy (Pianka, 1970). Since these early observations, life history theory has consistently predicted that increased environmental stability and decreased mortality risk will favor the allocation of resources toward growing slowly, reaching a larger size at maturation, and investing more in each individual offspring (Figueredo et al., 2006). To aid in describing this phenomenon, scholars have increasingly relied on the terms "faster" and "slower" in reference to a continuum of life history speeds (Figueredo, Andrzejczak, Jones, Smith-Castro, & Montero, 2011; Jones et al., 2008; Ricklefs & Wikelski, 2002).

In recent decades, researchers in the area have begun directly testing the possibility that variation along this fast-slow continuum, for a variety of organisms including humans, may also play a key role in explaining various developmental,

behavioral, and personality outcomes (Figueredo, Vasquez, Brumbach, & Schneider, 2004; Walker et al., 2006). In the next section, we discuss some of the research as well as the reasons why it is relevant, not only for understanding behavior, but ultimately for understanding violence in both human and nonhuman populations.

Life History Strategy, Personality, and Behavior

As we have already described, life history theory predicts that different life history speeds involve different approaches to dealing with daily challenges. In line with this prediction, correlations of life history traits with personality and physiology are so common in the natural world that the idea of a "pace of life syndrome" has been adopted by many studying this phenomenon (for a recent discussion see Réale et al., 2010). In fact, variation in life history styles has been invoked as a major explanatory factor for the maintenance of personality traits across animal populations in the wild (e.g., Biro & Stamps, 2008; Dingemanse & Wolf, 2010; Réale et al., 2010; Stamps, 2007; Wolf, Van Doorn, Leimar, & Weissing, 2007). Essentially, corresponding variation in personality styles—which guide how a given individual responds to everyday occurrences and challenges—should reflect an organism's expected current versus future reproduction.

Across a wide range of taxa, organisms embodying a slower life history style display personality traits that reduce risky behavior, such as increased shyness, being less exploratory, and showing lower activity levels, whereas faster life histories are associated with bolder, more aggressive, and more active/exploratory personalities (Biro & Stamps, 2008; Dingemanse & Wolf, 2010). At a proximate level, for instance, food intake and growth demonstrate a positive association with increased activity (e.g., damselflies, fishing spiders, domestic turkeys, rainbow trout, cattle, house mice, common lizards) as well as a negative association with fecundity and longevity (e.g., moths, damselflies, rainbow trout, house mouse; Biro & Stamps, 2008). Aggression and boldness also positively correlate with life history traits including fecundity, growth, and size at maturity (Biro & Stamps, 2008).

Humans are no exception to these patterns. At least some evidence suggests that the General Factor of Personality (GFP; a higher-order factor that the Big 5 personality traits load onto) correlates with a unified measure of life history traits termed the *K*-factor that subsumes variation across life history speed indicators (Figueredo et al., 2004). At the level of the Big 5 personality structure, a slower Life History Strategy (LHS) has been linked to higher Openness, Extraversion, Conscientiousness, and Agreeableness as well as to lower Neuroticism (Figueredo & Sefcek 2005; Figueredo, Vásquez, Brumbach, & Schneider, 2007; Figueredo et al., 2004; Gladden, Figueredo, & Jacobs, 2009). Caution is advisable at this point, however, as recent scholarship has raised methodological and theoretical questions about how well variation in the GFP maps onto variation in life history speed (see Del Giudice, 2012, 2014; Manson, 2017). Yet, it is worth noting that using an alternative measure of personality (the HEXACO dimensions), Manson (2015) linked slower LHS to

higher Extraversion, Agreeableness, Conscientiousness, and Honesty-Humility. Strouts, Brase, and Dillon (2016) also found a positive correlation between slow LHS and the same HEXACO dimensions as Manson, with an additional positive correlation with HEXACO Emotionality.

Life History Strategy, Aggression, Violence, and Crime

The intersection of life history speeds and personality is important because it provides insight into various behavioral tendencies. Beginning first with research conducted on nonhuman animals, faster reproductive maturation (first mating, first menses) has been linked to dominance and social status (male rhesus macaques: Bercovitch, 1993; female savannah baboons: Altmann, Hausfater, & Altmann, 1988; male fallow deer: Komers, Pélabon, & Stenström, 1997). Shorter life expectancies and early-life mating success are correlated with aggressiveness (big horn sheep: Réale, Martin, Coltman, Poissant, & Festa-Bianchet, 2009). Individuals with longer lives and lower fecundity (indicators of slower life histories) are more risk-averse (a trait correlated with aggression) than their shorter-lived, higher-fecundity counterparts (Panamanian birds: Ricklefs, 1977; dabbling ducks: Ackerman, Eadie, & Moore, 2006). Higher annual mortality rates and higher reproductive output per breeding attempt have also been linked with riskier nest defense behaviors (ducks: Forbes, Clark, Weatherhead, & Armstrong, 1994).

Low risk-aversion and aggression are often related to a third personality trait, boldness, which is the tendency to take risks and to explore (Wilson & Godin, 2009). Faster growth rates and/or higher food intake have been linked to `nese quail: Yang, Dunnington, & Siegel, 1998; fishes: McCarthy, 2001; Pottinger, 2006; Pottinger & Carrick, 2001; Metcalfe, Huntingford, & Thorpe, 1988; Metcalfe, Taylor, & Thorpe, 1995; Yamamoto, Ueda, & Higashi, 1998, and mithuns: Mondal, Rajkhowa, & Prakash, 2006). Similarly, boldness has been linked to food intake and/or higher growth rate (fish: Øverli, Sørensen, Kiessling, Pottinger, & Gjøen, 2006; Walsh, Munch, Chiba, & Conover, 2006; Ward, Thomas, Hart, & Krause, 2004; cattle: Müller & von Keyserlingk, 2006; Petherick, Holroyd, Doogan, & Venus, 2002; domestic pigs: Geverink, Heetkamp, Schouten, Wiegant, & Schrama, 2004). Boldness is also positively related to fecundity (fishing spiders: Johnson & Sih, 2005; laying hens: Barnett, Hemsworth, & Newman, 1992; Atlantic silverside fish: Walsh et al., 2006; big horn sheep: Réale & Festa-Bianchet, 2003; Réale, Gallant, Leblanc, & Festa-Bianchet, 2000; domestic pigs: Janczak, Pedersen, Rydhmer, & Bakken, 2003).

There is a growing body of work on this topic examining humans, as well, much of which has uncovered evidence that variation in aggression and violence correlates with LHS (Beaver, Wright, & Walsh, 2008; Charles & Egan, 2005; Rushton & Templer, 2009; Rushton & Whitney, 2002), both across populations (e.g., Rushton & Templer, 2009; Rushton & Whitney, 2002; Walker et al., 2006) and across individuals within a population (Charles & Egan, 2005; Figueredo, Vásquez, et al., 2005).

Additionally, the link between LHS and human behavior has been found when examining specific indicators of LHS (Minkov & Beaver, 2016), as well as when using scores on psychometric measures of LHS (Wenner, Figueredo, & Jacobs, 2005), such that higher rates of fast LHS indicators and lower scores on measures of the K-factor correspond with higher rates of violence and aggression.

At the population level, national and state differences in LHS traits predict cross-national and cross-state differences in violent crime. Rushton and Templer (2009) found average life expectancy to be a significant predictor of national differences in serious assault, rape, and murder across 113 nations. Along similar lines, Minkov and Beaver (2016) uncovered evidence that adolescent fertility and parental absenteeism predicted national differences in muggings, assaults, and murders across 51 countries. At the level of the state, Templer and Rushton (2011) found state differences (within the USA) in life expectancy to predict robbery, assault, and murder rates.

Higher mating effort, an indicator of a faster LHS, has been linked to self-reported delinquency (Charles & Egan, 2005; Rowe, Vazsonyi, & Figueredo, 1997) and coercive sexual behavior (Lalumiere, Harris, Quinsey, & Rice, 2005; Lalumiere & Quinsey, 1996). Sexual precocity has also been linked to violence (Copping, Campbell, & Muncer, 2013). In a multi-dataset study, Sherman, Figueredo, and Funder (2013) found faster LHS to be associated with increased expression of hostility and attempts to undermine or sabotage during various interpersonal situations in a laboratory setting. In a meta-analytic study, Figueredo et al. (2014) uncovered a correlation between faster life history strategies and antagonistic social strategies (i.e., pursuing one's own interests to the detriment of the interests of others, indicated by high scores on measures of aggression, prejudice, and psychopathic traits). Less remains known about the association between overt life history speed indicators and psychopathology, yet some work is beginning to emerge on that issue as well (Del Giudice, 2014; Smith-Woolley, Rimfeld, & Plomin, 2017).

The Modern Synthesis in Criminology

Drawing on much of the evidence just discussed, Boutwell,Barnes, et al. (2015) proposed an evolutionary theory of criminal offending grounded in life history theory and intended to unify a large swath of criminological insight. To be sure, decades of criminological research have produced an impressive amount of knowledge concerning individual-level correlates and risk factors for antisocial and violent behavior (Ellis, 1988; Gottfredson & Hirschi, 1990; Moffitt, 1993; Pratt & Cullen, 2000). There are core concepts in the field that seem to emerge, time and again, in empirical research. It has been widely acknowledged for some time, for example, that antisocial, impulsive, and criminal behavior peak shortly after puberty, and then begin declining as individuals enter adulthood (Moffitt, 1993).

In defiance of this age-crime curve, however, a small fraction of individuals seem to begin displaying antisocial propensities very early in life (Moffitt, 1993;

Moffitt, Caspi, Harrington, & Milne, 2002). With age, the severity of their behaviors increases, and when other members of the population are desisting, their propensity for engaging in crime and violence remains elevated (Moffitt et al., 2002). Additionally, any theory of crime would also have to account for the persistent evidence that criminality is partly (though not completely) heritable (Barnes, Beaver, & Boutwell, 2011; Rhee & Waldman, 2002). A complete crime theory would need to accommodate the robust gender (and race/ethnicity) differences in criminal behavior that exist (Boutwell and Barnes et al., 2015; Buss, 2015). Finally, and as we alluded to above, a unified theory would need to explain why crime prone individuals also tend to reach physical maturity relatively rapidly, why they display elevated sexual promiscuity, as well as the host of other correlates outlined by prior scholars (Boutwell, Nedelec, Lewis, Barnes, & Beaver, 2015; Ellis, 1988). In other words, a unified theory of crime would need to unite not only the rather obvious facts about crime (that it is a male driven, age-graded behavior), but also the more disparate correlates of offending (i.e., high sexual promiscuity).

Boutwell & Nedelec, et al. (2015) proposed that such patterns in criminality may represent natural variation within the human species along a spectrum of life history speed. In particular, Boutwell & Barnes, et al. (2015) (see also Ellis, 1988; Rushton, 1985) argued that individuals that display a slower life history strategy reach puberty later, have fewer children, and lead largely prosocial lifestyles. At the other end of the spectrum are individuals who develop a faster life history style. Toward this end of the curve, the predisposition to crime is high; however, it is reasonable to expect that these individuals may never commit a criminal act, per se. They will, however, be more impulsive, aggressive, and short-sighted. They will be less likely to invest in children; however, they will engage in riskier and more frequent sex with a greater number of partners, and produce more children than individuals of slower life history styles (see also Moffitt et al., 2002). Put another way, what we call criminality may reflect natural variation in life history speed for our species.

There are several advantages to a life history approach to criminological theory. First, it parsimoniously unifies crime correlates. Rapid sexual maturity, early onset of sexual activity, high impulsivity, poor health, short lifespan, and aggression, as well as a host of other well-documented risk factors for crime fit squarely under the umbrella of reproductive strategy and life history speed (Ellis, 1988). Second, a life history approach provides a mechanism for understanding when and how environmental risk factors may manifest as criminal behavior. It is true that life history speeds are calibrated (i.e., accelerated or decelerated) by genetic differences in the population (Barbaro, Boutwell, Barnes, & Shackelford, 2017). However, the heritability of life history speed variation is not 1.0; thus, genetic differences do not explain all individual differences in life history speeds.

Environmental factors may exert criminogenic effects via accelerating or slowing down life history speeds. For instance, exposure to dangerous, risky, and deprived environments might accelerate the life history speed of an individual (e.g., prenatal exposure to endocrine disrupting compounds has been associated with early sexual maturation in females; Rasier, Toppari, Parent, & Bourguignon, 2006),

ultimately manifesting in antisocial and criminal behavior. However, to adequately test this hypothesis, it will be necessary to employ research designs capable of distinguishing genetic from environmental influence (Barbaro et al., 2017). Although some supportive research has emerged (Boutwell & Nedelec et al., 2015), much more work is needed, especially independent replication by scholars across academic fields. Moving forward, however, a fruitful research program waits to emerge around testing the relationship between various life history speed indicators and a host of criminogenic and illegal outcomes.

Conclusion

The research we have reviewed is not meant to be exhaustive as it relates to life history theory and the behavior of humans and other animals. Nor do we present a complete overview of Boutwell and Nedelec et al. (2015) and their unified crime theory. Rather, the current chapter is intended to illuminate a particular theme: Evolutionary insights are the guiding principles for the study of life. The only exception seems to be that scholars in certain social science fields remain somewhat hesitant to apply evolutionary principles to human beings (Horowitz et al., 2014). There is no reason to suspect that human beings operate outside of the effects of natural selection. Very much to the contrary, there is evidence that ecological factors such as the incidence of disease, high altitude, severe cold, and the advent of agriculture have led to recent evolution in human beings, altering gene prevalence based on local ecological pressures (see Aidoo et al., 2002; Winegard et al., 2017). Thus, efforts to theorize about human behavior should be tethered to Darwinian insights. The "modern synthesis of criminology" proposed by Boutwell and Nedelec et al. (2015), and discussed briefly herein, is an effort to unify criminological knowledge under the umbrella of life history theory.

It cannot be emphasized enough that this synthesis is truly a blend of sociological and biological theory. It does not seek to discard all previous theory, but instead it seeks to broaden our current, typically proximal, explanations by highlighting ultimate causal factors. For example, rather than disregarding the criminological construct of self-control (Gottfredson & Hirschi, 1990), the modern synthesis suggests that individual differences in something like self-control might be better explained as a combination of evolved biological characteristics and environmental factors, rather than simply a product of socialization forces. It is doubtless true that more work needs to be done, and that other biological perspectives might provide a better unifying framework for criminology than life history theory (see Boutwell & Nedelec et al., 2015 for some discussion). Nevertheless, what is clear is that criminology needs evolution to advance intellectually. Ignoring biology will relegate us to obsolescence in the pantheon of natural science.

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Excruciating Mental States

Sarah A. Perry

Something has been missing from the study of suicide and mental illness. It is a phenomenon of major import, but it barely has a name (Meerwijk & Weiss, 2011): the subjective experience of extreme mental suffering. Properly understood, excruciating mental states explain the apparent relationship between mental illness and suicide. But suicide is the tip of the iceberg: excruciating mental states are themselves an enormous problem that current medical and mental health models do not address. Easing the suffering of excruciating mental states would be a moral accomplishment comparable to curing cancer—and the tools to do so already exist.

The Public Health Model and Mental Health Model

The Public Health Model asserts that suicide is a phenomenon that can be ameliorated by government surveillance and intervention. Typical interventions include means restriction (such as drug prohibition and gun restrictions) and public spending on campaigns to raise awareness about suicide and reduce the stigma of mental illness, in hopes that more people will seek treatment. The Public Health Model takes as a given the Mental Health Model of suicide: that suicide is caused by specific mental disorders, and that increasing the prevalence of treatment of the causal disorders will prevent suicides. Mann (2002, p. 308) restates the main claims of the Mental Health Model:

> Suicide is a complication of psychiatric disorders. The probability of suicidal behavior also depends on a diathesis that includes hopelessness and increased life-time impulsivity that may be related to a specific impairment of serotonergic input into the ventral prefrontal cortex. The management of suicidal behavior involves an assessment of risk, the treatment

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of the primary associated psychiatric disorder, and the reduction of access to highly lethal methods for committing suicide, such as guns.

Psychiatric disorders are taken to have a necessary or near-necessary (but not sufficient) role in causing suicide. A biochemical and/or neurological mechanism is presented; technical language hints that all will soon be known. Three interventions are suggested: surveillance, the treatment of primary psychiatric disorders presumed to cause suicidality, and means restriction.

Taken together, the Public Health Model and the Mental Health Model form the dominant model in governmental and medical approaches to suicide. This model is ubiquitous in government communications about suicide. "Suicide is a serious public health problem" is a string that occurs in that exact form in hundreds of journal articles. If suicide is mentioned in a news story, it is rare that the connection between suicide and mental illness is not also mentioned. This model has been promoted in government-funded "suicide awareness" campaigns since the turn of the 21st century, and people are hearing the message: more people endorse the beliefs promoted by the Mental Health Model (Deacon, 2013). Unfortunately, anti-stigma campaigns did not reduce the stigma of mental illness, and may have worsened it (Deacon, 2013).

The Public Health Model had great success over the 20th century in reducing all-cause mortality, infectious disease, and motor vehicle fatalities. In contrast, suicide rates have remained stubbornly stable, both in the United States and globally. Despite "alarming rise" being an extremely popular phrase in research articles and news stories about suicide, the overall picture of suicide rates, as long as they have been measured, is remarkable stability (Liu, 2009). Individual countries, local areas, and subpopulations experience fluctuation in suicide rates, and this fluctuation is occasionally dramatic, but over 90% of the variation in suicide rates over time is between-country, rather than within-country (Liu, 2009). Of course, it is possible that suicide rates would have risen dramatically without public health intervention. But the Public Health Model has not seen an easy victory with suicide. (The ever-rising prevalence of obesity is another embarrassment for the Public Health Model.)

Many have criticized the Mental Health Model in some form. Hjelmeland, Dieserud, Dyregrov, Knizek, and Leenaars (2012) highlight the problems with the psychological autopsy method used to establish the link between suicide and mental illness. In this study design, researchers match known suicides with controls, then interview their family members or other associates in order to posthumously diagnose or rule out mental disorders, often years after the death. The authors conclude that psychological autopsies "cannot constitute a valid evidence base for a strong relationship between mental disorders and suicide" (p. 605). Milner, Sveticic, and De Leo (2013) note that psychological autopsy studies performed in non-Western countries typically find rates of mental illness in suicide much lower than that found in the West. But whether or not there is a strong relationship between mental disorders and suicide, the Mental Health Model obscures the role of subjective suffering.

I argue that the dominant model excludes from consideration a most relevant phenomenon: extreme, prolonged mental agony. "Excruciating mental states" are the common factor among supposedly suicidogenic mental illnesses. It is this common factor, and not mental illnesses as syndromes, that is the necessary (but not sufficient) cause of suicide.

In fact, it would be surprising if psychological autopsy studies did *not* find an elevated rate of people who met the criteria for mental illnesses among suicides. This is because excruciating mental states are connected to mental disorders in three ways: mental disorders cause excruciating mental states; many mental disorders are themselves descriptions of excruciating mental states; and some mental disorders describe responses to excruciating mental states. The Mental Health Model fails to discriminate this factor common to mental disorder disease constructs and suicide.

The Excruciating Mental States Model

I provide an evolutionary account of excruciating mental states and argue that excruciating mental states themselves constitute an enormous problem, even aside from their connection to suicide. These mental states, much like chronic pain, impose an unnecessary burden of suffering; but unlike physical pain, the burden of excruciating mental states is one for which the Public Health Model offers neither acknowledgment nor help. The vast majority of people suffering a prolonged excruciating mental state do not commit suicide. I argue that they deserve *help* in the form of effective relief from this mental state; that effective tools are available to help them; that withholding these tools is not justified; and that the present public health and mental health system is not equipped to provide the kind of help that they need. If excruciating mental states, as I describe them, are a necessary causal element in suicide, then effectively treating these mental states could reduce suicide rates. But the more important reason to focus on extreme mental suffering is because, like excruciating physical pain, it is horrible in and of itself. It is also likely treatable in and of itself.

Direction and Magnitude of Allowable Error in the Evolution of Pain

Mental states are targets of selection, but selection's aim is not always precise. Hunger and thirst are mental states (or qualia) used as signals to trigger a specific behavioral response. Minor thirst or brief hunger do not constitute suffering; a gentle nudge is usually enough to get the proper response. After days of thirst or weeks of starvation, however, thirst and hunger rise to the level of *excruciating*. Specifically,

it is extreme in intensity, prolonged in duration, and no available behavioral response can alleviate it.

Adequate food ensures that a specific behavioral response to hunger is possible. Under those conditions, signals like hunger and thirst must be tuned within precise limits. Hunger (and satiation) are signals used to regulate the timing, amount, and type of food consumed. In most people (at least for most of human history), the signal is effective. It can go wrong in two ways: false positives and false negatives. In severe cases of polyphagia, the hunger signal never turns off, no matter how much food is consumed. Similarly, in polydipsia—which is often associated with schizophrenia—sufferers are always thirsty, and frequently present with water intoxication (de Leon, Verghese, Tracy, Josiassen, & Simpson, 1994). These are false positives. The absence of appropriate hunger and thirst sensations over a long period of time make up the false negatives. Either error is potentially lethal. In the case of the false positive errors, the danger lies in repeatedly performing the behavior indicated by the signal: feel thirsty, drink water, feel thirsty, drink even more water, die of water intoxication. Not all signals are associated with a clearly indicated behavioral response, however.

Now consider physical pain. Physical pain encompasses a wide variety of possible sensations from a wide variety of causes. Some physical pain is limited in duration and has a clear behavioral response (e.g., the pain of sudden skin contact with a flame, or the prick of stepping on something sharp). Most pain, however, has no particular behavioral remedy. The pain of migraine has no signal value. Pain can persist years or decades after a traumatic injury with no behavioral remedy. Neuropathic pain is especially useless.

The absence of the capacity to feel pain (false negatives) is dangerous and lethal, contributing to mortality in congenital analgesia as well as acquired analgesia, as in leprosy. Experiencing a great deal more pain than is necessary, however, seems to be the normal state of human (and probably all animal) life. False positives for pain, since they have no clear behavioral remedy that might alter an equilibrium, do not seem to detract from fitness much. The poor Similaun Iceman (Kean, Tocchio, Kean, & Rainsford, 2013) frozen in the ice for 5,000 years had healed fractures, arthritis, and spinal damage, and must have lived with extreme pain. His tattoos, arranged near his painful injuries, may have been attempts to treat this pain. Opiates have probably been in use for almost as long (Guerra-Doce, 2015).

It is useful to distinguish between two types of "errors" that we might imagine to be possible. First, if the pain system itself breaks down and signals pain when no stimulus is present, as in neuropathic pain, then this is an *empirical* error. Second, if the stimulus detection apparatus is working perfectly as designed by the process of selection, but causes the organism to suffer beyond reason, then this is a *moral* error. For instance, injury (in humans and other animals) often results in *sensitization*, causing the organism to experience innocuous stimuli as painful, and noxious stimuli as extremely painful. It may be the case that this pain response pattern evolved to protect injured animals against future predation (Crook, Dickson, Hanlon, & Walters, 2014), but people experiencing chronic pain have their own interests distinct from the pseudo-interests of selection. Since a great deal of pain

is useless (a "false positive"), relieving pain improves quality of life. From over-the-counter analgesics to opiates to anesthesia, the relief of pain *in and of itself* is a major priority for medicine.

Pain may play a role in suicide. Gray et al. (2014), for instance, found that 45% of Utah suicides in 1 year were experiencing acute pain, compared to 20% of accidental and undetermined deaths. Severe pain was also found to predict suicide in veterans (Ilgen et al., 2010). However, most people in extreme pain do not commit suicide. Only when the *excruciating mental state* of extreme pain is combined with other factors—such as hopelessness, a decreased perceived value of life, and available means—does suicide occur.

Finally, *mental pain* is often useful as a signal. The normal social emotions of longing, loneliness, jealousy, and grief are signals that help individuals form and maintain pair bonds and other social bonds (see, e.g., Fletcher, Simpson, Campbell, & Overall, 2015). Shame and guilt help individuals conform their behavior to social norms so that they may succeed within a group (see, e.g., Bowles & Gintis, 2005). As with all the examples above, however, mental pain can have false positives and false negatives. False positives are especially relevant here: as with physical pain, there is apparently little fitness penalty for mental pain signals that are excessive to signaling needs in duration and intensity. A physical injury may turn into a lifelong painful condition in which the pain is of no signaling value to the sufferer. Similarly, the loss of a job or a romantic breakup can sometimes result in a long period of excruciating mental suffering out of proportion to any behavior-conforming value. Judging from the prevalence of this kind of "false positive" mental pain, there is apparently little fitness cost involved in suffering needlessly. Like those experiencing severe pain, most people who suffer in this way do not commit suicide. Nature has had no reason to spare us suffering.

This category of suffering—"false positive" instances of prolonged excruciating mental states—has substantial overlap with the common mental disorders to which a causal role in suicide is attributed. Some mental disorders, such as schizophrenia, describe underlying etiologies that themselves *cause* excruciating mental states. Other disorders are, in part, *descriptions* of what excruciating mental states look like from the outside (e.g., depression). Still others describe *responses* to excruciating mental states (e.g., substance abuse disorders).

From this perspective, bipolar disorder appears to represent a cycling between "false positive" and "false negative" errors. However, the picture is more complex. When people with bipolar disorder were asked about their manic episodes, most reported experiencing negative emotions during these periods, including anxiety, irritability, depression, and even suicidal ideation (Henry et al., 2003). True "pathological euphoric states" are rare. Most of the errors of the emotional signaling apparatus appear to be in the negative direction.

Predictions of the Excruciating Mental States Model

If the Excruciating Mental States Model predicts suicide better than the Mental Health Model, we would expect that, within mental disorders, those symptoms associated with excruciating mental states would predict suicide more than other symptoms. Although the investigation of emotional pain as a cause of suicide has not been the subject of a major research program, there is some evidence that it is the case. The hallucination symptom in schizophrenia (not associated with extreme suffering) predicted *less* risk of suicide within schizophrenia, whereas symptoms associated with suffering, such as depression and fear of mental disintegration, did predict suicide (Hawton, Sutton, Haw, Sinclair, & Deeks, 2005). Within depression, weight loss, guilt, insomnia, and feelings of worthlessness predicted suicide, whereas fatigue, difficulty concentrating, and indecisiveness predicted less risk of suicide (McGirr et al., 2007). Second, according to the Excruciating Mental States model, extreme mental suffering should itself predict suicide. Hendin, Maltsberger, Haas, Szanto, and Rabinowicz (2004) found that the experience of intense affective states, such as desperation and anguish, distinguished suicides from severely depressed non-suicide controls being treated by the same therapists. Hendin et al. reviewed therapists' notes of suicide decedents and controls, rather than relying on later interviews, as in the psychological autopsy method. "Intense negative affects" is likely a near-synonym of the construct here called excruciating mental states. This suggests that excruciating mental states predict suicide within the mental disorder construct of depression.

Verrocchio et al. (2016) reviewed 42 studies published since 1995 on the connection between mental pain and suicide, and concluded that there is evidence that mental pain itself predicts suicide and suicidal ideation in both clinical and non-clinical populations, even in the absence of a diagnosed mental disorder. De Leon, Baca-Garcia, and Blasco-Fontecilla (2015) have gone so far as to suggest abandoning the "serotonin model" of suicide in favor of the mental pain model.

The practice of treating mental pain itself (rather than an underlying mental disorder) has had relatively little attention. Shattell (2009) argues that mental pain should be treated much like physical pain. In a small trial, Yovell et al. (2015) treated patients with severe suicidal ideation (likely an indicator of excruciating mental states) who were not experiencing physical pain (Yovell & Bar, 2016) with ultra-low-dose opioids. The suicidal ideation of treated patients did decrease, compared to the placebo group, but more evidence is needed. The Excruciating States Model would predict that not only would treating excruciating mental pain reduce suicidal ideation and behavior, but also improve outcomes for those experiencing mental suffering.

Policy Implications

People experiencing excruciating mental states unrelated to physical pain have no medical recourse except within the Mental Health Model. If a person in an excruciating mental state presents to the emergency room, he will not be given immediate relief. He may be labeled a "drug seeker" or, at best, diagnosed with a mental illness or substance abuse disorder and given various forms of treatment for these conditions. The most common form of treatments are medications referred to as selective serotonin reuptake inhibitors (SSRIs) that typically take weeks to have any effect, and in the majority of cases, have little to no effect (Little, 2009). His treatment will not include relief from the excruciating mental state as such. Opiates, for example, are not given for excruciating mental states unrelated to physical pain; under the current system, a person employing opiates for this purpose is a substance *abuser*.

The Excruciating Mental States Model centers on the experience of extreme, prolonged mental suffering. Relief from this state is just as important whether it is physical or mental. Misery is not a medical condition, but some of the tools of medicine might be useful in treating it. However, nonmedical approaches may be more accessible, humane, and effective than services within the present medical system.

Most drugs that are effective at relieving excruciating mental states are illegal to purchase and use. Some drugs are banned outright (e.g., MDMA, cocaine, heroin, psychedelic drugs). Other drugs are only available through doctors, who act as gatekeepers in compliance with specific rules; a doctor has no authority, for instance, to prescribe ketamine or GHB for an excruciating mental state.

An institutional response within the Excruciating States Model paradigm would include a research program exploring the hundreds of substances and practices that are effective at relieving excruciating mental states, in order to identify those that can be used safely. The more safe, effective possibilities available to those experiencing excruciating mental states, the less they will have to suffer, and the less they will employ unsafe and ineffective means to relieve their anguish. Delivery methods outside of the present medical system should be explored in addition to medical options.

People in excruciating mental states cope as best they can. Alcohol remains legal in the United States, and is very popular as an acute treatment for misery. Marijuana is widely available, and some states are easing their prohibitions. Obtaining illegal drugs on the black market is an option that many choose, if they have the knowledge or social connections. Some people experiencing excruciating mental states engage in physical self-harm to cope, such as by cutting their skin (Chapman, Gratz, & Brown, 2006). This is not so far distant from the pain management strategies apparently used on the Similaun Iceman. Moderate or even severe physical pain is often preferable to an excruciating mental state, and can provide some relief.

People in excruciating mental states will attempt to use whatever means they have for relieving these states. Worse, many will simply give up, suffering helplessly. A small proportion will commit suicide. Providing more, better, and safer options allows suffering people to dispense with harmful, ineffective options. They

cannot simply be bullied into doing whatever the current public health policy prescribes.

The primary objection to providing effective remedies for excruciating mental states in and of themselves is the possibility of addiction. But the vast majority of people who use illegal drugs, even heroin and crack, do not become addicted (RTI International, 2008). The same is true of prescription opioids (Vowles et al., 2015). Small studies have recently been conducted using opioids in treatment-resistant depression, with results indicating effective relief and no withdrawal symptoms after treatment (e.g., Fava et al., 2016). And even if addiction occurs, this may be preferable to the sufferer compared with a prolonged excruciating mental state. As with severe physical pain, the risk of addiction is one of many factors to be balanced. Any treatment that is less addictive and more effective than the default choice, alcohol, ought to be given serious consideration. Note that the benefit is not just the possibility of decreased use of alcohol, but better and safer relief of prolonged mental anguish.

Hewitt (2013) argues that because mental pain is as real as physical pain, mentally ill people experiencing extreme suffering should not be categorically denied access to assisted suicide. I concur, but expand the scope of interventions to include access to substances and practices that might ease or relieve excruciating mental states, whether suffered by mentally ill people or people without a diagnosis. Taking mental pain seriously means a radical reevaluation of drug prohibition policies and the medical system itself, as well as an openness to practices and institutions that might work to alleviate excruciating mental states.

Inside Excruciating Mental States

The "excruciating mental state" phenomenon described here may be defined as:

1. An intense affective state, which may be described as anguish, desperation, misery, suffering, or hopelessness
2. That is higher in intensity and longer in duration than reasonably necessary to motivate appropriate behavior

Such a definition, however, is incapable of conveying the nature of the phenomenon. It is interesting that this phenomenon does not appear to have a generally accepted name, though there are many words that capture part of the sense. "Anguish" describes the mental state itself. "Desperation" connotes the unbearableness of the experience and the desire for relief. "Hopelessness" conveys the sense of a time late in the process, in which the sufferer cannot find an available remedy and loses faith that one exists. "Suffering" is too broad, for it includes mental states that have a function or meaning to the sufferer. In the most excruciating mental states, function and meaning are impossible.

Most people, thankfully, will never experience the most intense forms of this phenomenon. Authors writing about this state outside of modern academic

psychology have displayed a suspicion that it cannot really be conveyed to one who has not experienced it. In *The Varieties of Religious Experience*, William James (1902/1985) used long quotations from sufferers to communicate the phenomenon to his audience at the turn of the 20th century. He begins by explaining the phenomenon of anhedonia, the inability to take pleasure in activities. This grey world seems bad enough. But he goes on to describe a much worse state of existence:

> So much for melancholy in the sense of incapacity for joyous feeling. A much worse form of it is positive and active anguish, a sort of psychical neuralgia wholly unknown to healthy life. Such anguish may partake of various characters, having sometimes more the quality of loathing; sometimes that of irritation and exasperation; or again of self-mistrust and self-despair; or of suspicion, anxiety, trepidation, fear. The patient may rebel or submit; may accuse himself, or accuse outside powers; and he may or he may not be tormented by the theoretical mystery of why he should so have to suffer (p. 124).

James calls this mental state "morbid-mindedness," though not as a pejorative. The reality of this mental state is a problem for the prevailing religious mood, which James calls "the religion of healthy-mindedness"—"the method of averting one's attention from evil, and living simply in the good" (p. 136). An adequate philosophical doctrine, James says, must account for the evil facts of the world, for they have much to teach us.

James (1902/1985, p. 135) connects the intense negative affective state to the need for help:

> In none of these cases was there any intellectual insanity or delusion about matters of fact; but were we disposed to open the chapter of really insane melancholia, with its hallucinations and delusions, it would be a worse story still—desperation absolute and complete, the whole universe coagulating about the sufferer into a material of overwhelming horror, surrounding him without opening or end. Not the conception or intellectual perception of evil, but the grisly blood-freezing heart-palsying sensation of it close upon one, and no other conception or sensation able to live for a moment in its presence. How irrelevantly remote seem all our usual refined optimisms and intellectual and moral consolations in presence of a need of help like this! Here is the real core of the religious problem: Help! help! No prophet can claim to bring a final message unless he says things that will have a sound of reality in the ears of victims such as these. But the deliverance must come in as strong a form as the complaint, if it is to take effect; and that seems a reason why the coarser religions, revivalistic, orgiastic, with blood and miracles and supernatural operations, may possibly never be displaced. Some constitutions need them too much.

James (1902/1985) offers another method that may sometimes be useful for excruciating mental states: sensorily intense religious ritual. Baumeister (1991) proposes that charismatic religion, using provocative, tactile rituals like snake handling, laying on of hands, and glossolalia, allows participants to escape from excruciating affective states related to the self. Religious rituals that seem alarming from the outside may in fact be very effective in relieving mental suffering. Attending a boring church service would likely not have the desired effect.

A particularly important description of the extreme end of excruciating mental states comes from fiction. David Foster Wallace, who himself later committed suicide, suffered from severe depression. In *Infinite Jest* (Wallace, 2011), he describes the phenomenon from the perspective of a character called Kate Gompert. Like

William James, Wallace begins by explaining the phenomenon of anhedonia, and then contrasts this with the more severe form (pp. 662–698). Gompert refers to the excruciating mental state as "It," conveying Wallace's suspicion that the phenomenon cannot be named. This passage of fiction is more valuable than 100 science journal articles in conveying the texture and contours of the phenomenon. I quote it at length, but recommend the entire section to those interested in understanding the phenomenon from an inside perspective:

> It goes by many names—*anguish, despair, torment*, or q.v. Burton's *melancholia* or Yevtuschenko's more authoritative *psychotic depression*—but Kate Gompert, down in the trenches with the thing itself, knows it simply as *It*.*It* is a level of psychic pain wholly incompatible with human life as we know it … *Its* emotional character, the feeling Gompert describes *It* as, is probably the most indescribable except as a sort of double bind in which any/all of the alternatives we associate with human agency—sitting or standing, doing or resting, speaking or keeping silent, living or dying—are not just unpleasant but literally horrible.

The sufferer is unable to obtain *help*, in part because she is unable to communicate her condition. Like a "drug seeker" who cannot demonstrate a physical basis for pain, Gompert's suffering is not real, proper suffering, because there is no cause for it that others can see and verify:

> The authoritative term *psychotic depression* makes Kate Gompert feel especially lonely. Specifically the *psychotic* part. Think of it this way. Two people are screaming in pain. One of them is being tortured with electric current. The other is not. The screamer who's being tortured with electric current is not psychotic: her screams are circumstantially appropriate. The screaming person who's not being tortured, however, is psychotic, since the outside parties making the diagnoses can see no electrodes or measurable amperage. One of the least pleasant things about being psychotically depressed on a ward full of psychotically depressed patients is coming to see that none of them is really psychotic, that their screams are entirely appropriate to certain circumstances part of whose special charm is that they are undetectable by any outside party ….

Finally, suicide is understood as a desperate flight from this mental state, when it has become unbearable and no relief is available:

> The so-called "psychotically depressed" person who tries to kill herself doesn't do so out of quote "hopelessness" or any abstract conviction that life's assets and debits do not square. And surely not because death seems suddenly appealing. The person in whom *Its* invisible agony reaches a certain unendurable level will kill herself the same way a trapped person will eventually jump from the window of a burning high-rise. Make no mistake about people who leap from burning windows. Their terror of falling from a great height is still just as great as it would be for you or me standing speculatively at the same window just checking out the view; i.e. the fear of falling remains a constant. The variable here is the other terror, the fire's flames: when the flames get close enough, falling to death becomes the slightly less terrible of two terrors. It's not desiring the fall; it's terror of the flames. And yet nobody down on the sidewalk, looking up and yelling 'Don't!' and 'Hang on!,' can understand the jump. Not really. You'd have to have personally been trapped and felt flames to really understand a terror way beyond falling.

Again, only a small proportion of people experiencing excruciating mental states will commit suicide. However, a higher proportion of people long for, pray for, wish for, or fantasize about death, in the absence of suicidal action or even intent. A

"passive death wish" is not uncommon; a general survey of adults 50 years and older in Europe found that 15% of those 75 years and older reported a death wish (Ayalon & Shiovitz-Ezra, 2011). This percentage decreased with age group; just under than 5% of those 50–64 years reported a passive death wish. Fantasizing about suicide may even be a form of relief for some people (Maltsberger, Ronningstam, Weinberg, Schechter, & Goldblatt, 2010). Although not necessarily a predictor of suicide, the presence of a passive death wish or suicide fantasies is a common feature in excruciating mental states. It is not clear how common passive death wishes are outside of excruciating mental states; if happy people rarely long for or pray for death, the passive death wish could be used along with other criteria to identify the presence of excruciating mental states. Certainly, a person who longs for death is in need of help and relief.

Communicating the Hidden Obvious

In evaluating physical pain, doctors employ pain scales to gauge the severity of pain. Patients who can communicate verbally can be asked to rate their pain on a numeric or graphic scale; those who cannot communicate (because they are too young to speak, or in a coma) can be evaluated based on outward signs (e.g., Voepel-Lewis, Shayevitz, & Malviya, 1997).

There are at least six numerical scales or instruments for assessing mental pain (Tossani, 2012), but none of these is commonly in use. If excruciating mental states are to be studied and treated, a widely accepted intensity measure would provide a basis for evaluation and comparison.

The person experiencing an excruciating mental state has two communication problems. First, he may be unable to describe his mental state. Second, even if he manages to describe it articulately, others capable of helping him have no mental category with which to understand it. "Mental illness" is the nearest category, but this concept barely connotes the excruciating mental state. "Whining" is another possible match, and I have already mentioned "drug seeker." The mere existence of a socially accepted name for this category of experience could allow more suffering people to get help.

A common moral intuition, affirmed by theoretical work in ethical philosophy, is that those who are worst off are especially deserving of help. People suffering from extreme forms of excruciating mental states are at the bottom of the utility distribution. Under the present system, not only are they denied help, but they are denied the ability to help themselves. Policies that would help sufferers of excruciating mental states are not without risk. But these risks should be considered in light of the burden currently placed on people suffering the worst forms of human experience. There is no suicide crisis, but their situation is truly an emergency.

Conclusion

Current models of suicide emphasize the connection between suicide and mental illness. The focus on mental disorders and the rare phenomenon of suicide has shielded a more important problem from view, one that is obvious upon reflection but rarely named: extreme subjective mental suffering or excruciating mental states. Excruciating mental states mediate the relationship between mental illness constructs and suicide. Prolonged excruciating mental states do not serve any purpose that can justify refusing to ameliorate them. Excruciating mental states can be relieved directly, rather than as a hoped-for consequence of the treatment of a mental illness. Unfortunately, drug prohibition policies preclude the use of safe, effective solutions. The difficulty of communicating extreme mental pain prevents sufferers from being helped; simply having a conceptual category for the phenomenon may ease the difficulty of communication.

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Anthropathology: The Abiding Malady of the Species

Colin Feltham

Introduction

A view of negative human evolution is put forward here by way of balance against the many positive, sometimes romantic, 'ascent of man', or academically cautious and narrow accounts.[1] Anthropathology is advanced as a hypothetical, quasi-singular entity with multiple roots and micro-manifestations, with some attempt being made to suggest a chronology and a speculative aetiology. Anthropathology is characterised by damaging features such as violence, greed, deception, extended niche construction, and complex suffering on a scale never been known among other species. This is an interdisciplinary endeavour that will probably not satisfy readers with demands for detailed, specialist, and evidence-based prose. No attempt is made to proffer solutions to the existential problem of anthropathology. Given certain controversial aspects, a look at cognate disciplines and epistemological tensions is included.

What Is Anthropathology? An Overview

As the name suggests, anthropathology is the core sickness[2] of the human species. But some difficulties immediately arise. First, there is no such established entity or discipline that studies it, merely a hypothesis put forward by Feltham (2007). Secondly, the objection is made that the concept of pathology cannot be legitimately applied to an entire species. Thirdly, it is understandable but incorrect that some

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equate anthropathology with misanthropy. These points will be addressed in due course. Anthropathology is a countercultural proposition, if we accept that mainstream culture is built on an assumption that life is good, or a mixture of good and bad but generally always progressing. It proposes that human existence is far from alright, that it is deeply warped, and that our nature is to some extent negatively engrained morally, psychologically, and neurologically. Humanity has a confused consciousness that commandeers and consumes its environment, denies many problems, and externalises its confusion.

It is clear that *Homo sapiens* has evolved beyond the evolutionary stage of even our nearest mammalian relatives. It is equally clear, however, that humanity has developed negatively both in terms of our own welfare and that of the biosphere. While we acknowledge our animal origins and kinship, and congratulate ourselves on our cognitive and cultural superiority, we cannot ignore the vast harms we do that make us compare poorly with other animals (Masson, 2014). Our violence, war, and destruction are superfluous to our survival needs, and indeed threaten them. Our vast, earth-spanning population dwarfs that of many other species, and our habitat encroaches on those of other species. We are grappling here with what we call the human condition and human nature.

The objection can be made that a pathological entity like cancer can usually be clearly identified, categorised, understood, and treated, successfully or otherwise. Cancer is distressing, painful, and often life-threatening, indeed often disfiguring and fatal. But anthropathology is not readily identified or treated, and is not obviously fatal. Like psychopathology, anthropathology is an umbrella term covering many distressing entities. We do not fully understand or have satisfactory treatments for psychopathological problems (psychiatric, clinical-psychological, and psychotherapeutic protests aside), and many of them are disputed in terms of aetiology and discreteness. But psychopathological entities afflict individuals and groups, whereas anthropathology appears to have a much broader remit. However, insofar as anthropathology is posited as a human universal, we can regard it as the sum total of all our unwieldy *kluges*, or makeshift adaptations that outlive their original purpose (Marcus, 2009). We might also use the term *equifinality* to account for anthropathology's many causal threads, insofar as *all roads lead to Rome's decline and fall*. Evolving haphazardly, we have inherited many viable and some elegant features, and many dysfunctional and ugly features. At the individual level, we each have a unique micro-anthropathological profile—one is greedier, more violent, more deluded, more prone to anxiety than the other, for example. Among different institutions, we can witness variable manifestations of dehumanisation, exploitation, and absurd rituals (macro-anthropathology). Anthropathology permeates all human endeavours and comorbidity is the norm at all levels. True, it is usually intertwined with some 'good' features (in the Church, for instance), which duality I examine below.

Insofar as human neonates do not exhibit (yet, many) anthropathological features, we might regard them as nonetheless possessing the neural wiring that is ready to imitate and accept common cultural conditioning. In other words, many thousands of years of cumulative anthropathology have primed us to activate

individually the capacity for lying, dissembling, dissatisfied cogitation, empty social ritual, concealed suffering, and so on. As well as walking upright, wearing clothes, building houses, and being cooperative and altruistic, we cheat, exploit, and hurt other human beings and animals, and we create elaborate ways of killing time, overcoming meaninglessness, and denying our mortality. Few of us are free of this two-sidedness, which we inherit and pass on. 'Man hands on misery to man/It deepens like a coastal shelf', as the poet Philip Larkin put it. Often attributed psychoanalytically to our parents as incompetent, damaged, or malevolent, they are better understood as the hapless transmitters of a transgenerational pathology. Anthropathology is in this way transmitted vertically through generations but also, via our cultural and built environment, horizontally. We are infected, as it were, and constantly reinforced in our pathological behaviours, by our unwitting peers and our shared, damaging environment: we know not what we do, nor how to extricate ourselves.

How Anthropathology Might Have Evolved

Most religions contain narratives of a Fall. Life was originally good, we were created, loved and protected by God, and lived together harmoniously, but we sinfully disobeyed God's injunctions and were condemned to lives of sin, evil, suffering, and death. Some take this story literally, some dismiss it altogether, but some of us suspect it may contain an oral and allegorical history of a major bifurcation in human affairs. Unfortunately, religions persist well beyond the point at which they initially offered something useful (besides emotional comfort to some) and their primitive, distorted grasp of matters we now know much more about is an obstruction to mature knowledge. Buddhism and other Asian religions should be briefly mentioned, however, as godless and polytheistic religions that do not focus on evolutionary or historical causes so much as a mythical *samsara* (endless cyclical rebirths of the individual characterised by suffering) and a methodology for overcoming this in a final transcendence (*nirvana*). However, although we now have increased and better scientific theories of anthropathology-related matters, none is wholly satisfactory and many are in some conflict with each other. In this section, I examine some of the most interesting and promising of these theories of flawed humanity.

Big history, overlapping with deep history,[3] is the attempt to go beyond the traditional, limited purview of recorded history by beginning at the very beginning of everything (Spier, 2011) and including as many large variables as possible. In this way, big history is academically challenging. Big history starts 13.8 billion years ago with the Big Bang and seeks to trace historical development from non-complexity to complexity, through the 'Goldilocks conditions' in which we have emerged on earth, to the present day. Given the overall cosmic trajectory, it looks inevitable that humanity would have arisen, would increase in complexity, and also eventually perish, in line with the entropic principle. 'Exhaustion of critical resources and growing entropy' (Spier, 2011, p. 200) are the key issues facing us.

Regardless of moral judgements of how we have acted historically, in different eras and places, the question of how we as a species address the problem of available energy is crucial. Big history holds up the largest of pictures in a way that reduces the significance of our past good and bad actions, perhaps even implies that human history could not have unfolded otherwise, but confronts us with the merciless facts of near-term threats and ultimate extinction. In the big picture, the details are probably unimportant, or are of mainly scholarly interest rather than practical, problem-solving use.

Palaeobiology should hold clues to the origins of suffering, pain, and predation. Humans cannot be said to have invented these since all animals are susceptible to them. What we must ask here is whether primitive life forms inevitably contained the potential for later, complex forms of suffering, pain, and predation. Both simple and complex organisms continue to exist, each having its merits for survival. Biological complexity often involves co-evolution of host and pathogens, not necessarily in active but in passive terms, for example, adaptations by pathogens for evading immune systems. Moalem and Prince (2007) reinforce the case that certain diseases have benefited us, and by extension we might have to resign ourselves to the prospect that anthropathology in various forms must always remain with us. Also, speculatively implicated in the research of Nithianantharajah et al. (2013) is a genetic accident 550 million years ago, to which vertebrate cognitive complexity and susceptibility to mental illness may be traced. Such lines of enquiry suggest that evolved human intelligence may never be sundered from accompanying high risks of psychopathology, unless genetic engineering becomes supremely sophisticated.

Zoological evolution and its quirks also contain the phenomena of mourning among some animals and gratuitous aggression and rape-like behaviour in others (Wrangham & Peterson, 1997); and comparative psychopathology suggests the capacity among many animals for species-abnormal behaviour (McKinney, 1988). If we are indeed the pinnacle of evolution, it is not necessarily in the sense of magnificent superiority but of highly complex, kluge-like adaptations. We are, we remain, part of an evolutionary arms race. Even today, our most advanced medical research battles to find alternatives to antibiotics as microbes become resistant to them. Retrospectively, it appears inevitable that natural selection should have led to *Homo sapiens*, an organism so complex and ecologically pervasive as to become, arguably, ultimately and fatally unwieldy and threatening to the biosphere. In this sense, we can be seen as a cancer-like manifestation of entropic complexity, doomed to join all other species in eventual extinction.

Distinctive human consciousness is usually celebrated as a superior attainment but it remains poorly understood and little agreement exists as to its origins. Animals have some form of primary consciousness but none appears to approach the threshold of our own. Sterelny (2003) advances a nuanced argument combining early human preferences for niche construction, pain avoidance, co-operation, and eventual complex cognition. Most evolutionary psychologists favour the concept of modularity of mind according to which domain-specific modules have evolved in response to selection pressures. Other academics wish to emphasise epigenetics and neuroplasticity, whereby the brain is portrayed as more oriented to new learning. I

want to sidestep most of these controversies about how the mind works and instead focus on how it malfunctions, or rather how it can be considered to be malfunctioning in a largely unrecognised manner.

Anthropathological consciousness may be considered a paradox. Growing consciousness appears to be self-evidently and invariably a *good thing* but a little analysis might suggest otherwise. The Norwegian philosopher Peter Wessel Zapffe (1933/2004) argued that a form of surplus consciousness evolved in us that made us unfit for satisfied survival. Diamond (1991) later speculates on connections between the male bird of paradise and its extravagant tail, and the tendency of many human males to become addicted to high-risk substances and activities, a perverse feature of sexual selection that Miller (2001) has further elaborated. In Zapffe's account, our consciousness is an overdevelopment of the cognitive faculty that renders us so knowledgeable of natural limitations—the indifference of nature to our existence, our loneliness in the cosmos, the godlessness of existence we must face, the inevitability of deterioration with age, personal death, and the ultimate extinction of everything—that we have to deny this knowledge in some way. Too late for us never to have been, but still better not to know the brutal truths of our circumstances and fate, and better not to have to constantly wrestle with them internally.

Zapffe's conclusion was that we resort to four main defence mechanisms (he was inspired partly by Freud): 1. *Isolation* (or denial) by altogether blocking out the threatening knowledge from awareness. 2. *Anchoring* ourselves within constructed belief systems such as religion, politics, and hope for the future. 3. *Distraction* of all kinds that allows us to focus on matters outside ourselves. 4. *Sublimation* as the focusing of awareness of what is threatening in an aesthetic manner, for example, writing artfully about death. Zapffe developed these ideas from a philosophical base, before and independently of those of the cultural anthropologist Ernest Becker (1973) and the terror management psychologists. While these suggested defence mechanisms could be extended and improved, they present a fair picture of the existential contortionism we indulge in. It may be self-evident that these defences are only necessary, or even possible, when the fundamental necessities of life have been satisfied. That is, as long as our attention is devoted to sheer survival we do not focus on our own thoughts of death and meaninglessness. The paradox here is that our very success as a species in surviving and allowing ourselves the luxury of cognitive reflection creates the conditions for the surplus consciousness which, reflux-like, then torments us.

Zapffe's views are partly replicated by Varki and Brower (2013), without their knowledge of Zapffe's writings (still mostly untranslated into English at the time of writing). While Zapffe's thinking came from philosophical and environmental sources, Varki and Brower come from scientific backgrounds (glycobiology, and molecular and cellular biology, respectively). Their starting point was to ask why no other animal has ever come close to the sophistication and complexity of human consciousness and the behaviour that flows from it. Using theory of mind as their guide, they postulate that when an animal witnesses a fellow's suffering and death there is often an affective response, but not a vivid inference that such experiences will afflict oneself. In other words, developing consciousness encounters a problem

when it is capable of realising that one must die; it is a problem because it upsets the survival instinct. It is perhaps a problem for sexual selection insofar as those morbidly preoccupied are less likely to attract mates. So Varki and Brower argue that a full theory of mind, or full consciousness, could arise only when another mechanism was in place—the ability to deny our awareness of our own mortality. For whatever reason, *Homo sapiens* was the species that, perhaps around 40,000 years ago, evolved simultaneously and paradoxically a consciousness of death and the ability to deny it—surely the ultimate in cognitive dissonance. As Trivers (2013) notes, deception is an important evolved strategy, and we are paradoxically better deceivers when we are self-deceived.

Spikins (2015) advances the hypothesis that from 100,000 to 6000 years ago when the outermost parts of the earth were explored, the relatively rapid worldwide dispersal of *Homo sapiens* can be explained by moral disputes between small groups. Evolving emotional complexity led to moral disputes that in turn became violent, reinforced by a drive to punish those whose actions did not appear to match another group's moral code. In this account, the 'dark side' of human nature may be inferred as coming from emotional complexity and moral tensions sometimes erupting into lost trust, altruistic anger, spite, vengeance, and hatred, and the need to put distance between one group and another.

A quite different take on distinctive human evolution is offered by the archaeologist Timothy Taylor (2010). In this account, technology is central. For Taylor, 'there was an actual moment when we became human' (p.2). Brushing aside theories of religion or death awareness as pivotal, he goes back over two million years to the first origins of tool use. As the weakest of the seven great apes, we are now wholly reliant on technology. This has come about, in his account, because dependency on artificial aids has domesticated us, altering us profoundly. From primitive weapons, tools, containers, and decorations, we gradually moved to clothing, burial sites, homes, fire use, agriculture, domestication of animals, fences, towns, and on towards the wheel, boats, and other means of overcoming natural selection pressures. Fast-forward to the present, and we have bigger, better, and more of everything artificial. We are dependent on weather-specific clothing, heated homes, appliances, eye glasses, medications, as well as (most of us) tea, coffee, alcohol, meat, cosmetics, books, cars and roads, computers, cellular phones; and (some of us) illegal drugs, planes, and ships. Smail (2008) reflects as a deep historian on the role of stimulants, among other things, in neurophysiologically meshing culture and evolution. Collectively, we seem unable to live without offices, churches, shops, schools, factories, mass entertainment, hospitals, and armaments. Many of us owe our longevity to medications and surgery, and millions depend on the daily support of prosthetics. In Taylor's terms (and see Spengler, 1931/2015), we cannot now survive without technology. The overarching trade-off is that as we grow more technologically complex and powerful collectively, we also become weaker individually, losing our sensory acuity and muscular strength.

Taylor does not focus much on what Tallis (2003) calls the tool-of-tools, the human hand. Not matched elsewhere in the animal world, our hand has a fully opposable thumb, and is multi-functional; it is the basis of all our weaponry and

artefact construction but also of our communication and numbering. Tallis outlines the steps that may have taken us to being the conscious human agent, all traceable to the hand.

A number of theories date widespread human malfunctioning closer to the present. Most evolutionary mismatch theory refers to the transition from hunter-gatherer existence to agriculture about 10–12,000 years ago, the end of the Pleistocene era. This mismatch is between our long evolved biological adaptations with 'stone age minds' to a stable natural environment and today's unnatural environment, and includes behavioural problems such as phobias, jealousy, and criminal aggression, and medical problems like myopia, diabetes, and osteoporosis. For Diamond (1987) the advent of agriculture 1000 years ago was 'the worst mistake in the history of the human race'. Coincidentally, this is possibly the temporal point at which patriarchal dominance took hold, and where anarcho-primitivists like John Zerzan (2002) place our putative wrong turn. In Zerzan's view, only the complete rejection of modern industrial-technological society (and indeed agriculture) can restore human sanity. Note that this conclusion, based on something not so far from the reasoning of Taylor (2010) as to the technological route to our malaise, comes to quite the opposite conclusion of that of Taylor, namely, that we cannot go backward but only forward into further technological advances. To complicate matters, Homer-Dixon et al. (2015) call for new conceptual frameworks to understand the 'deep causes of synchronous failure' now facing us. Noting a 'long fuse big bang' mechanism at work in our impending global crisis, the authors nevertheless ignore the evolutionary, indeed big history, factors contributing to the long fuse.

Out of interest, we could refer here to any number of related hypotheses, most of which have been discarded, remain in doubt, or are highly contentious. On an actual historical Fall, De Meo (2011) argues that a process of geographically specific desertification approximately 6,000 years ago triggered a huge wave of violence, war, and unnatural behaviour. Taylor (2005) supports this account. Much more specifically, Cline (2015) puts civilizational collapse centring on Egypt at 1177 B.C. We can certainly ask whether dramatic climate changes in some eras led to famine and population decimation, accompanied by rapid ingenuity and harsh decisions regarding the fit and unfit to survive (Calvin, 2002). We should note that more environmentally rapacious populations may have been forced to adapt to harsh climates by inventing intensive food production methods, patriarchal controls, and eventually industry. Indeed, some Afrocentric hypotheses centring on the role of melanin attribute to northward migration to inhospitable cold climates the evolution of white people as barbaric and unfeeling. Elaine Morgan's largely dismissed aquatic ape hypothesis disputes the argument that we lost our hair so that we could run and sweat while hunting on the savannah, and places emphasis on human evolution in and near water, as well as focusing on the evolution of children and women, and on salient inherited anatomical features (Morgan, 2000). Morgan's is one among several hypotheses giving more weight to a female perspective on evolution.

Julian Jaynes (1976) proposed the novel theory that before only 3,000 years ago human consciousness was characterised by a bicameral (or hemispherical) mind in which one half 'spoke' authoritatively (much like a god) while the other heard and

obeyed. Put differently, humans experienced their existence somewhat like schizophrenics receiving commands and this state of affairs broke down in antiquity when we evolved our now well-known subjective consciousness. Most do not now regard Jaynes's thesis as plausible but some sympathy for it remains. McGilchrist (2009) proposes another argument for the significance of our left and right brain hemispheres that reverses Jaynes's conclusions. In this, the right hemisphere is wisely and holistically connected to the earth, emotion, poetry, and dream, while the left is dominated by rationality and analysis. We have become unhealthily over-controlled by the left hemisphere, such that the guidance of the right, or the proper balance between the two, has been lost. Many support McGilchrist's thesis for the emphasis it gives to an agenda of re-enchantment in opposition to 'dehumanising' modernity. But for McGilchrist, schizophrenia is a disease stemming from only the 18th century, whereas Horrobin (2002) has argued contentiously that it may go back to between 80,000 and 140,000 years, connected with increased human consumption of fatty acids, the hungry brain, and creativity. Also, while McGilchrist sees the right hemisphere as holding hope for our survival and necessary re-humanisation, Hecht (2013) argues that *pessimism* is neurologically dominant in the right hemisphere. Similar problems regarding brain structure arise in relation to different interpretations of the tripartite brain, with the over-rational neocortex supposedly dominating the limbic functions of affect and sensory acuity, and reptilian mobility.

Many interpreters of the human condition prefer to believe that things have gone seriously awry only since the advent of the 15th century 'age of discovery', experienced as invasion, infection, slaughter, colonisation, and slavery by its non-Western victims. The native American Hopi noun *koyaanisqatsi*, meaning something like 'untenable disorder and craziness', captures this perception of a serious breach in the natural order. Other analysts opt for the industrial revolution, the growth of capitalism and of late modernity (Clark, 2002; Hookway, 2015) as culprits. Needless to say, such accounts often come from the political left, who may harbour a rather rosy view of human nature, oppose deterministic aetiologies rooted in evolutionary theory, and await a socialist revolution. But it is also possible to argue that the stressful complexities of modern capitalist life (Rosa, 2015) blend together with trends going back thousands of years, and possibly accelerated by the consequences of agriculture combined with increased gene flow across once-separated populations. In this account, biological evolution did not reach a stasis 50,000 years ago but has speeded up. Indeed, anthropologists Cochran and Harpending (2009) interpret recent population genetics data to argue that evolution has accelerated in the past 10,000 years by a factor of 100 times compared with the previous six million years. A very dark reading of these trends might see them as that admixture of elements that presages an irreversible late stage of social and species entropy.

Given the tendency of archaeology to periodically turn up findings that subvert our current assumptions,[4] we should exercise some caution as to which hypotheses we prematurely elevate and which we consign to the annals of the disproven and ridiculous. If we accept that something akin to anthropathology exists, we yet have no decisive aetiological account of it. It could have arisen as a useful accident. It

appears to be a cumulative series of adaptations that have become too complex and too path-dependent to continue to benefit us indefinitely. But acceptance of its existence, importance, destructiveness, and urgency would at least demand that it be taken seriously alongside other disciplines.

How Anthropathology May Be Transmitted

Anthropathology is transmitted somewhat haphazardly by evolutionary, cultural, developmental, and cognitive means. We appear to inherit many traits, positive and negative, from our distant and near ancestors, and it appears true that we have quite recently learned to become less violent, for example. The philosopher of biology Kim Sterelny (2014) focuses on the positive benefits of human intergenerational knowledge transmission but downplays the many negatives. Nested within us are many animal instincts, and also many evolutionary mismatch problems which we cannot simply shake off (Clack, 2009; Gluckman & Hanson, 2008). Dean (1997) applies genetic, neurobiological, and chaos theory to individual lives of substance misuse, suggesting many unpredictable, fractal outcomes. But our cultures also load many values on to us, some of which appear optional and others difficult if not impossible to resist. For most of us a job is necessary, for example, not only for income but as a means of structuring time, appearing normal, gaining status, attracting mates, and providing for our families. But today, in many cultures we can choose to embrace or deny religion. Biological imperatives—eating, drinking, self-protection, mating—remain, our ability to commit suicide or refuse to reproduce admittedly being major exceptions, and mostly achieved technologically. Indeed, it can be argued that some cultures have swung from rigidly limited behaviours to a counterproductively choice-saturated individualism.

One of the transmission routes for human values and behaviour, good and bad, is that of early individual development. Human upright gait, relatively frequent pregnancies, narrow birth canal, perilous birth process, and long vulnerable infancy make for a high-risk beginning to life, including intrauterine trauma, and extended dependency, all foci which certain models of psychotherapy have sometimes controversially explicated. We have no early choice but to balance our animal instincts (to cry in pain and hunger, or for attention, and to urinate and defecate) with our caregivers' conditional nurture, preferences, and whims. Parents as social agents have no realistic choice but to transmit expectations to us: we must learn to walk, speak, and behave in socially accepted ways. It is established that we are neurologically primed to learn language and to learn it correctly. We are taught not to refer to ourselves in the third person ('Johnny hungry') but in the first ('I'm hungry'). We are not born thinking but feeling, but language and social injunctions enable and probably force us to internalise and symbolise our feelings. We emote less as we grow, and think more, a developmental change paralleled in our evolution (Campbell, 1975). The thinker, according to this line of reasoning, becomes lodged in our heads as 'I', the detached ego. An internal struggle is established between the 'uncivilised

animal baby' and the individual who must fit in by suppressing disallowed spontaneous expressions. Freud's tripartite model of id, superego, and (judicious) ego roughly covers this dynamic process. Suppression and loss of some human functions underpin civilisation (Freud, 1908/2001), a thesis elaborated by Hobbes and opposed by anarchists.

As we grow (the 'we' here is, admittedly, presumptuously generalised) we inevitably experience some conflicts between our raw perceptions and needs, and the culturally normative, dominant narrative. Somewhere along the line, sensory awareness reduces, idiosyncratic qualities are somewhat smoothed out, and the person accepts social conditioning, much of which entails living and perpetrating a lie. (Consider the characters of Leo Tolstoy's Ivan Ilyich and Arthur Miller's Willy Loman as key examples.[5]) In spite of residual awareness that the normatively transmitted worldview contains many glaring flaws and lies, sooner or later most of us must succumb to resignation, a point Griffith (2004) places somewhat arbitrarily at the age of 12 years but which many probably know from adolescence.

We are not born wholly anthropathological, then, but have it thrust upon us. In this model, the vast majority of us have no choice but to adopt the *anthropathological false self*. I believe this equates roughly with what Bohm (1994) refers to as 'thought', which contains a 'systemic fault'. This is the same as the 'I the thinker' but it is inevitably a self painfully divided as it struggles to reconcile what it actually sees and feels with what it is told it sees and feels. The sheer pressure of living in mass civilisation, which constantly reinforces the falsely adapted self, keeps us both on track and in perpetual conflict with ourselves. Call this alienation or any other name. Each of us searches for a social niche, a haven of relational, familial, cultural, and occupational comfort, in which we can survive, dimly aware that our society both protects and threatens us. In this way, we are all simultaneously perpetrators and victims of anthropathology.

Social brain theory (Burns, 2007) suggests that psychoses result from that aspect of evolution that has demanded 'a capacity for complex social and interpersonal relationship' (p.181) that exceeds the ability of some of us to cope. What is sometimes referred to as our 'extreme sociality' and 'hyper-co-operation' is also an Achilles' heel, pushing some into madness and many into borderline states where the requirements of constant social monitoring and responding appropriately become too costly. Although space restrictions prevent further exploration of the topic, related phenomena in mass psychology are relevant here. To take just two phenomena, consider trance-like and stampede-like behaviours. An example of the first might be climate change denial and associated policy inertia. As regards the second, anything from the fashion for tattooing, and nothing-to-lose migration, to a return to fundamentalist religion, might qualify. With contemporary mass populations we see such phenomena on a scale not known before, but where unconscious evolutionary and historical drivers augur badly for our prospects. While fragile individuals might be said to implode into mental illness, groups spread anthropathology outwardly in the direction of social chaos and incipient wars. Walsh (2014) takes up some related biosociological themes.

The Duality of Human Consciousness and Behaviour

Homo duplex, we are long accustomed to thinking in terms of good and evil, both theologically and psychologically. It is counterintuitive to assert that *all* human life and achievement is bad, just as its opposite affirmation is not credible. As E.O. Wilson (2014) puts it, 'We are all genetic chimeras, at once saints and sinners, champions of truth and hypocrites … because of the way our species originated across millions of years of biological evolution' (p.28). Just recall how the Catholic Church, for all its good works, has been sullied historically by violence and corruption, and rocked by paedophile scandals in recent years. Few would argue that human existence contains no good and no bad elements. But we will certainly argue over the balance of joy, beauty, love, humour, and achievement versus suffering, ugliness, evil, decay, and death. Among a few others, Benatar (2016) has argued that life is bad enough (indeed asymmetrically weighted towards the bad) to make a strong moral case against further procreation, and a sober review by psychologists Baumeister, Bratslavksy, Finkenauer, and Vohs (2001) concluded that bad outweighs good. De Waal (2006), among others, makes a case for a two-sided or bipolar nature illustrated by reference to our ape cousins, chimpanzees, and bonobos. Diamond (1991) remarks on the two-edged sword that agriculture represented in our emergence. Talbot (2005) makes use of paradoxical systems theory to analyse our evolved dualist readiness to respond to challenges in different ways. In any case, we seem unable to transcend thinking in such dualistic terms. And as we have seen, Jaynes, McGilchrist, and many others have attempted to explain and resolve our hemispherical problems.

Bohm (1994) argues that we humans have great difficulty in being aware of when our actions are going dangerously off course to adjust them in a timely manner. Comparing subtle bodily proprioception with cognitive stubbornness, he searches for ways in which the human mind might recapture some of this subtle, agile, corrective cognitive ability. Although gifted problem-solvers, we are also susceptible to failures in awareness and adjustment. This may be accounted for by our fierce (sometimes homicidal or self-defeating) attachment to habits and traditions, our path dependency, and our tendency to overcorrectiveness in some matters. We often assume that if one course is unprofitable or wrong, its opposite must be right. For example, in politics we are often sharply divided in our affiliations and we can sometimes swing from harsh dictatorship to ineffective soft democracy, probably neither of which is optimal. We can even perform this *volte-face* within ourselves, for example, the radically left-wing young person becoming a rigidly right-wing advocate in old age.

In the domains of politics and technology, say, quite often we fail to anticipate the unintended consequences of our actions, some of which may be disastrous. Ingenious problem-solving in the medical area, to take one example, can lead to new problems of antibiotic resistance, hospital-borne diseases, old age diseases and disabilities, high costs of desired drugs, and unsustainable costs of increasing lifespans. Very commonly we do not appreciate the operation of diminishing returns, as

our hopes and investments in one political, economic, or other endeavour yield poor repayment for great effort. We are hardly alone among species in such adjustment problems, but the scale of our projects is often so large that the consequences can be hugely damaging. Nuclear arms, rampant capitalism, and unsustainable levels of carbon emissions are clear examples. I have elsewhere referred to these self-defeating dynamics as anthropathological loops (Feltham, 2007), our very capacity for inventiveness also being a curse.

Manifestations of Anthropathology

Anthropathology subsumes violence, tribalism, greed, deception, untenable expansion, and pervasive suffering under its aegis. We have only recently spoken more of shared psychological pathology than moral evil (Staub, 2003), and the literature on culturally variable evil is limited (Parkin, 1985). Our Western epistemic tradition since at least the time of Aristotle has divided knowledge into discrete disciplines which facilitate ever greater expertise but far too little consilience or actual problem-solving. We assume that progress is made in this way but Bohm (1994) suggests we deceive ourselves, or rather *thought* deceives us into believing we are judiciously running the show, while in fact thought itself long ago took over, with its problematic fragmentation of perception. As the pessimistic historian Oswald Spengler (1931/2015) put it, 'Man has become the slave of his thought' (p.52). Our common thought system tells us that the individual and society are separate, that mental illnesses are discrete entities, internal distress is different from criminal acting out, most people are good or have only peccadilloes but violent criminals are beyond the pale, capitalist exploitation is legitimate but personal freeloading and cheating is not, and so on. But looked at without these assumptions, we might see all such foci as mere manifestations of the same underlying pathological dynamic.

Already we see debates about the reality or illusion of different mental health diagnoses and their putative aetiologies. It is less clear than it once was how depression and anxiety are distinct from each other, for example, comorbidity being more likely. Anti-psychiatrists or critical psychiatrists and critical psychologists dispute the existence of schizophrenia and attention deficit disorder. It has been suggested, and I concur, that we are all 'neurotic' in one form or another (Charlton, 2000; Ratey & Johnson, 2004), just as we all necessarily differ in personality from each other (Rich Harris, 2007). Reasonable evolutionary explanations for differing mental illnesses are offered by Gilbert (1989), Nesse (2005), and Stevens and Price (2015), among others, but these focus on mental ill health as if it is discrete. Evolutionary explanations for post-traumatic stress disorder (PTSD) are offered (Cantor, 2005), but the likelihood that civilisation is partly constructed around the *avoidance* of trauma is barely explored; much medical progress can be regarded as driven by strategies for avoiding terror and pain. The psychopathic (or sociopathic, depending on UK/USA conventions) manager who leads a profitable business effectively is understood to be common and is not necessarily diagnosed. The

academic, particularly in science and mathematics, whose achievements are not matched by his interpersonal skills or happiness, may nevertheless thrive reputationally. Some diagnoses are apparently prevalent in certain places and non-existent in others. Shyness is normal in Japan but a social phobia in the USA.

In the common assumptive world, the majority is normal, well adjusted, and happy, with only a minority being deviant. In a certain 'politically correct' interpretation, we are all different and equally valuable; or in another, patriarchal dominance is to blame for everything that goes wrong. The right-wing may insult the left as the 'loony left' while the left freely diagnose homophobia and xenophobia among the right. The religious may speak of the sins of the infidels, while atheists can retaliate in terms of 'religious delusions'. In practice, we increasingly recognise that modern life can be stressful and hence many need to consult psychotherapists who, however, are themselves only human and riddled with problems of their own (Adams, 2014). Irish writers James Joyce and Samuel Beckett wrote respectively of 'unhappitants of the earth' and 'you're on earth, there's no cure for that'. In other words, we are all affected by and complicit in suffering. Erich Fromm (2011) discussed what he saw as the pathology of human normalcy, constituted by narcissism, alienation, consumerism, and a religious vacuum. He was certainly not alone in his diagnosis of society as sick. Foucault (1989) too, of course, drew attention to the ways in which social ills are projected on to individuals and some minority groups. Farmer (2004) writes of the pathology of the indifferent healthy and uber-wealthy, while for Zerzan (2002) our whole way of post-hunter gatherer life is pathological.

Hardest of all for most of us is to acknowledge our own shameful shortcomings, indeed our anthropathology, which is evident at the levels of ego and tribe but concealed by our blind spots. Contrary to this, one extreme of self-denigration is the person suffering from obsessive-compulsive disorder who falsely believes he has committed grave crimes, a problem shared with those whose religiosity may see them flagellating themselves. But it has long been those in positions of perceived greater integrity or virtue—priests, politicians, psychiatrists, doctors, lawyers, academics—who probably find it hardest to admit to serious personal and occupational errors or failures. Indicting our leaders, however, may also remind us that they can serve our need for the illusion that at least some of us escape from anthropathology. Surely Jesus was without sin and resurrected from the dead, surely the Buddha attained enlightenment even if very few of us do. Surely our selfless professors are rapidly discovering important new life-enhancing, disaster-averting theories and practices, and not merely obsessing over pet theories, jostling for status among their peers, and advancing their own careers.

Anthropathology and Pessimism

Anthropathology carries a dark view of human nature but is not based misanthropically on hatred or an endorsement of voluntary human extinction. It may have diverse roots and manifestations but arguably its central dimension relates to

consciousness producing uncomfortable knowledge that confirms inescapably our individual mortality, probable species extermination, and eventual cosmic annihilation. We immerse ourselves in life-affirming projects that can banish death awareness to the periphery of our consciousness and that act as an existential shield against thanatophobia, but we cannot banish death (O'Mahony, 2016). Death therefore remains a tacit shadow over all our projects. Whatever we achieve must perish in accord with the entropic principle. Even as I write this chapter, for example, probably within a decade or two of years I will be dead and forgotten, this book will probably be relatively little read, and evolutionary psychology itself may well be passing into the dusty archives of intellectual history within decades. We are, said Hamlet, 'quintessence of dust'. This is not a cynical view or a pessimistic one in the sense of being unrealistic, but an undeniable view (Feltham, 2016). We can certainly argue that it is unhealthy to dwell overmuch on death. But since our ancestors solved the problem of acute death awareness through religious mythologies, in our own time of accelerating knowledge and decreasing mythological defences we may find it harder to avoid death thoughts. It is unsurprising that depression and suicide are increasing worldwide but we seem to reflect relatively little on the vote of no confidence in human existence this represents.

Even the most positive of evolutionary psychologists concede that pessimism has some useful role in human survival, with the usual argument being that pessimistic vigilance and preparedness for things going wrong served ancestral fitness (Leahy, 2002; Nesse, 2000). Prophets of doom have warned against the dangers of always expecting the best. We would, however, expect pessimism to remain a minority disposition, and that is probably the case. Insofar as pessimism is entwined with introversion, depression, and some withdrawal from energetic life projects, it does not commend itself. But regardless of the size of its fan base, pessimism puts forward a negative evaluation that cannot easily be ignored. In line with pessimistic philosophers and other commentators such as Arthur Schopenhauer, Edgar Saltus, E.M. Cioran, Peter Wessel Zapffe, John Gray, David Benatar, Thomas Ligotti, and Ray Brassier (see Feltham, 2016), we can reinforce the analysis that the evolution of distinctive human consciousness was a problematic occurrence that was sure to lead to great and irreversible suffering. In other words, it is not only the future that is incidentally tainted with dark probabilities but the evolutionary past and intrinsic entropy that determine the ultimate fate of humanity. William Golding's (1955) novel *The Inheritors* depicts in highly imaginative terms the deadly encounter between Neanderthals and *Homo sapiens*, in which our own inventiveness is coupled with cruelty, and consciousness of guilt.

It should be noted, however, that some who agree with a deeply negative analysis of human behaviour do not come to pessimistic conclusions about our future prospects. The *dukkha* of Buddhism and sin of Christianity (interestingly similar in their etymological roots of de-centred, or off the mark, respectively) hold out hope of salvation by enlightenment via meditation and by the 'good news' of Christ. A pessimistic anthropathological take on Buddhism, however, is that even supposing the Buddha fortuitously understood and transcended suffering himself approximately 2500 years ago (*supposing*, because we can never know), vanishingly few appear to

have been able to follow his example since. Jeremy Griffith (2004) too, who fully acknowledges the horrors of the present human condition, believes optimistically that we now stand at the threshold of a radically new freedom. Paul Gilbert (1989, 2014) has based his understanding of compassion-focused self-help and therapy on the informed recognition that the human 'tricky brain' has been a 'complete mess' for two million years, which resonates with Kurzban's (2011) 'fragmented brain'. Somewhat similarly, Anthony Stevens (1993) believes that the 'two million-year-old self' can be accessed by Jungian archetypal psychiatry to effect 21st century therapeutic change. The earth scientist and inventor James Lovelock (2014), while expecting climate change to decimate humanity in the decades to come, believes enough will survive to engineer our species' important future role in cosmic self-consciousness and exploration. Some who endorse evolutionary psychological views as to deep causes nevertheless see hope of steady progress in evidence of declining violence, a major ingredient of anthropathology (Pinker, 2012).

Hope springs eternal. It is quite possible to base one's view of the human future on the heartwarming statistics of Steven Pinker's *Better Angels* and the inspirational lyrics of John Lennon's *Imagine*. Or indeed on the therapy-for-all, anti-capitalist, and re-enchantment agendas of assorted right-hemisphere enthusiasts.

Cognate Disciplines, Discarded Hypotheses, and Epistemological Problems

I have already mentioned some relevant academic disciplines and sub-disciplines in passing: the theology of the Fall and sin; Buddhist philosophy; big history; palaeobiology; evolutionary psychology. These by no means concur on the kind of anthropathology thesis I put forward here. There is sometimes a line between cranks and legitimate academics that is hazy, and it would be difficult to judge where psychoanalysis, for example, sits. Disagreement on many salient matters is rife among academics. Scientific research is always incomplete and many traces of human origins may never be found. Hasty conclusions may be avoided but we tend to fill in the gaps in our knowledge with interpretations based on bias. Each of us brings our personality biases (indeed our 'epistemologically different worlds') to such ventures; the nature of our chosen discipline and its perspectives influence our conclusions; our partly unconscious and usually undeclared politics often determine our interpretations of data; and finally we may be susceptible to denial. The pessimistic taste for negative evaluations of the human condition is a minority one.

There is, however, a curious paradox here. Among texts and authors supporting an anthropathological view, implicitly or explicitly, we have the following, many already mentioned: Bohm (1994), Burns (2007), Diamond (2011), Ehrlich and Ehrlich (2004), Gray (2002), Smith (2012), Varki and Brower (2013), Szent-Györgyi (1970), Taylor (2010), Zapffe (1933/2004), and Zerzan (2002). I raise this diverse spread of contributors here, both to remark on their different disciplines and to

question why in the light of such contributions we still have nothing approaching consilience. The paradox is that a vital topic addressed so passionately by writers like these should remain so uncoordinated. In part it is due to their differences of view, in part to writing at different times and places. But it is probably also due to academic pressures for specialisation and to an assumption that no convergent focus is of sufficient interest.

Among Western academics and the liberal public, mythologies of a supernatural creator and human Fall have largely given way to an alliance with scientific method and technological progress as overcoming deficiencies. Religious, paranormal, and transpersonal aetiologies and remedies may be fading fast[6] but academia is infested with mythologies, hypotheses-in-limbo, and premature triumphs of its own. What, we might ask, is the current status of theories of panspermia or the infinite universe? Theories of classical behaviourism, sociobiology, the aquatic ape, the bicameral mind, and others have either been discarded or severely criticised and modified. New disciplines such as deep history, biohistory, and biosociology emerge and await evaluation.

One still current hypothesis that enjoys interest is the correlation in evolution between increased brain size and cooking (Wrangham, 2010). Yet as Cornélio, de Bittencourt-Navarrete, de Bittencourt Brum, Queiroz, and Costa (2016) demonstrate, there are good reasons for doubting this hypothesis, based on mathematical modelling. In certain cases, such as archaeology, questions are raised as to whether the discipline has sufficient theory, or clarity on its use of theory, beyond its 'stones and bones' remit, concerns about dating, and interpretation of data (Johnson, 2006). Anthropology has been remarked upon as suffering from the 'lonely anthropologist' problem but also from an implicit political bias which has sought to aggressively downplay reports of violence, vengeance, and the abduction of women among 'noble savages' (Chagnon, 2013).

Recalling Zapffe's notion of surplus consciousness (not to mention the Buddhist concept of *dukkha*, which includes common desire for reality to be different from the way it is and frustration at not being able to get what one wants), we can posit the idea that it is not only ordinary undisciplined humans who indulge in wasteful, delusional, and self-harming thinking, but also academia collectively, and in particular the social sciences, psychology, and the arts and humanities. The STEM subjects (science, technology, engineering, and mathematics) have their problems, but have some reasonable built-in safeguards against protracted error. This topic is, however, a minefield of nuances and polemics (McGilchrist, 2009; Radnitzky & Bartley, 1987) and academia probably reflects the confusion and complexity of modernity.[7]

Parallel to language and tribalism, we have a babel of academic disciplines and specialisms. Research proceeds very slowly, with major breakthroughs coming rarely in science and hardly at all in the social sciences. No Darwin-sized figure has appeared since Darwin himself and arguably no Darwin has ever appeared among social scientists. Some of this slow pace is inevitable and necessary but some is simply due to tradition and competition. A problem for anthropathology as a putative discipline is its highly interdisciplinary nature, which calls either for rare

polymathic minds or massive transdisciplinary cooperation that is unlikely ever to materialise. Interestingly, some cited here have been relatively independent scholars (Darwin, Lovelock), thinkers writing outside their own disciplines (Bohm, Szent-Györgyi, Varki and Brower), or polymaths (Diamond).

Anthropathology and Evolutionary Psychology

'The long-term scientific goal toward which evolutionary psychologists are working is the mapping of our universal human nature' (Tooby & Cosmides, 2005, p. 5). These authors speak too of a 'natural science of humanity'. The same claims can be made for anthropathology, except that here the focus is on the denied, universally dark side of human nature. Anthropathology shares with evoIutionary psychology a refusal to succumb to contemporary pressures from postmodernist writers to eschew the concept of human nature, grand narratives, and other essentialist foci (Ashworth, 2000). But evolutionary psychology tends to favour the concepts of domain-specificity and modularity of mind, and in speculative debates on the architecture of the mind, even 'moderately massively modular' arrangements are considered (Carruthers, 2003). Anthropathology, more monolithic, leans towards a concept of universally distorted cognitive processes now situated in individual, egoic, surplus, mischievous consciousness, as described by Zapffe and Bohm in particular.

Evolutionary psychology prefers to chunk its material for analysis and contains some implicit optimism for social change, whereas anthropathology, like big history, remains focused on the big human picture, which is certainly hard to grasp, and tends towards the view that our negative trajectory is irreversible. Evolutionary psychology refers somewhat vaguely to our human ancestral environment, while anthropathology suspects complex causes going back to animal and physical origins (for example, scarcity, predation, stealth, entropy). The paleoanthropologist Ian Tattersall (2012) rejects both evolutionary psychology accounts of modularity and human universals theories, arguing that our 'brains are makeshift structures, opportunistically assembled' (p.227), and he refers ironically to our 'accidental cognitive prowess' (p.229) in an overall scheme of evolutionary experimentation.

In some ways, anthropathology resembles terror management theory in its partial focus on one overwhelming negative—death, but anthropathology both seeks historical origins and suspects future futility. As is well known, antagonism exists between evolutionary psychology and terror management theory, which continues in spite of bids for rapprochement (Landau, Solomon, Pyszczynski, & Greenberg, 2007). But while proponents of terror management theory and evolutionary psychology may compete with each other on some points, in anthropathological terms all such conflicts suggest an underlying tribalism. It is difficult to express this without seeming abrasive and rude towards academic colleagues (especially one's editorial hosts), but a core of scepticism running through anthropathological investigation demands a high level of truthfulness. But I, too, am in epistemological competition here. As Trivers (2013) argues, deception and self-deception are pervasive and

'self-deception deforms disciplines' (p.319). Or, to borrow from Bohm's (1994) concept of (dysfunctional) thought about (dysfunctional) thought, we do not recognise when our erudition deceives us.

Evolutionary science focuses on human development in anatomy, neurology, and behaviour, and it sometimes does so in uncritically celebratory or positive terms. Dunbar (2014), for example, in asking what evolutionary science must explain, says, 'The substantive difference [between us and animals] lies in what we can do inside our minds' (p.17). But the substantive difference surely also lies in our habitual over-thinking, deception, and destructive behaviour, in what extensive harm we do outside our minds that we do not even notice ourselves doing, and that we apparently cannot control. Specialist scholarly research can appear to be isolated from or indifferent to the anthropogenic havoc all around us, or perhaps regard it as political terrain that is not relevant to human evolution. This is, however, an ongoing thorny problem of pure versus applied research (McIntyre, 2006).

Insofar as evolutionary psychology and anthropathology share some deterministic and pessimistic features, they are disliked and critiqued by optimistic, Enlightenment-embracing writers such as Tallis (1999, 2011). In general, those enamoured of optimistic, determinism-denying attitudes, such as left-wing political thinkers (Clark, 2002), feminist writers, and many philosophers (e.g. O'Hear, 1999), often dislike and reject evolutionary psychology, neurophilosophy, and similar disciplines for suggesting that our behaviour is significantly limited by our deep past. Radcliffe Richards (2000) applies philosophical analysis to the typically superficial reading of a 'selfish genes and moral animals' polarity. Tattersall (2012) puts his view of evolutionary psychology thus: 'This view has a wonderful reductionist appeal; but in reality our brains are the ultimate general-purpose organs, not adapted 'for' anything at all' (p. 228). Hagen (2005) and Buss (2014) answer some of these criticisms. Insofar as anthropathology is deterministic and pessimistic, it falls within similar criticisms.

Does Anthropathology Have a Future?

One can answer this succinctly and provocatively in four ways: yes, no, no, and no.

1. It has a future insofar as it has persisted as a phenomenon for millennia and is unlikely to end while large human populations exist.

2. It probably has no future as a putative academic discipline because (a) it is so repellently negative in its characteristics and impossibly global in its negative scope; and (b) it logically 'incriminates' all humans, including academics, whose affective subjectivity typically defends against it by denying it.

3. It certainly has no *long-term* future insofar as humanity itself ultimately has no long-term future.

4. However, let us suppose that in the short term at least some of us find it compelling enough to investigate, and perhaps even minimally optimistic enough to question whether it might be negated.

The most pessimistic scenario—linking numbers 1 and 3 above—is that our continuing unacknowledged anthropathology takes us forward into one or another catastrophe that exterminates or decimates us (Oreskes & Conway, 2014). Anthropogenic climate change is currently the frontrunner for such disaster scenarios but nuclear war and other possibilities remain on the table. The adverb 'probably' within number 2 allows for a small possibility that anthropathology might receive serious attention, and might be acted upon in a timeframe that could conceivably alter something significantly. Alternatively, a rigorous evaluation of anthropathological claims might just result in its decisive refutation. Number 4, the most optimistic scenario, would demand that we were able to clearly define anthropathology and operationalise its ingredients for meaningful research.

A combination of 2 and 4 would demand some quantifiable and testable phenomena. To some extent, such work has already been accomplished, for example, in tracing prolific warfare historically and counting the dead. History, politics, and economics can tell us about the incidence of genocides, murderous dictatorships, economic inequalities, and so on. Climate science should be able to calculate the effects of and prognosis for anthropogenic climate change. Two further problems present themselves, namely the undeclared political bias of researchers and the difficulty of creating meaningful and rigorous research in laboratories, in the field, or in other special conditions. Evolutionary psychology shares this problem, however. It may be more promising to identify groups of human beings who do not exhibit significant anthropathological behaviour. Here, I am thinking of infants young enough not to have been inducted into anthropathology; people who are neurologically atypical (e.g. those with Down Syndrome or autism, feral innocents); those who by virtue of special conditions (e.g. epilepsy, stroke) sometimes experience unusual mental states; those who claim, or are believed, via meditation or fortuitous 'enlightenment' experiences, to enter alternate or higher states of consciousness; those who ingest mind-altering drugs such as LSD; and those in some cultures who have not suffered from chronic 'nature deficit disorder', dense urban living conditions, and 'everyday trauma' (Epstein, 2014). Clearly this wish list emphasises neurological research, perhaps with some anthropological opportunities for observation too, and novel investigations in experiential groupwork.[8] Research might look for signs of egolessness, inability to lie or low tendencies to lie or dissemble, no or low levels of malice and unnecessary aggression, acceptance of death, absence or low levels of psychological suffering, and so on. Of course, we would run into some difficult choices, such as deciding whether religious beliefs are or are not delusional, the larger question here being 'who decides what is and is not anthropathological?' But in principle much of this research could be conceived and undertaken.

Conclusions

As science advances and mythological and emotive worldviews shrink, as religions recede and rationality strengthens its hold, simultaneously we are experiencing exponential information complexity and overload. This is partly addressed by the scientific study of information as a thermodynamic entity, and maintenance of distinct professional areas of expertise, but is accompanied by growing difficulties in discernment, by 'dumbing down', and by cognitive exhaustion. If we are unfit for high levels of sugar consumption without medical penalties, for example, we may also be susceptible to 'infobesity'. We may find strategies to circumvent such difficulties, or alternatively we may be passing into an evolutionary stage of complexity and disorder from which we cannot recover—civilizational or species-wide collapse. As social science, psychology, and humanities academics, we face difficult choices between generating ever more divergent theory and argument, most of it inconclusive and of little obvious utility; judgements as to the urgency of threatening factors in our environment and our anthropathological trajectory; and the possibility of having to significantly change course. Having evolved to deal with an average expectable environment, and now often depending on our academic vocations for egoic, economic, and status-maintenance purposes, it feels counterintuitive to most of us to believe that a radically different cognitive focus and activity might be called for.

Anthropathology may or may not be a viable hypothesis but its core elements are these. Some combination of factors took *Homo sapiens* out of the animal world into an earth-spanning species with amazing constructive abilities alongside terrible destructiveness. Specifying the detailed historical mechanisms for this is not currently possible and may never be. But it seems likely that pre-existing biological tendencies made it inevitable in the long term. Simultaneously, the physical laws of the universe mean that our increasing complexity must eventually end in extinction. Human creativity stemming from dexterous hand-use and tool-making led to a culture of artificial technology that is all-pervasive and both convivial and life-threatening; and puts us on course for a transhuman future of robotics, artificial intelligence, and space exploration, the consequences of which are currently unknowable. Human sociality in mass societies underpins our progress but also overtaxes us. Our advanced consciousness makes us aware of nature's indifference to our fate, and to inevitable individual and species death. In order to maintain morale, we are obliged to deny much that impinges on our consciousness. We thus remain locked into many social and belief systems that are anachronistic and absurd. Even though violence may be declining, it can never reliably or finally reduce to near zero, and our overall anthropathology drives us onward via greed, restlessness, and maddening symbolism, into continuous suffering.

Anthropathology might be understood concisely in these terms: our being too successful in having overcome natural disasters, food scarcity, predators, and other forms of adversity, and hence becoming over-populated; our concomitant over-reliance on technology, which has compromised us biologically and threatens the

earth; our complex consciousness which has a runaway, self-deceiving, thanatophobic downside; our failures to adapt to unintended consequences in a timely fashion; and also our being towards the senescent end of an entropic arc. In spite of metaphorical linkage with sin and evil, anthropathology is not choice-based but driven by deeply embedded and entangled forces we barely notice, let alone control. Anthropathology is lodged like a concealed deadly bacterium in our neural systems and habits of perception and behaviour such that we cannot recognise or shift it. Yet some of us, deluded or not, claim to recognise it. *If* such a self-deceiving entity as anthropathology—our chronically troublesome species trait—exists, and *if* some claim to know a way to overcome it, such as Jesus, the Buddha, psychoanalysts, their enthusiastic followers, et al., why does it remain largely undetected, misunderstood, unresolved, and troublesome?

Notes

1. Exceptions can always be found. Diamond (1991), for example, examines violence, genocide, sexual problems, addictions, ageing and death, and catastrophe; but seeks no unified explanation. Kaplan and Kaplan (2010) focus on many areas of logical error-making—a sizable field now in its own right—but do so somewhat jocularly and suggest such mistakes may be the 'handmaiden of adaptability'.
2. 'Core sickness' is a tentative descriptor and might be read as 'core moral, psychological (Staub, 2003), or neurological sickness'; or paraphrased as the sum of negative aspects of evolved and current human behaviour.
3. Deep history (Shryock & Smail, 2011) takes nine million years ago as its starting point.
4. As I write, for example, it is speculated that human remains in the Rising Star Cave in South Africa may point to a burial rite associated with *Homo naledi* up to 2 or 3 million years ago (Green, 2016).
5. In Tolstoy's novella *The Death of Ivan Ilych*, and Miller's play *Death of a Salesman*, respectively.
6. Readers will decide for themselves where to place various figures such as Rudolph Steiner, Pierre Teilhard de Chardin, William Irwin Thompson, and Ken Wilber. Wilber (1996), incidentally, speaks of two Falls, the first ('theological Fall') 15 billion years ago, the second ('scientific Fall') about 4000 years ago, when humans became conscious of their illusory separation from original oneness. Hands (2015), presenting a recent ambitious and critical consideration of all sciences concerning origins, nevertheless leans towards some of the above writers with his favoured concept of 'psychic energy'.
7. On complexity and confusion, consider the notion that (messy) modular minds lead to massive inconsistency, hypocrisy, and splintered perceptions (Kurzban, 2011); and then reckon with Seabright's (2010) reminder that we trust and cooperate on a massive scale economically. These may both appear at odds with a

monolithic anthropathology, yet it is the dark undertow of fragile, kluge-like functionality that unites them.

8. While much psychological groupwork (such as encounter groups) aims at enhancing human potential, group analysis focuses somewhat on psychopathology, and existential group therapy commonly considers problems of isolation, meaninglessness, death, and freedom. However, all these are implicitly directed towards solutions, and a rigorously investigative anthropathology group would have to allow for the possibility of there being no solutions.

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