

Anxiety and Posttraumatic Stress Disorder in the Context of Human Brain Evolution: A Role for Theory in *DSM-V*?

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The “hypervigilance, escape, struggle, tonic immobility” evolutionarily hardwired acute peritraumatic response sequence is important for clinicians to understand. Our commentary supplements the useful article on human tonic immobility (TI) by Marx, Forsyth, Gallup, Fusé,

and Lexington (2008). A hallmark sign of TI is peritraumatic tachycardia, which others have documented as a major risk factor for subsequent posttraumatic stress disorder (PTSD). TI is evolutionarily highly conserved (uniform across species) and underscores the need for *DSM-V* planners to consider the inclusion of evolution theory in the reconceptualization of anxiety and PTSD. We discuss the relevance of evolution theory to the *DSM-V* reconceptualization of acute dissociative-conversion symptoms and of epidemic sociogenic disorder (epidemic “hysteria”). Both are especially in need of attention in light of the increasing threat of terrorism against civilians. We provide other pertinent examples. Finally, evolution theory is not ideology driven (and makes testable predictions regarding etiology in “both directions”). For instance, it predicted the unexpected finding that some disorders conceptualized in *DSM-IV-TR* as innate phobias are conditioned responses and thus better conceptualized as mild forms of PTSD. Evolution theory may offer a conceptual framework in *DSM-V* both for treatment and for research on psychopathology.

Key words: anxiety concept, *DSM-V*, evolution, PTSD, tonic immobility. [*Clin Psychol Sci Prac* 15: 91–97, 2008]

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In this commentary, we will interchangeably use “innate,” “hardwired,” “evolutionarily conserved,” “evolved,” and “prepotentiated” fears for the nonassociative mode-of-acquisition-based fears traditionally termed “unconditioned,” “primary,” “instinctual,” “prepared,” “ancestral,” “anachronistic,” “primeval,” or “atavistic.” Additionally, we will mostly use the neurobiological research-based etiological terms “memory-trace-overconsolidation-based disorders,” “overconsolidated-fear disorders,” or “overconsolidational anxiety disorders” (Bracha, 2006; Pitman & Delahanty, 2005). These are newly proposed terms for the associative-mode-of-acquisition-based, PTSD-like disorders traditionally termed “secondary,” “learned,” “conditioned,” “posttraumatic,” or “acquired.”

INTRODUCTION

The published research agenda (Kupfer, First, & Regier, 2002) for the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V)* advocated the “development of a physiologically based classification system in the *DSM-V*” (Charney et al., 2002). Psychophysiological research on PTSD is expanding at all levels of inquiry;

for example, neuron counting methods (adopted from Parkinsonism research) have shown damage to the locus ceruleus in chronic PTSD (Bracha, Garcia-Rill, Mrazek, & Skinner, 2005). The *DSM-III*, *DSM-IV*, *DSM-IV-TR* (American Psychiatric Association, 1980, 1994, 2004), and *ICD-10* (World Health Organization, 2004) have judiciously minimized discussion of etiologies to distance clinical psychology and psychiatry from Freudian psychoanalysis. This goal has been largely achieved, and several authors now argue that sufficient empirical evidence has accumulated to re-introduce etiological factors into *DSM-V* (Akiskal & Akiskal, 2005; Bracha, 2006; Bracha, Ralston, et al., 2005, 2007; Cosmides & Tooby, 1999; Nesse, Stearns, & Omenn, 2006; Tooby & Cosmides, 1990). A related problem in *DSM-IV-TR* and *ICD-10* is that the classification of anxiety disorders is neither mode-of-acquisition-based nor brain-evolution-based (Bracha, 2006).

FREEZE, FLIGHT, FIGHT, FRIGHT

Discussing the “Hypervigilance, Escape, Struggle, Tonic-Immobility” sequence, Bracha et al. (2004) asked the question, “Does ‘fight or flight’ need updating?” Because the phrase “Hypervigilance, Escape, Struggle, Tonic Immobility” may be difficult to remember, the authors coined the catchphrase “Freeze, Flight, Fight, Fright” (the four Fs of mammalian fear). Tonic immobility (TI) in humans is essentially what European psychiatrists call “fright” or “effroi” (Bracha et al., 2004). The resemblance of TI to the fear behavior of other animals is striking. For example, a well-documented component of TI in the opossum and some other animals is loss of bowel control. A full 3% of acutely traumatized humans of both sexes also admit to having experienced this sign of extreme fear (Bracha et al., 2003).

The motor rigidity seen in TI superficially resembles rigor mortis as well as “decorticate rigidity” or “decorticate spasticity,” which is a hallmark sign of diffuse injury to the cortex. The neurobiology of this similarity is outside the scope of our commentary; however, it is one reason for the alarm that TI often produces in the emergency room setting. TI manifests as “waxy” flexibility and one tragic consequence for combat veterans has been misdiagnosis as psychotic catatonia. Consequently, veterans with severe combat-related PTSD may have received a less appropriate treatment. The often reported resolution

of the catatonic form of TI following Amytal interview or intravenous administration of a single dose of benzodiazepine is consistent with the above view of tonic immobility. Some catatonic-like states presenting in the emergency room are, in fact, a misdiagnosed, fear-triggered tonic immobility, rather than a psychosis-based mannerism. Benzodiazepines and sodium amytal will not treat a psychosis-based condition, but are clinically useful as an emergency measure for tonic immobility.

The comprehensive review article by Marx, Forsyth, Gallup, Fusé, and Lexington (2008) on the TI stage of the four Fs of mammalian fear sequence provides lucid clarification of clinically relevant human behavior in the context of an evolved predator defense. TI is a neglected but extremely important concept and should be discussed as a peritraumatic risk factor in the PTSD section of *DSM-V/ICD-11*. TI might even be considered for inclusion in the conceptualization of Criterion-A experiences of PTSD in *DSM-V/ICD-11*. Extreme tachycardia is always a part of TI, and during and immediately after traumatic events, it is a major treatable risk factor for PTSD (Pitman et al., 2002; Pitman & Delahanty, 2005; Vaiva et al., 2000, 2003a, 2003b). Unfortunately, clinicians tend to conflate the tachycardic “fright” (associated with TI), which *follows* a failed attempt to flee or fight, with the bradycardic “freezing” orienting response that *precedes* the flight, fight, fright sequence, or with the bradycardic flaccid immobility that sometimes follows TI and presents as psychogenic fainting or acute dissociative–conversive symptoms (Bracha, 2004; Bracha et al., 2004).

EXAMPLES OF THE RELEVANCE OF EVOLUTIONARY BIOLOGY TO TAXONOMIC CLASSIFICATION IN THE DSM-V PTSD-Like Agoraphobia and Innate Agoraphobia Should Have Separate Taxonomic Classification in DSM-V/ICD-11

Using evolutionary theory, *DSM-V* and *ICD-11* may be able to separately retaxonomize both the (associative-mode-of-acquisition-based) PTSD-like postpanic attack agoraphobia and prepotentiated innate agoraphobia. Following work by Nesse (Nesse, 1997; Nesse et al., 2006), Bracha (Bracha, 2006; Bracha et al., 2006a, 2006d) proposed that both agoraphobia subtypes could be taken out of the panic disorder section in *DSM-V* and *ICD-11*. Prepotentiated innate agoraphobia (i.e., with no prior Criterion-A traumatic event) could be categorized

with the other innate specific phobias. In contrast, conditioned, posttraumatic, memory-trace-overconsolidation-based agoraphobia could be categorized alongside (a more narrowly defined) PTSD in a new grouping of anxiety/stress disorders titled “Overconsolidated-Fear Disorders” or “Overconsolidational Anxiety Disorders.”

Contrary to dogma, a panic attack away from home is not the only Criterion-A event which (if untreated) is often followed by secondary (memory-trace-overconsolidation-based) conditioned agoraphobia. Experiences away from home, often followed by secondary agoraphobia, include psychiatric and nonpsychiatric medical conditions such as chronic motor or vocal tic disorder, trichotillomania, narcolepsy, and grand-mal seizures. Criterion-A experiences such as being bullied, threatened, or physically assaulted by school or neighborhood peers are underresearched events (Bracha et al., 2003). These particular traumas are also often followed by conditioning-based PTSD-like agoraphobia.

RETAXONOMIZE SOME ANIMAL PHOBIAS WITH PTSD IN DSM-V

Anxiety disorder classification in the official diagnostic manuals is based neither on mode-of-acquisition nor on brain evolution. One example is the clustering together by *DSM-IV-TR* and *ICD-10* of snake phobia (which has been shown experimentally to be innate; Cook & Mineka, 1989) with the PTSD-like (overconsolidational) dog phobia (which has been shown to be a PTSD-like conditioned fear; King et al., 1997). In light of 15,000 years of symbiotic human/canine co-evolution, and the long dependence by humans on dogs for hunting and protection, evolutionary reasoning predicts that fear of dogs has an associative mode of acquisition (Bracha, 2006; Bracha et al., 2006a, 2006d). The anecdotally reported improved sleep achieved by having a dog share an anxious patient’s bed also suggests that an “anxiolytic effect of dogs” is innate and that fear of dogs develops from overconsolidation. Further support comes from a study of 30 children with dog phobia. More than 86% of parents “were able to attribute their child’s phobia to one of Rachman’s three (noninnate) conditioning pathways: direct conditioning, modeling, or transmission of information” (King et al., 1997). Elsewhere, the empirical evidence demonstrating the same argument for bird and bat phobia (which also needs to be retaxonomized in

DSM-V alongside PTSD) has been reviewed (Bracha, 2006).

EVOLUTIONARY BIOLOGY AND THE THEORETICAL RECONCEPTUALIZATION OF DSM-V

In a series of articles, Akiskal applied evolutionary theory to bipolar-II disorder (Akiskal & Akiskal, 2005; Niculescu & Akiskal, 2001), and Nesse applied evolutionary theory to numerous other psychopathologies (Marks & Nesse, 1997; Nesse, 1984, 1999a, 1999b, 2001; Nesse & Young, 2000; Nesse et al., 2006). Specific phobias are classic examples of how evolutionary theory can apply to common anxiety disorders (Cook & Mineka, 1989, 1990; Gibson & Walk, 1960; Poulton et al., 1998; Poulton & Menzies, 2002). An excellent example is social phobia (i.e., fear of scrutiny by strangers; Stein, 1998; Stein & Bouwer, 1997). During much of the human environment of evolutionary adaptedness (EEA), being stared at by a large group of nonsmiling, nonkin conspecifics was more likely than not to be followed by negative consequences. Evolution is not forward looking and could not anticipate a future where being stared at by nonsmiling strangers may be followed by applause and enhanced reputation rather than injury or death (see Bracha, 2006). Consider some recent contributions of evolutionary theory relevant to the *DSM-V* research agenda, with the suggestion that the *DSM-V* designers reconsider the near total exclusion of theory found in successive editions from *DSM-III* on.

Blood-Injection-Injury-Type Phobia Should Be Reconceptualized as a Survival-Enhancing Ancestral Fear Response

Bracha et al. reviewed literature suggesting that blood-injection-injury phobia is a conserved fitness-enhancing ancestral fear response (Bracha, 2004; Bracha, Bienvenu, & Person, 2006; Bracha et al., 2005c). During prehistoric warfare, an encounter with “a stranger holding a sharp object” was consistently associated with threat to life. A heritable hardwired or prepotentiated disposition to abruptly faint rather than freeze or attempt to flee or fight in response to an approaching sharp object, a minor injury, or the sight of blood may have evolved as an alternative acute stress-induced anxiety response. Such a reaction in response to these stimuli may have increased some noncombatants’ chances of survival. This possibility provides an explanation for the unusual age

and sex pattern of fear-induced fainting (Bienvenu & Eaton, 1998; Bracha et al., 2006a).

Acute Conversive Symptoms Should Be Reconceptualized as Survival-Enhancing Ancestral Fear Responses

There is little discussion of evolutionary etiologies as applied to conversive symptoms: particular acute pseudoneurological sociogenic symptoms such as psychogenic nonepileptic attacks (pseudoseizures), the conversive motor deficits “pseudo-paralysis and pseudocerebellar symptoms,” and psychogenic blindness. There is a growing need to understand conversive symptoms resulting from terrorism against civilians as well as military personnel in a war zone. These perplexing pseudoneuro-logical stress-triggered symptoms, which constitute psychopathology in extant humans, may be traceable to the ancestral EEA (Bracha et al., 2005b). During ancestral combat, conversive symptoms (not unlike blood-injection-injury-type phobia) may have increased the survival odds for some noncombatants by visually (i.e., “nonverbally”) signaling to predatory conspecifics that one does not present a danger (Bracha, Yoshioka, et al., 2005b). This theory is consistent with the age and sex pattern of conversive disorders, and with nonverbal signals of submission used by many other species.

Although acute pseudoneurological symptoms are not part of the pan-mammalian hardwired fear response sequence (freeze, flight, fight, fright; Bracha, 2004; Bracha et al., 2006), certain pseudoneurological acute anxiety responses were selected into the human genome following nearly continuous warfare during the ancestral EEA. Fear-induced acute pseudoneurological symptoms were most likely non-fitness-enhancing for postpubertal males (ancestral war-fighters) resulting either in death, severe injury, or, at minimum, a drop in mate availability or mate choice hierarchy. In contrast, for reproductive-age women and for weaned prepubertal individuals, fear-induced acute pseudoneurological symptoms may have been fitness-enhancing, because they increased the likelihood of preserving life and passing on one’s genes to the next generation.

Epidemic Sociogenic Disorder (Epidemic “Hysteria”) Should Be Reconceptualized as a Survival-Enhancing Ancestral Fear Response

Epidemic sociogenic disorder (epidemic “hysteria”) may be especially ripe for an evolutionary conceptualization.

During prehistoric intertribal combat, seeing a peer drop to the ground, appearing severely injured, was a nonverbal signal to others in the group that there was cause for alarm (Bracha, Yoshioka, et al., 2005). A predisposition to faint in response to fainting of a member of one’s peer group may have increased the odds of survival. Epidemic faintness in modern clinical settings may be a manifestation of this prepotentiated ancestral alarm-signaling response, now encoded in some genomes.

CONTRIBUTIONS OF EVOLUTIONARY BIOLOGY TO THE “ASSOCIATED PHYSICAL EXAMINATION FINDINGS” SECTIONS IN DSM-V

In addition to shifting the focus from categorical disorders to broader categories and dimensions future editions of the *DSM* may opt to include a larger number of clinically observable signs. Assessment of anxiety symptoms in *DSM-IV-TR* depend almost exclusively on retrospective self-report, yet the psychometric literature has demonstrated retrospective distortions in questionnaires and structured interviews for trait anxiety and recent subjective distress. Both are vulnerable to over- and underreporting. Inclusion of observable, physical signs of anxiety may be a logical way to increase the validity of anxiety diagnoses.

One such sign could be bruxism or teeth grinding. Arguably, during the early human EEA, jaw clenching was adaptive because it strengthened the masseter and temporalis muscles, enabling a stronger, deeper, and therefore more lethal bite in warfare with conspecifics. Similarly, sharper incisors produced by teeth grinding may have served as weaponry during early human combat.

Extreme nonconscious jaw clenching is a neglected aspect of TI in humans (Bracha, Person, et al., 2005; Bracha, Ralston, et al., 2005a; Bracha et al., 2006b; Bracha, Williams, Person, et al., 2004). It may be partially responsible for the often reported inability of victims of motor vehicle accidents and of sexual assault to adaptively vocalize. Often, the only evidence for this peritraumatic, tonic jaw clenching is when the patient reports bruxism or temporal mandibular tenderness a few days after the TI experience (Bracha, 2006; Bracha et al., 2006b; Bracha, Williams, Person, et al., 2004; Bracha, Yoshioka, et al., 2005).

One diagnostic criterion of PTSD that is likely to undergo a revision in *DSM-V* is Criterion-D (persistent

fear-circuitry activation not present before the trauma). Research toward *DSM-V* should examine the possible utility of incorporating observable new-onset bruxism and jaw-clenching-induced facial pain as a clinical sign of PTSD Criterion-D.

OVERCOMING IDEOLOGICAL OBJECTIONS TO ETIOLOGICAL RECONCEPTUALIZATION IN THE *DSM-V*

Ideological objections to any mention of evolutionary etiological factors for many of the disorders in *DSM-V* are to be expected. However, such objections are difficult to sustain for anxiety disorders. It is axiomatic that a mild degree of fear is often helpful, and that one can have “too much of a good thing.” Therefore, a prudent strategy in the research agenda for *DSM-V* may be to focus on evolutionary factors in the anxiety spectrum. A second prudent approach is to incorporate evolutionary theory into the etiology of the dissociative conversion disorders. The non-evidence-based, untestable and unfalsifiable psychoanalytic etiological conceptualization of conversion disorders has been retained in the *DSM-IV-TR*. Replacing it with an evolutionary conceptualization should not meet strong resistance from empiricists, because evolutionary hypotheses are much more amenable to objective, controlled, and replicable research (Bracha et al., 2006a).

SUMMARY

With regard to anxiety disorders and PTSD, there is ample evidence for neurobiological underpinnings (Charney, Barlow, et al., 2002; Bracha, Garcia-Rill, et al., 2005); however, linking anxiety disorders to their evolutionary origins has had limited success in attracting the mainstream interests of mental health clinicians, Marx et al. (2008) being a rare exception. For example, Seligman’s preparedness theory attempted to do this for specific phobias (Mineka & Öhman, 2002a, 2002b; Öhman & Mineka, 2001; Seligman, 1971). Bracha and colleagues have focused on PTSD, other anxiety disorders, and acute stress-induced conversive disorders (Bracha, 2006; Bracha, Williams, Haynes, et al., 2004, 2006d; Bracha & Hayashi, 2006; Bracha, Vega, & Vega, 2006; Bracha, Yoshioka, et al., 2005). Marx et al. comprehensively explain a previously misunderstood motor behavior, reported by victims of sexual assault, as an evolved predator defense. In so doing they eloquently highlight the clinical insights that evolution theory brings to clinical traumatology.

Although *DSM* revisions have moved toward empirical findings, there is still no mention of linkage between psychopathology and evolution. *DSM-III* was a paradigm shift in psychiatry and clinical psychology (Klerman, 1990; Maser et al., in press), but it continues to lack an overarching and unifying theory within which its symptoms and etiologies can be understood. While evolution theory has brought considerable clarity and unification to other specialties in biology and even to internal medicine (Nesse et al., 2006), it has not done so for psychopathology. Nesse’s editorial stressed the need for an evolutionary approach to psychopathology (Nesse et al., 2006), and Bracha has made similar arguments for a host of anxiety disorders. There may now be enough data in certain areas that the inclusion of theory should be reconsidered for *DSM-V*. The theory does not have to be perfectly correct, if it serves to stimulate research. Anxiety and its disorders could easily be a testing ground for inclusion of evolution theory in *DSM-V*, since those forms of psychopathology have the most empirical data. Along with the many changes that are being suggested for *DSM-V*, we urge the planners to seek out empirical studies and/or theories that place psychopathology in an evolutionary context. The field will then have a connection to broader issues in biology, the data on psychopathology can be placed within a widely accepted concept, and clinicians will have the possibility of developing more effective behavioral treatments (e.g., Levine, 1997).

ACKNOWLEDGMENTS

This material is based on work supported in part by the Office of Research and Development, Medical Research Service, Department of Veterans Affairs (VA), VA Pacific Islands Health Care System, Spark M. Matsunaga Medical Center. Support was also provided by a National Alliance for Research on Schizophrenia and Depression Independent Investigator Award, and the VA National Center for PTSD. The authors thank Auke Tellegen, PhD, for helpful comments on an earlier draft of this manuscript, as well as Caitlin E. Macy, Elise Freeman, and Stacey E. Lenze for editorial assistance.

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Received January 15, 2007; accepted January 17, 2007.