

Social conflict models: Can they inform us about human psychopathology?

Kim L. Huhman*

Department of Psychology, P.O. Box 3966, Center for Behavioral Neuroscience, Georgia State University, Atlanta, GA 30302-3966, USA

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Abstract

Social conflict models have been proposed as a powerful way to investigate basic questions of how brain and behavior are altered by social experience. Social defeat, in particular, appears to be a major stressor for most species, and in humans, this stressor is thought to play an important role in the onset of a variety of psychiatric disorders including depression and post-traumatic stress disorder. Aggressive experience, on the other hand, may promote disorders involving inappropriate aggression and violence. Current research using animal models of social conflict involves multiple levels of analysis from genetic and molecular to systems and overt behavior. This review briefly examines a variety of these animal models of social conflict in order to assess whether they are useful for advancing our understanding of how experience can shape brain and behavior and for translating this information so that we have the potential to improve the quality of life of individuals with mental illness and behavioral disorders.

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While the purpose of most basic behavioral neuroscience research is to understand how the brain functions to control complex behavior, this research is often driven by an underlying interest in generating knowledge that may improve human health and welfare. Because direct manipulations cannot usually be performed in humans, scientists rely on animal models to discover the principles that explain the relationship between brain and behavior. Traditionally, this basic research was seen as having intrinsic value and as contributing importantly to our base of scientific knowledge, but recently there has been a growing view that scientists should prioritize basic research that can be “translated” to humans in such a way as to promote well-being. The purpose of this brief review is to examine basic research that employs a variety of social conflict models with particular emphasis on whether this research appears to have translational value.

Social conflict

Agonism or agonistic behavior refers to behaviors that are observed in conflict situations. This term therefore refers to a wide range of aggressive as well as submissive and defensive behaviors, and a variety of experimental approaches have been used to attempt to understand the antecedents, physiological concomitants, and long-term effects of these behaviors. Whether one is a “winner” or a “loser” in an agonistic encounter can be of critical importance in resource allocation and in determination of social rank in humans and non-humans. Winning and losing can also strongly, and differentially, affect both the physiology and the subsequent behavior of participants in agonistic encounters.

Particular interest has been paid in the scientific literature to “losers” and to the ensuing social stress that is selectively experienced by subordinate individuals. One reason for this interest is that social defeat in humans (e.g., exposure to loss in aggressive social encounters or to bullying or abuse) leads to an increase in depression, loneliness, anxiety, social withdrawal, and submissiveness and to a loss of self-esteem (Nemeroff,

* Fax: +1 404 651 3929.

E-mail address: khuhman@gsu.edu.

1998; Agid et al., 2000; Bjorkqvist, 2001; Heim and Nemeroff, 2001; Gardner, 2001). Relatedly, it has been proposed that social conflict models in animals may be useful for studying the physiological concomitants of a number of psychiatric disorders including major depression (Koolhaas et al., 1990; Blanchard et al., 1995; Kudryavtseva and Avgustinovich, 1998; Van Kampen et al., 2002), generalized anxiety and post-traumatic stress disorder (Blanchard et al., 2001a; Bremner, 2004), drug abuse (Kabbaj et al., 2004), aggressive psychopathologies (Blanchard and Blanchard, 2003), and even schizophrenia (Selten and Cantor-Graae, 2005) and eating disorders (Troop et al., 2003). Investigation of social defeat models of human psychopathology is relatively new, and while these models are intuitively appealing, their validation in some cases is in its early stages.

Social defeat and depression

Major depression affects as many as one in five people in the U.S. during their lifetime (Angst, 1992; Kessler et al., 1994), and the economic burden of this disorder, including medical, suicide-related mortality, and workplace costs, was estimated at over 83 billion dollars in 2000 (Greenberg et al., 2003). The pervasive nature of this illness with its characteristic alterations in mood, basic drives, and cognition begs the question as to how or why this disorder occurs in humans. One interesting perspective is that the brain and depressive behavior have evolved so that individuals have a mechanism whereby they can accept social defeat and low social rank (Gilbert and Allan, 1998; Rohde, 2001; Gilbert et al., 2002). If an individual learns that it does not have the ability to become dominant in a particular situation, that individual benefits from a mechanism either to keep it from acting aggressively until such a time when it might be more competitive or to allow it to accommodate its subordinate status indefinitely. The Social Competition Hypothesis of Depression, for example, proposes that animals have genetically determined losing strategies that allow them to respond to social defeat appropriately (Sloman et al., 1994; Price et al., 1994). Behaviors supporting acceptance of low social rank would then serve to protect subordinates from injury or death that could occur if these individuals continue to act in an aggressive manner. Similarly, social rank theories of psychopathology (Gilbert and Allan, 1998; Gilbert et al., 2002) suggest that the existence of social hierarchies require the development of mechanisms that are attuned to winning or losing and that individuals who lose are particularly susceptible to pathology. These theories then provide an evolutionary explanation for the existence of behaviors that are thought of as “depressive” (e.g., anhedonia, social withdrawal, low self-esteem) and serve to emphasize that these behaviors have a physiological basis that can be ascertained and exploited in order to develop better treatment options.

Stressful events are known to exacerbate or lead to the onset of bouts of depression (Post, 1992; Holsboer and Barden, 1996; Kessler, 1997; Kendler et al., 1999; Gold and Chrousos, 2002), and early exposure to social loss appears to be a major

contributor to future depression (Heim and Nemeroff, 2001). Because most of the stressors that are experienced by humans, and other animals for that matter, are social in character (Brown and Prudo, 1981; Kessler, 1997; Bjorkqvist, 2001), ethologically relevant animal models that examine social conflict may be particularly useful for determining how social experience alters the brain and subsequent behavior. The most common social conflict models involving laboratory animals are variations of the resident–intruder model wherein one animal (the intruder) is placed into the home cage of another animal (the resident). The resident, particularly if its body mass is heavier, generally attacks and defeats the intruder.

Exposure to social stress leads to a myriad of long-lasting behavioral and physiological changes, particularly in the defeated opponent, including decreased locomotor activity/exploratory behavior in a variety of tasks (Meerlo et al., 1996a; Koolhaas et al., 1997; Rygula et al., 2005). This change may be indicative of increased anxiety or reduced motivation and may also be analogous to the psychomotor retardation seen in depressed individuals. Relatedly, social stress also leads to changes in circadian rhythmicity (Meerlo et al., 1996a, 2002) and feeding and body weight (Van de Poll et al., 1982; Meerlo et al., 1996b; Bartolomucci et al., 2004; Foster et al., 2006) and, of course, human depression is also associated with disruptions in biological rhythms and consummatory behavior. In addition, defeated animals exhibit increases in heart rate and blood pressure and suppression of immune responses. This constellation of effects is accompanied by a variety of neurochemical changes in the brain including alterations in serotonin, catecholamine, and neuropeptide systems (for excellent reviews, see Koolhaas et al., 1997; Martinez et al., 1998; Blanchard et al., 1998, 2001b). Finally, social stress has also been shown in rodents to reduce preference for a sweet sucrose solution, a change that is thought to mimic anhedonic states in human depression (Von Frijtag et al., 2000, 2002; Rygula et al., 2005). This reduction in sucrose preference can persist for at least 100 days after social defeat and can be reversed with antidepressant treatment (Von Frijtag et al., 2002). Thus, social defeat produces a wide variety of effects that bear a striking resemblance to the symptoms of depression, and many of these effects in animal models can be reversed with treatments (e.g., drugs or controlled sleep deprivation) that have antidepressant effects in humans (Fuchs et al., 1996; Meerlo et al., 1996a; Berton et al., 1999). Many of these defeat-induced changes are persistent which further supports the argument that social conflict models are particularly valuable for exploring the neurobiological basis of human psychiatric disorders.

Social defeat in hamsters

Syrian hamsters are a particularly useful species with which to examine social conflict because they, unlike rats and many other social rodents, readily exhibit agonistic behavior in the laboratory without any provocation or need for complex social housing. In addition, their agonistic behavior is easily categorized and quantified (Grant and Mackintosh, 1962;

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