Ramifications

Volume 2
Issue 1 *Fall 2020*

Article 3

2020

The Human Instinct to Resilience: Clinical and Evolutionary Efficacy of Intrinsically Motivated Stressor Exposure

Brady Barley
West Chester University of Pennsylvania, bb909044@wcupa.edu

Introduction

An individual experiences heightened stress when the demands of their environment exceed their abilities (Lazarus, 1984). Numerous psychiatric disorders are related to pathologically elevated stress, including anxiety disorders (Remes, 2016), major depressive disorder (Nandam et al., 2020), posttraumatic stress disorder (Heim, 2009), and substance use disorders (Lin, 2020). The capacity to endure stress without experiencing pathologically impaired behavioral functioning is known as resilience (Wu et al., 2013). Thus, the potential for the development of a stress-related disorder emerges when an individual encounters environmental stressors that demand more resilience from them than they are capable of manifesting in their current form. Therefore, if they are to survive such stressors without experiencing behavioral impairment, an increase in resilience must occur. The facilitation of this adaptation is the fundamental aim of psychotherapeutic treatments for stress-related disorders.

Voluntary Stressor Exposure and Resilience

Certain behaviors have the efficacious result of increasing an individual's resilience. Treatments for anxiety work by inducing plasticity of an individual's central nervous system that results in increased resilience (Min et al., 2013). Such treatments, known as exposure therapies, require an individual to take voluntary actions that expose themselves to stressors (Grohol, 2016). Animal research has begun to observe that physically identical stressors can elicit different neural responses, depending solely upon whether exposure to the stressor is controllable or uncontrollable. Neural responses to controllable stressors are not associated with the commonly recognized negative effects of stress that would be associated with psychopathology in humans. In rats, uncontrollable stressors can lead to numerous maladaptive behavioral outcomes, including reduced social dominance, neophobia, impaired fear conditioning, immobility, and reduced food and water intake. Maier et al. (2015) observed that activity among serotonergic neurons (HT-5) in the dorsal raphe nucleus (DRN) in response to uncontrollable stressors is causally connected with these maladaptive behavioral outcomes. During controllable exposure to stressors, the DRN receives equal excitatory input as during uncontrollable exposure, but less HT-5 activity, suggesting that controllability mitigates against negative behavioral outcomes by actively inhibiting DRN HT-5. The condition of controllability not only has this effect on the contemporaneous impact of the controlled stressor, but also blocks the same maladaptive behavioral and neurochemical responses to future uncontrollable stressors. This immunizing effect is conserved across environments, and even across physically different stressors. For example, controllable exposure to shocks blocks the maladaptive behavioral and neurochemical impacts of social defeat experienced one week later in a different environment. On the other hand, Maier (2015) explains that exposure to uncontrollable stressors does not attenuate the effects of later uncontrollable stressors, but frequently exaggerates them, indicating

that it is not mere prior exposure, but rather "the experience of control over the stressor" (p. 13) that is responsible for its immunizing effect.

In this study, the rats' initial exposure is involuntary, and behavioral control consists only in the termination, rather than the initiation, of shock. However, it has long been known that sufficiently motivated rats will voluntarily expose themselves to painful electrical shocks in order to move toward an incentive (Olds, 1958). It is arguable that voluntary stressor exposure involves a higher degree of behavioral control than involuntary exposure since, in the case of voluntary exposure, both initiation and termination of the stressor are under the subject's control. Research on another stressor—physical exercise—has found that voluntary exercise also attenuates maladaptive behavioral effects of uncontrollable shocks in rats, but that this same effect is not produced by forced exercise (Greenwood et al., 2012). If this effect of voluntary exposure is mediated by controllability, then it would follow that voluntary stressor exposure is therefore likely to produce resilience-like behavioral outcomes as a consequence of its inherently high degree of control. This is consistent with the clinical literature, demonstrating the high efficacy of exposure therapy for the treatment of anxiety in humans, since the central requirement of exposure therapy is the voluntary exposure of an individual to the source of their anxiety. Given that voluntary exposure reduces maladaptive behaviors associated with anxiety disorders in humans, it is possible that similar neural mechanisms are at work in the cultivation of resilience in humans to those observed to immunize against the maladaptive behavioral effects of stress in animals. In both humans and animals, voluntary, controllable exposure is, by definition, a behavior. The strengthening of resilience through voluntary exposure is therefore an example of an organism's physiology being functionally altered by its own behavior. Thus, voluntary exposure facilitates the change necessary for an individual to endure stressful conditions without experiencing behavioral impairment and subsequent pathology.

Intrinsic Motivation and Evolution

Voluntary exposure need not be introduced artificially in clinical contexts. Evidence exists to suggest that the facilitation of resilience via voluntary exposure is a naturally occurring process that fortifies organisms against threats to optimal behavioral functioning posed by environmental stressors. This view is supported by the observation that humans and other animals display instinctual motivation toward behaviors that serve no apparent direct evolutionary function yet require stressor exposure. Evolutionary hypotheses for behavior attempt to explain a particular behavior by its ability to mitigate the danger imposed by a particular threat to survival, known as a selection pressure. Hypotheses have been proposed for various behaviors in humans and other animals, based primarily upon the way in which behaviors might have enhanced fitness in relation to past selection pressures. However, many behaviors in modern humans have no apparent impact on fitness. Baldassarre et al. (2014) point out that "higher mammals, and especially humans, engage in activities that do not appear to directly serve the goals of survival, reproduction, or material advantage." (p. 1) Based on the observation that "autonomously setting goals and working to acquire new forms of competence

are….examples of activities that often do not confer obvious evolutionary benefit," they go on to conclude that, "activities like these are thus said to be driven by intrinsic motivations." (p. 1). Intrinsic motivations produce behaviors that occur without external reinforcement, and are thus oriented toward the mere performance of a particular behavior without necessary reference to a stimulus. Intrinsic motivations are contrasted with extrinsic motivations, which produce behaviors followed by rewarding external stimuli, and are thus oriented toward behaviors that facilitate access to a particular stimulus. Intrinsic motivations, by definition, are not consequences of associative learning, since they lack external reinforcement. They are likely also not consequences of cognitive learning, since they arise as early as infancy (Schlesinger, 2013), and because some intrinsic motivations, such as exploratory behaviors, are conserved across phylogeny, being present in multiple species (Panskepp, 1998). These properties make it theoretically parsimonious to conceptualize intrinsic motivations as innate. But this idea represents a paradox, given the difficulty of identifying a selection pressure to account for a seemingly innate motivation that can produce behaviors that appear unrelated to survival. Baldassarre et al. (2014) hypothesize that intrinsic motivations may enhance fitness indirectly, by facilitating "the cumulative and virtually open-ended acquisition of knowledge and skills that can later be used to accomplish fitness-enhancing goals." (p. 1) However, this hypothesis does not identify specific selection pressures that can account for an "open-ended" trait. Because behaviors produced by intrinsic motivations seem unrelated to survival or reproduction, the identification of a selection pressure that can account for their natural selection is lacking.

Consider, for example, the motivation among humans to improve at the performance of a certain behavior, known as competence-based intrinsic motivation. This motivation may manifest in the domain of any activity where a gradient of competence is possible. This contrasts with the motivation to perform merely at a level of competence sufficient to produce external reward, which is inherently extrinsic. The distinct motivation to become successively more competent at the performance of a behavior beyond the level where reward ceases to increase is more coherently conceptualized as intrinsic since it persists without increased reward. Motivation to perform at a fixed target level is also more easily attributable to a specific evolutionary function because a behavior need only be performed at a level of competence sufficient to result in enhanced fitness in order to be favored by natural selection. Motivation to become competent beyond the level where a hypothetical evolutionary advantage would cease to increase is thus not easily accounted for by such an advantage. In addition, the investment of resources in the development of unnecessarily high competence may actually decrease fitness, due to wasted resources. Thus, the motivation to improve, not in relation to a target level of competence, but merely *to improve as such,* appears intrinsic, and appears to evade evolutionary explanation. This is consistent with the view that behaviors oriented toward the attainment of competence are intrinsically motivated and are difficult to explain in evolutionary terms due to a lack of direct fitness enhancement (Baldassare et al., 2014).

Intrinsic Motivation and Neuroendocrine Plasticity

The majority of evolutionary hypotheses for behavioral traits locate a behavior's fitness in the way the behavior alters aspects of an organism's environment in its favor. However, behavior can also induce plasticity of an organism's physiology that results in fitness-enhancing behavioral outcomes. Thus, plasticity-inducing behaviors produced by innate motivations may enhance fitness not by virtue of the way that they alter an organism's environment, but by virtue of the way that they alter the organism's own physiology. For example, behaviors that require voluntary stressor exposure may induce functional neuroendocrine plasticity that results in increased stress resilience. Physical exercise elicits physiological stress responses including activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, which is associated with the fight-flight-freeze response (Stanahan et al., 2008). The detrimental effects of chronic stress are attributed in part to elevated glucocorticoid levels caused by prolonged HPA axis activation (Sapolsky, 2003). In mice, voluntary exercise has been observed to alter HPA function such that HPA reactivity is both more rapid and shorter-lasting than it was previously (Hare et al., 2014). This is adaptive in one sense because a blunted HPA response protects against the deleterious effects of sustained glucocorticoid exposure on physical and psychological functioning (Hare et al., 2014). It is adaptive in another sense because a quicker, briefer HPA response may allow greater opportunity for higher-order cognitive systems to generate more effective behavioral strategies in response to situations in which fight-flight-freeze is suboptimal. Both a reduction in stress-related behavioral impairments and the capacity for optimally effective behavioral responses contribute to resilience. Thus, voluntary exercise induces functional neuroendocrine plasticity that results in increased resilience. Given that exercise itself elicits stress responses, the implication is that this resilience is facilitated via voluntary exposure to stress, since the same adaptive effects do not result from involuntary exercise. This may be because voluntary exposure involves greater controllability, although research is still lacking concerning the neural and behavioral effects of voluntary exposure to stressors other than exercise, such as shock. Regardless, if voluntary stressor exposure uniquely alters reactivity of stress response systems in a way that results in increased resilience, then any trait which functions to facilitate voluntary exposure would be capable of mitigating evolutionary selection pressure exerted by environmental stressors. The intrinsic motivation to improve requires voluntary stressor exposure, whether it be stress imposed by risk of failure, by social judgement, or by the difficulty of specific behaviors involved in the process of improvement (Levine et al.,1993). Assuming that stress exposure necessary for improvement becomes more intense as one's competence level increases, then the motivation to continue to improve can be explained by its facilitation of progressively stronger resilience across time, which increases with or without reinforcement. The relationship between voluntary stressor exposure and resilience may thus provide ground for the hypothesis that the intrinsic motivation to improve is a product of natural selection that functions to orient an individual toward behaviors that, if voluntarily engaged, will strengthen them psychologically, and that enhance fitness by mitigating selection pressure exerted by environmental stressors.

It is not unreasonable to hypothesize that intrinsically motivated stressor exposure is genetically transmissible, as similar traits have been shown to be so, including sensation-seeking

(Dickson et al., 2018), novelty-seeking (He et al., 2018), risk-taking (Rao et al., 2018; Heitland et al., 2012), and exploratory locomotion (Zhou et al., 2019). The earliest vertebrates contained the fundamental components of the HPA axis (Denver, 2009), making the system phylogenetically at least 525 million years old (Shu et al., 1999). If natural selection favors a trait for as long as it is capable of adaptively modulating the stress response, and if intrinsic motivation toward stressful behaviors is genetically transmissible, then the motivation to improve may be a very ancient instinct that is species-typical among humans.

Clinical Relevance

The potential for the development of stress-related pathology emerges when an individual encounters a situation whose stressors exceed their current capacity for resilience. Therefore, in order to withstand the stress of the situation without being rendered vulnerable to pathology, an increase in resilience must occur. This adaptation is naturally facilitated by innate motivations oriented toward behaviors that require voluntary stressor exposure, andthat increase resilience. If the active engagement of such motivations inoculates an individual against the risk of psychopathology, then the cause of psychopathology can be conceptualized, in part, as a failure of the individual to engage motivations that, if engaged, would have endowed them with psychological strength sufficient to withstand the stress of their present situation without being behaviorally impaired. This is in contrast to a commonly held supposition that stressful or traumatic events are, themselves, causes of psychiatric disorders, which is a supposition held in contradiction of evidence that posttraumatic stress symptoms are experienced by a minority of traumatized individuals (Bisson et al., 2015; Wu et al., 2013). If humans possess an instinct to resilience, then stress-related disorders can be conceptualized as outcomes of a dysfunction or inhibition of this instinct.

The choice not to engage the instinct to resilience may be fundamentally rooted in ethics, since this instinct requires behaviors which cause an individual extreme discomfort and anxiety, and offers no evidence of their efficacy until after the fact. To put oneself through what one knows will cause oneself emotional pain willingly, without any guarantee of benefit, may even be considered a form of irrational self-harm. The implication is that an overvaluation of ethics and rationality in the interpretation of one's motivations facilitates vulnerability to psychopathology since it allows for the denial of the instinct to behaviors which would have resulted in increased resilience. This raises the question of whether happiness and the absence of pain really ought to be the highest values in psychology, rather than strength or resilience, which are obtained only in the voluntary exposure to distressing and aversive stimuli. Another salient question is whether the cause of psychopathology is really irrationality, ignorance, or a cognitive insufficiency, rather than cowardice in relation to intrinsically motivating, yet anxiety-inducing behaviors, cowardice which itself is encouraged by rational and ethical considerations. But perhaps the most pragmatic question that emerges here is whether the cure for stress-related psychopathology really consists primarily in cognitive alterations, as is assumed by certain forms

of psychotherapy, rather than in action, in the willing engagement of distressing, yet efficacious, instinctual motivations.

Prediction

The hypothesis that intrinsic motivation toward behaviors that require voluntary stressor exposure enhances fitness by mitigating selection pressure exerted by environmental stressors via the facilitation of increased resilience predicts that human or non-human individuals who display higher rates of competence-based intrinsic motivation will experience smaller maladaptive behavioral impacts of naturalistic stressor exposure.

References

Baldassarre, G., Stafford, T., Mirolli, M., Redgrave, P., Ryan, R. M., & Barto, E. (2014). Intrinsic motivations and open-ended development in animals, humans, and robots: an overview. *Frontiers in Psychology*, *5*, Article 985. https://doi.org/10.3389/fpsyg.2014.00985

Bisson, J., Cosgrove, S., Lewis, C., & Robert, N. (2015). Post-traumatic stress disorder. *BMJ: British Medical Journal*, *351*, https://www.jstor.org/stable/26523549

Denver, R. J. (2009), Structural and Functional Evolution of Vertebrate Neuroendocrine Stress Systems. *Annals of the New York Academy of Sciences*, *1163*, 1-16. https://doi.org/10.1111/j.1749-6632.2009.04433.x

Dickson, P. E., Roy, T. A., McNaughton, K. A., Wilcox, T. D., Kumar, P., & Chesler, E. J. (2018). Systems genetics of sensation seeking. *Genes, Brain & Behavior*, *18*(3), 1–12. https://doi.org/10.1111/gbb.12519

Greenwood, B., Loughridge, A. B., Sadaoui, N., Christianson, J. P., & Fleshner, M. (2012), The protective effects of voluntary exercise against the behavioral consequences of uncontrollable stress persist despite an increase in anxiety following forced cessation of exercise. *Behavioural Brain Research*, *233*(2), 314-321. https://doi.org/10.1016/j.bbr.2012.05.017

Grohol, J. M. (2016, May 17). *What is Exposure Therapy?*. Psych Central. Retrieved on April 15, 2020, from https://psychcentral.com/lib/what-is-exposure-therapy/

Hare, B. D., Beierle, J. A., Toufexis, D.J., Hammack, S.E. & Falls, W.A. (2014). ExerciseAssociated Changes in the Corticosterone Response to Acute Restraint Stress: Evidence for Increased Adrenal Sensitivity and Reduced Corticosterone Response Duration. *Neuropsychopharmacology*, *39*(5), 1262-1269. https://doi.org/10.1038/npp.2013.329

He, Y., Martin, N., Zhu, G., & Liu, Y. (2018). Candidate genes for novelty-seeking: A metaanalysis of association studies of DRD4 exon III and COMT Val158Met. *Psychiatric Genetics*, *28*(6), 97–109. https://doi.org/10.1097/YPG.0000000000000209

Heim, C., & Nemeroff, C. B. (2009). Neurobiology of Posttraumatic Stress Disorder. *CNS Spectrums*, *14*(1 Suppl 1), 13–24.

Heitland, I., Oosting, R. S., Baas, J. M. P., Massar, S. A., Kenemans, J. L., & Böcker, K. B. (2012). Genetic polymorphisms of the dopamine and serotonin systems modulate the neurophysiological response to feedback and risk taking in healthy humans. *Cognitive, Affective & Behavioral Neuroscience*, *12*(4), 678–691. https://doi.org/10.3758/s13415-012-0108-8

Lazarus, R.S., Folkman, S. (1984). *Stress, appraisal, and coping*. Springer Publishing Company.

Levine, F. M., Krass, S. M., & Padawer, W. J. (1993). Failure hurts: The effects of stress due to difficult tasks and failure feedback on pain report. *Pain*, *54*(3), 335–340. https://doi.org/10.1016/0304-3959(93)90034-M

Lin, S.-Y., Fried, E. I., & Eaton, N. R. (2020). The association of life stress with substance use symptoms: A network analysis and replication. *Journal of Abnormal Psychology*, *129*(2), 204–214. http://dx.doi.org/10.1037/abn0000485

Maier, S.F. (2015). Behavioral control blunts reactions to contemporaneous and future adverse events: Medial prefrontal cortex plasticity and a corticostriatal network. *Neurobiology of Stress*. *1*, 12-22. https://doi.org/10.1016/j.ynstr.2014.09.003

Min, J.-A., Yu, J. J., Lee, C.-U., Chae, J.-H. (2013). Cognitive emotion regulation strategies contributing to resilience in patients with depression and/or anxiety disorders. *Comprehensive Psychiatry*. *54*(8), 1190–1197. https://doi.org/10.1016/j.comppsych.2013.05.008

Nandam, L. S., Brazel, M., Zhou, M., & Jhaveri, D. J. (2020). Cortisol and major depressive disorder—Translating findings from humans to animal models and back. *Frontiers in Psychiatry*, *10*, Article 974. https://doi.org/10.3389/fpsyt.2019.00974

Olds, J. (1958). Self-stimulation of the brain: Its use to study local effects of hunger, sex, and drugs. *Science, 127*(3294). 315–324. https://doi.org/10.1126/science.127.3294.315

Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions*. Oxford University Press.

Rao, L.-L., Zhou, Y., Zheng, D., Yang, L.-Q., & Li, S. (2018). Genetic Contribution to Variation in Risk Taking: A Functional MRI Twin Study of the Balloon Analogue Risk Task. *Psychological Science*, *29*(10), 1679–1691. https://doi.org/10.1177/0956797618779961

Remes, O., Brayne, C., , R. V. D., & Lafortune, L. (2016). A systematic review of reviews on the prevalence of anxiety disorders in adult populations. *Brain and behavior*, *6*(7), https://doi.org/10.1002/brb3.497

Sapolsky R.M. (2003). Stress and plasticity in the limbic system. *Neurochemical Research* 2*8*(11). 1735–1742. https://doi.org/10.1023/a:1026021307833

Schlesinger, M. (2013). Investigating the origins of intrinsic motivation in human infants. In G. Baldassarre & M. Mirolli (Eds.), *Intrinsically motivated learning in natural and artificial systems* (pp. 367–392). Springer Science + Business Media. https://doi.org/10.1007/978-3-64232375-1_14

Shu, D-G., Luo, H.-L., Morris, S. C., Zhang, X.-L., Hu, S.-X., Chen, L., Han, J., Li, Y., Chen, L.-Z., Zhu, M. (1999). Lower Cambrian vertebrates from south China. *Nature*, *402*(6757): 42–46. https://doi.org/10.1038/46965

Stranahan, A. M., Lee, K., & Mattson, M. P. (2008). Central Mechanisms of HPA Axis Regulation by Voluntary Exercise. *Neuromolecular Medicine, 10*(2), 118-127. https://doi.org/10.1007/s12017-008-8027-0

Wu, G., Feder, A., Cohen, H., Kim, J. J., Calderon, S., Charney, D. S., & Mathé, A. A. (2013). Understanding resilience. *Frontiers in behavioral neuroscience, 7*, Article 10. https://doi.org/10.3389/fnbeh.2013.00010

Zhou, Z., Blandino, P., Yuan, Q., Shen, P.-H., Hodgkinson, C. A., Virkkunen, M., Watson, S. J., Akil, H., & Goldman, D. (2019). Exploratory locomotion, a predictor of addiction vulnerability, is oligogenic in rats selected for this phenotype. *Proceedings of the National Academy of Sciences of the United States of America*, *116*(26), 13107–13115. https://doi.org/10.1073/pnas.1820410116