

Resilience through Sleep

Julie Poulin*, Wun Jung Kim, Anne Germain

University of Pittsburgh Medical Center, Western Psychiatric Institute and Clinic, Pittsburgh USA

The functions of sleep have been an ongoing question for clinicians and researchers interested in the sleeping brain. Historically, sleep was considered as a general restorative process where the brain and the body were mostly inactive. This concept had to be revised first when “paradoxical sleep”, also known as rapid-eye movement (REM) sleep was discovered with the surprising observation of an intense activation in the polysomnographic signal while the sleeper appeared completely inactive physically, a sleep stage also named rapid-eye-movement sleep (REM) [1]. Subsequent studies have shown that REM and non-REM sleep states contribute to the homeostasis in functions such as emotion regulation and learning and memory, as well as cardiovascular, metabolic, and immune functions [2]. An interesting suggestion about the possible function of REM sleep has been proposed by Crick and Mitchison in 1983 [3]. Using a computer model, these authors suggest that one mechanism of REM sleep is to remove certain mode of interactions between neurons, a process they named “reverse learning” or “unlearning”. These authors discussed that one function of REM sleep could be to “forget” irrelevant information as well as to remember relevant information for survival and adaptation. Using polysomnography in humans [4,5], Feinberg and Campbell proposed that slow-wave-sleep (SWS) is important for cortical pruning, a process essential to neuroplasticity. Neuroplasticity refers to the molecular and cellular changes in neuronal networks occurring in response to inputs from the environment. Such a neurophysiologic process is an important part of learning, memory and adaptation mechanisms.

Healthy adaptations are essential to the maintenance of psychological health. The ability to successfully adapt to and recover from adverse conditions such as traumatic experiences in humans is called resilience. Psychological resilience refers to actions and attitudes the individual can use to adapt to challenging situations and to reach a new level of functioning; it can also produce a ‘steeling effect’ that promotes growth. Psychological resilience can be promoted by reducing risk factors for poor psychiatric outcomes following traumatic experiences and by increasing or reinforcing protective factors. Most studies on risk and protective factors have investigated psychosocial factors. Biological factors, such as sleep, also deserve to be seriously considered as an important part of resilience, since the disruption of sleep is a robust risk factor, whereas the treatment of sleep disturbances improves daytime emotional functioning. Therefore, the detection of sleep disturbances before or early after traumatic exposure could lead to increased resilience and accelerated recovery from trauma reactions.

The hypothesis suggesting that sleep is an important mechanism of psychological resilience is ripe for empirical testing. An increasing amount of research is being conducted and recent advances in technologies bring new and exciting results supporting this hypothesis. One of the most productive fields of research on the relationships between trauma and sleep is found in the studies on post-traumatic stress disorder (PTSD). The diagnosis of PTSD requires that the individual has been exposed to a traumatic experience and presents symptoms of hyperarousal (sleep disturbances, hypervigilance, irritability, anxiety), re-experiencing (flashbacks, nightmares), and avoidance. The prevalence estimate of trauma exposure in the general population ranges from 50% to 90%; of these exposed individuals, 5%-10% will suffer from PTSD, with estimates as high as 15%-30% in those exposed to interpersonal violence. From those individuals who will develop a PTSD following a traumatic experience, 94% will suffer from insomnia compared to

10-30% in individuals who do not develop a PTSD following comparable traumatic experiences. The same is also observed for nightmares (75%-95% in PTSD compared to 4%-8% in non-PTSD) and for sleep-disordered breathing (50%-80% in PTSD compared to 2%-24% in non-PTSD). These results are obtained irrespective of the type or severity of the traumatic experience [6]. Objective studies using polysomnography show sleep disturbances during non-REM and REM sleep in PTSD subjects compared to non-PTSD controls while other studies fail to show differences between groups [7]. One positron emission tomography (PET) study show hypermetabolism in limbic and paralimbic brain structures during wakefulness and during REM sleep in combat exposed veterans with PTSD compared to combat-exposed veterans without PTSD [8]. While these observations indicate that neurobiological changes characterize REM sleep in PTSD, it is not clear whether diurnal hypervigilance and anxiety cause brain hypermetabolism during REM sleep, or whether disturbed REM sleep impairs the ability to function in a well adapted manner during the day. It is shown that disrupted sleep, along with increased fatigue, pain and cognitive dysfunctions, follow stress and trauma in combat exposed military veterans [9,10]. Interestingly, studies also observe that subjective and objective sleep disturbances following trauma exposure predict later PTSD, as well as other psychiatric outcomes including major depression, other anxiety disorders, and substance use disorders [11-15].

Trauma-related sleep disturbances appear to be a biological, yet modifiable threat to psychological resilience. Fortunately, sleep disturbances can be effectively treated, which in turn may contribute to increased resilience and better psychiatric outcomes. Studies show that behavioral treatment of nightmares and insomnia reduces daytime PTSD symptoms, depression and anxiety [16,17,8]. These studies aim to determine if sleep preservation or restoration can lead to increased psychological resilience, accelerated recovery and reduced risks of poor outcomes. Translational research efforts are needed to identify the sleep-specific genetic, molecular and neurobiological pathways that contribute to psychological resilience.

References

1. Aserinsky E, Kleitman N (1953) Regularly Occurring Periods of Eye Motility, and Concomitant Phenomena, during Sleep. *Science* 118: 273-274.
2. Siegel JM (2005) Clues to the functions of mammalian sleep. *Nature* 437: 1264-1271.
3. Crick F, Mitchison G (1983) The function of dream sleep. *Nature* 304: 111-114.
4. Feinberg I (1990) Cortical pruning and the development of schizophrenia. *Schizophr Bull* 16: 567-570.
5. Feinberg I, Campbell IG (2010) Sleep EEG changes during adolescence: an index of a fundamental brain reorganization. *Brain Cogn* 72: 56-65.

*Corresponding author: Julie Poulin, University of Pittsburgh Medical Center, Western Psychiatric Institute and Clinic, Pittsburgh, USA, Tel: 412-246-6005; Fax: 412-246-5560; E-mail: poulinj@upmc.edu

Received January 12, 2012; Accepted January 12, 2012; Published January 21, 2012

Citation: Poulin J, Kim WJ, Germain A (2012) Resilience through Sleep. *J Sleep Disord Ther* 1:e105. doi:10.4172/2167-0277.1000e105

Copyright: © 2012 Poulin J, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

6. Germain A, Buysse DJ, Shear MK, Fayyad R, Austin C (2004) Clinical correlates of poor sleep quality in posttraumatic stress disorder. *J Trauma Stress* 17: 477-484.
7. Kobayashi I, Boarts JM, Delahanty DL (2007) Polysomnographically measured sleep abnormalities in PTSD: a meta-analytic review. *Psychophysiology* 44: 660-669.
8. Germain A, James J, Mammen O, Price P, & Nofzinger EA. Functional Neuroimaging of REM Sleep in Returning Veterans with PTSD: An [18F]-FDG PET Study. *Sleep*, 34(Suppl.): A242, 2011.
9. Kroenke K, Koslowe P, Roy M (1998) Symptoms in 18,495 Persian Gulf War veterans. Latency of onset and lack of association with self-reported exposures. *J Occup Environ Med* 40: 520-528.
10. Roy MJ, Koslowe PA, Kroenke K, Magruder C (1998) Signs, symptoms, and ill-defined conditions in Persian Gulf War veterans: findings from the Comprehensive Clinical Evaluation Program. *Psychosom Med* 60: 663-668.
11. Breslau N, Roth T, Rosenthal L, Andreski P (1996) Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biol Psychiatry* 39: 411-418.
12. Koren D, Arnon I, Lavie P, Klein E (2002) Sleep complaints as early predictors of posttraumatic stress disorder: a 1-year prospective study of injured survivors of motor vehicle accidents. *Am J Psychiatry* 159: 855-857.
13. Mellman TA, Bustamante V, Fins AI, Pigeon WR, Nolan B (2002) REM sleep and the early development of posttraumatic stress disorder. *Am J Psychiatry* 159: 1696-1701.
14. Harvey AG, Bryant RA (1999) The relationship between acute stress disorder and posttraumatic stress disorder: a 2-year prospective evaluation. *J Consult Clin Psychol* 67: 985-988.
15. Bryant RA, Creamer M, O'Donnell M, Silove D, McFarlane AC (2010) Sleep disturbance immediately prior to trauma predicts subsequent psychiatric disorder. *Sleep* 33: 69-74.
16. Germain A, Nielsen T (2003) Impact of imagery rehearsal treatment on distressing dreams, psychological distress, and sleep parameters in nightmare patients. *Behav Sleep Med* 1: 140-154.
17. Germain A, Shear MK, Hall M, Buysse DJ (2007) Effects of a brief behavioral treatment for PTSD-related sleep disturbances: a pilot study. *Behav Res Ther* 45: 627-632.