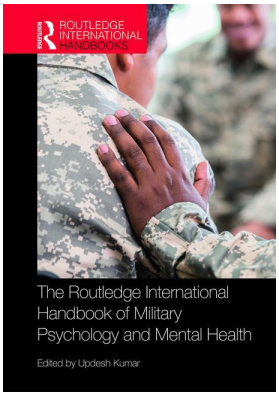

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MILITARY-RELATED MENTAL HEALTH MORBIDITIES

A neurobiological approach

Shobit Garg and Jyoti Mishra

The nature of military-related morbidities has changed over time considerably. Preventive measures like improved protective gear have been effective in reducing military morbidity and mortality. Moreover, survival rates of post-battle physical injuries have improved due to better evacuation and tertiary care facilities. Therefore, recently psychological morbidities as an effect of war have started to outnumber physical injuries. Behavioral neuroscience has grown leaps and bounds studying normal and abnormal psychology of humans. Studying abnormal psychological phenotypes, that is, military psychological morbidity, from a behavioral neuroscience perspective is pertinent to understand the etiology, progression, presentation, treatment and prevention of illnesses. A simplistic tool to execute this is neurobiology.

What is neurobiology? Neurobiology is a branch of biology that deals with nervous system functions and structures. This branch focuses on how cellular level functions interact to model the various circuits involved in execution of normal and abnormal behavior. A neurobiological approach is the understanding of the neurochemical and neuro-electrical dynamics brought upon by genetic underpinnings or environmental conditions. In the following sections, we will deal with important military-related mental health morbidities like suicide, post-traumatic stress disorder (PTSD), sleep disorder, substance use disorders (SUDs) and traumatic brain injuries from a neurobiological perspective.

Suicidal and related issues

Suicide is a significant public health issue within the Department of Defense. It currently remains the leading cause of mortality in the West (Ghahramanlou-Holloway, Baer, Neely, Koltko, & Nielsen, 2017; Armed Forces Health Surveillance Center (AFHSC), 2014). The unadjusted suicide rates per Western data for the four services have been reported as 23.8 (Army), 18.5 (Air Force), 17.9 (Marine Corps), and 16.3 (Navy) per 100,000 (Ghahramanlou-Holloway et al., 2017).

There are several risk factors reported, mostly via cross sectional or retrospective studies. These factors are demotion within the last two years, early military separation, dishonorable discharge from duty, relationship issues, and access to means (e.g., firearms). Specific subpopulations within the military such as infantrymen and combat engineers are also at higher risk for suicide. Military personnel suffering from physical pain, injury, or sleep problems, as well as increased rates of both

Axis I and Axis II disorders, significantly elevate suicide risk (Ghahramanlou-Holloway et al., 2017). Recent studies have found no correlation between deployment status and suicidal rates, but if a correlation exists, it has been found to be mediated by the development of depression and PTSD.

Factors protecting against suicide include adequate social support, higher unit cohesion and good workplace relationships, training and preparation, religious involvement, spouse's preparedness for deployment, and individual resilience (Ghahramanlou-Holloway et al., 2017). Suicide is a heterogeneous condition. Research pertaining to the neurobiology of suicide is mainly from postmortem studies of nonmilitary populations. Differences from the civilian population are largely because of epigenetic interactions citing various risk and protective factors as mentioned above. Many explanatory and predictive models of suicidal behavior have been hypothesized and a few are explained below (Nizamie & Garg, 2012).

Unified model of suicide

Suicidal behavior is conceptualized as the outcome of the balance between an individual's threshold for suicidal acts and triggers of suicidal behavior. Threshold refers to the propensity for manifesting suicidal behavior and may be considered trait related. In contrast, triggers are precipitant contributing to the probability of suicidal acts and are considered state related. Various risk factors for suicidal behavior have been categorized according to whether they affect the threshold or the trigger. Risk factors affecting threshold could be genetic (e.g., family history), biological (e.g., chronic combat stress), neurochemical (e.g., low 5-HIAA in CSF), and so on, whereas factors affecting triggers for suicidal behavior could be social, financial, and family crisis in a military context. This model functions in the line of biopsychosocial theory (Mann & Arango, 1992).

A four-pathway clinical-biochemical model

There are four hypothesized pathways to suicide in terms of clinical features and biochemical variables. Of these pathways, the elevated CRH-anxiety agitation pathway appears to be an acute/state pathway. Hypothesized low serotonin function (correlated with low serum cholesterol) associated with impulsiveness could be a state or trait pathway. Hopelessness appears to be a trait pathway, although its severity is certainly triggered to the severity of clinical depression. Anhedonia severity appeared as an acute suicide risk factor, suggesting a state variable, and it may be a secondary variable affected by the severity of anxiety or other depression-related symptoms (Fawcett, Busch, Jacobs, Kravitz, & Fogg, 1997).

Stress-diathesis model

The relation between risk factors can be described in explanatory models of suicide, such as the stress-diathesis model. Acute psychosocial crises and psychiatric disorders such as depression are common proximal stressors leading to suicidal behavior, while pessimism or hopelessness and aggression or impulsivity are components of the diathesis for suicidal behavior. Familial or genetic factors, childhood experiences, and other factors, including cholesterol concentrations, could influence the diathesis (Hawton & Heeringen, 2009).

Postmortems of suicidal probands have shown greater serotonin neurons, more tryptophan hydroxylase 2 gene expression in the brainstem with reduction in dentate gyrus volume, and immature neuronal architecture. Molecular studies have demonstrated that in people who have attempted suicide, prefrontal localized hypo-function and impaired serotonergic responsivity was found to be proportional to the lethality of suicide attempts (Heeringen & Mann, 2014).

Neurobiological perspectives shed light on the treatment options available for suicidal risk. Electroconvulsive therapy has short-term life-saving effects in high suicidal risk. More recently, ketamine in subanesthetic doses was reported to have an acute antisuicide effect that lasted for about one week (Kellner et al., 2005; Larkin & Beautrais, 2011). Most antidepressant drugs enhance serotonergic levels, leading to a putative antisuicidal effect. A single randomized clinical trial of suicidal patients showed that a selective serotonin-reuptake inhibitors had a better antidepressant effect and greater reduction in suicidal ideation than did a noradrenergic–dopaminergic drug (Grunebaum et al., 2012; Heeringen & Mann, 2014).

Combat stress reaction

During World War I (WWI), combat stress was referred to as shell shock, effort syndrome, war neurosis, Da Costa's syndrome, or irritable heart syndrome. The U.S. Department of Defense (DoD) describes **combat stress reaction** as the "expected, predictable, emotional, intellectual, physical, and/or behavioral reactions of service members who have been exposed to stressful events in combat or military operations other than war." One of the most commonly studied postcombat disorders is PTSD (Campise, Geller, & Campise, 2006).

PTSD is a significant issue that ought to be addressed and monitored in the military. PTSD can occur after someone experiences or witnesses a traumatic event. Examples can be combat, a terrorist attack, sexual or physical assault, a serious accident, or a natural disaster. PTSD can be a significant condition, especially when symptoms continue for more than one month after exposure to a trauma and cause significant distress or impairment in social, occupational, or other important areas of functioning. There are four main categories of symptoms: intrusions (e.g., nightmares), avoidance, negative alterations in cognitions and mood (e.g., guilt), and alterations in arousal and activity (e.g., hyperarousal). The vast majority of people recover quickly and have no long-term effects. The incidence rate of PTSD for active duty personnel is 0.6%. These rates could be artificially low due to stigma, minimization of symptoms, and mistrust (or misunderstanding) of mental health professionals. Post-deployment PTSD rates rise to 10% of the respective population (Mehlum & Weisaeth, 2002; Johnston, Robinson, Earles, Via & Delaney, 2017). The Vietnam War raised the awareness of PTSD and the decades that followed taught that "PTSD is not a constant or static condition, but a disorder that may actually wax and wane throughout a lifetime" (Falk, Hersen, & Van Hasselt, 1994; Johnston et al., 2017).

Neurological model of post-traumatic stress disorder

Various cognitive aspects of PTSD have recently gained attention, including attentional biasing, reduced recall of both traumatic and nontraumatic material, and impaired executive skills. Several brain areas are implicated in PTSD (Scaglione & Lockwood, 2014):

Over-activation of the amygdala: Hyper-reactivity of the amygdala is a robust finding in PTSD. In PTSD the amygdala also has exaggerated responses to non-trauma-related affective material, such as fearful facial expressions as well as attentional tasks. The degree of amygdala activation is also correlated with PTSD severity.

Underactivation and cell loss of the hippocampus: In PTSD, diminished activation of the hippocampus is often found. A meta-analysis of MRI volumetric studies of PTSD by Smith (2005) indicated a 6% loss in hippocampal volume. Cortisol neurotoxicity (due to chronic stress) is a neuropathological correlate of these findings. The hippocampus has some inhibitory influence on the amygdala under physiological conditions. In PTSD,

the amygdala-hippocampal circuits related to attentional biasing, encoding, and memory processes are overactive, possibly due to increased innervation from the original traumatic incident and a decrease in neuronal volume (Mahan & Ressler, 2012; Morey et al., 2012).

Dysregulation of frontal-subcortical circuit: Normal brain functioning requires complex feedback circuitry between subcortical and cortical systems. The frontal lobes function as a cortical-subcortical network. All **frontal-subcortical circuits** are closed-loop "final effector mechanisms" which require integration of external and internal/limbic information and the initiation of appropriate responses. There is a dynamic balance maintained between bottom-up survival-oriented activity and top-down directing of goal-oriented planning behavior which is found to be disrupted in PTSD, particularly anterior cingulated **frontal-subcortical circuits** and the medial division of the orbitofrontal systems which involve connections to the amygdala. This dysregulation could account for a number of neurocognitive processes in PTSD, such as fear conditioning, habituation, and extinction, cognitive–emotional interactions, and emotional processing (Scaglione & Lockwood, 2014).

Treatment

Cognitive-behavioral therapy is the most empirically supported treatment for PTSD. It is effective in only about 50% of PTSD patients. No straightforward pharmacotherapy is effective in PTSD either. Cognitive rehabilitation of executive functions has also shown to be useful in PTSD, especially in veterans returning from combat with head injuries in addition to PTSD (Scaglione & Lockwood, 2014). Prolonged exposure (PE) therapy is also an evidence-based therapeutic option in PTSD (IOM, 2012).

Substance use disorders

Historically, the worst substance problems were evident in 1971 in the Vietnam War, where 34% of soldiers admitted to marijuana use and 50% to the use of heroin. More service members were medically evacuated for drug use than for war wounds. Alcohol abuse and other substance use have been the 5th and 10th most commonly occurring disqualifiers for the Navy personnel. Those who are heavy drinkers (five or more drinks at least once per week) are more likely to be late to work, to leave work early, to exhibit decreased job performance, and to suffer more on-site injuries (Fisher, Hoffman, Austin-Lane, & Kao, 2000). The military lifestyle has numerous challenges like deployment to danger zones and loss of personal freedom not encountered by civilian population. When challenged, those with inadequate coping styles would consume alcohol in harmful patterns (25% of both military men and women). Substance use disorder is a common endemic affecting the young, adult male population. One five-year longitudinal study found that 75% of U.S. Navy recruits used alcohol prior to enlistment and 31% had used illegal drugs (Kennedy, Jones, & Grayson, 2006). One of the core features of SUDs and major factors of relapse is craving, which has several neurobiological underpinnings explained below as psychobiological models (Garg, Dharmadhikari, & Sinha, 2012).

Psychobiological model

These models are directly influenced by biological neural systems (neural circuitry, reward systems, and neuroanatomical systems) and are as follows.

Neuroadaptive model

There is hyper-sensitization of the dopamine neural transmitter system (implicated in reward pathways) in substance abuse. This in turn increased the incentive salience of drugs. "Incentive salience"' makes stimuli more attractive and turns ordinary wanting into excessive drug craving (Robinson & Berridge, 1993). Drugs cause neurobiological adaptations to maintain homeostasis, and if drug consumption ceases, an imbalance in cerebral activity results in craving. In the early withdrawal state, craving occurs to alleviate the imbalance. In a later recovery state, the altered brain functions return to their original state, but cravings may appear suddenly. This reactivation can be triggered by stress, which may activate the reward memory (Anton, 1999). The reward memories may also reactivate neurochemical processes associated with past experiences of drug use and cause craving.

Theory of neural opponent motivation

Chronic drug exposure causes allostasis, defined as "a state of chronic deviation of the regulatory systems with establishment of a new set point." An allostatic state involves a feed-forward mechanism rather than the negative feedback mechanism of homeostasis. Over-activation of the brain reward system triggers the brain stress circuit or anti-reward system in order to limit the reward. Long-term drug intake dysregulates the underlying neurochemical functions, resulting in an allostatic state. An anti-reward system and neurotransmitter changes create powerful negative reinforcement. Craving arises from the action of memory of the rewarding effects of drug use superimposed on a negative emotional state (Koob & Moal, 2008).

Temporal-difference reinforcement learning model

In a temporal-difference reinforcement learning model (TDRL), actions are selected to maximize future rewards which are based on the strength of a value signal, defined as the expected future reward discounted by the expected time to the reward. This value signal is carried by dopamine and produces temporal-difference learning in the normal brain. TDRL is based on assumptions about cocaine, which produce a phasic increase in dopamine directly and push a person towards irrational behavior (Redish, 2004).

Model of interoceptive dysregulation

Interoception refers to the sensations that originate from the interior of the body. The interoceptive state is mediated by the anterior insular cortex which has bidirectional connections to the amygdala and the ventral striatum. Alteration in interoceptive processing is due to an altered "prediction error," which refers to the difference between the value of the anticipated sensation (i.e., the hoped-for result) and the value of the current interoceptive state. Dysregulation of the insular cortex minimizes the body prediction error, causing non-adaptive adjustment of the body prediction error, which causes craving in the addiction process (Paulus, Tapert & Schulteis, 2009).

Treatment of substance use disorders

Although treatment review in detail is beyond the scope of this chapter, few salient points like principles of treatment, tobacco cessation program, and preventive strategies are discussed. Military personnel with substance abuse diagnoses warrant outpatient treatment (Level I). Similarly, a

substance dependence diagnosis generally warrants either intensive outpatient treatment (Level II) or residential treatment (Level III). Exceptions might be those who previously completed treatment for substance dependence and remain sober for a significant period of time but then had a brief relapse. If they want to remain sober and demonstrate motivation to follow a recovery plan, they may be best served by an outpatient treatment (Kennedy et al., 2006).

Tobacco cessation

Smoking is the single most important health risk in military personnel. Smoking affects personnel readiness through lower levels of physical fitness, increased injuries, and more sick days. Again, increasing tobacco use is due to stressful circumstances like deployment and high workload. The prevalence of smoking in the military (any smoking in the past 12 months) is as follows: Army, 35.6%; Navy, 36.0%; Marine Corps, 38.7%; Air Force, 27.0%. Almost every military installation in the West offers programs for tobacco cessation. Most of these programs use a combination of a behaviorally based program combined with nicotine replacement therapy (NRT) such as patches and gum and buproprion hydrochloride. Abstinence rates with these tobacco cessation program remain high (Peterson, 2006).

Prevention of substance use disorders

Various military policies around the globe mandate prevention training for 100% of new military members, and annual training is required for all troops, in addition to random urine drug testing. The basic objectives of promoting mission readiness and the health and wellness of troops through the prevention of substance abuse are promoted via these policies. The *Navy's program* is a good example of how to disseminate best-practice information on alcohol abuse prevention (navdweb.spawar.navy.mil). The "three Rs" (relationship, relevance, and responsibility) are identified as a core program: a positive mentoring relationship, the relevance of everyone's role in the success of the mission, and the responsibility to learn and integrate expectations, as well as leadership's responsibility to provide information and facilitate the prevention program (Kennedy et al., 2006).

Sleep issues in military psychology

According to a survey, around 48% of the military population suffers from significant sleep problems with no significant differences between deployment status and high and low combat exposure. Moreover, 31% of respondents were in "extreme short sleeper category" (five or fewer hours of sleep per night) (Campbell et al., 2017).

Sleep regulation

There is two-process model of sleep regulation regulating many aspects of sleep including its timing, duration, stages, and quality. The first biological process, called sleep homeostasis, is a drive for sleep that increases with time awake and attenuates with time asleep. The sleep homeostatic drive would continuously increase with sleep deprivation such as on a typical day of 18 hours of being awake. The second biological process of sleep regulation is circadian rhythm, which is a 24-hour biological clock that synchronizes the timing of sleep and wakefulness. Various hormones like melatonin and core body temperature are directed by the circadian clock. These two processes are physiologically distinct; when aligned, they interact to promote consolidated

periods of sleep during darkness at night and wakefulness during the day. Regulated sleep is known to have vital implications for healthy cognition and behavior (Campbell et al., 2017).

Health consequences

The American Academy of Sleep Medicine and Sleep Research Society recently recommended that adults obtain 7–9 hrs of sleep per night (Watson et al., 2015). Sleep durations below this recommended amount, or poor sleep quality, have been associated with negative health consequences like weight gain, insulin resistance, increased risk of heart failure and coronary artery disease (due to insomnia), and immune dysfunction, to name a few (Campbell et al., 2017). Per the most comprehensive study by Mysliwiec et al. (2013) in military personnel who underwent polysomnography, the most commonly occurring sleep disorders were: mild obstructive sleep apnea (OSA; 27.2%), insomnia (24.7%), moderate-to-severe OSA (24%), behaviorally induced insufficient sleep syndrome (8.9%), snoring (5.3%), and paradoxical insomnia (5.1%). The most prevalent psychiatric co-morbidities were depression (22.6%), anxiety (16.8%), and PTSD (13.2%). Sleep problems could precede or co-occur or be a consequence of psychiatric co-morbidities. Moreover, deployment status is associated with poor quality of sleep (around 70%) but mediated by mental health morbidities (Campbell et al., 2017). Fatigue remains a challenge to high performance, and sleep disruption is one of the several contributors to fatigue. Fatigue is especially relevant in light of continuous operations and sustained operations due to related performance and safety decrements and possible adverse outcomes. The Aerospace Medical Association has released a position statement as countermeasures for aviation fatigue (Caldwell et al., 2009):

1. Fatigue is a physiological problem that cannot be overcome by motivation, training, or will power.
2. People cannot reliably self-judge their own level of fatigue-related impairment.
3. There are wide individual differences in fatigue susceptibility that must be taken into account but which presently cannot be reliably predicted.
4. There is no one-size-fits-all "magic bullet" (other than adequate sleep) that can counter fatigue for every person in every situation.
5. Valid counter-fatigue strategies will enhance safety and productivity, but only when they are correctly applied.

Maritime operations

Shift work is an unavoidable reality in maritime operations. Shift work that is not aligned with the day/night, wake/sleep cycle is a threat to psychological and physical health, resulting in loss of sleep homeostasis and a pathological condition known as shift work disorder (SWD). Of particular concern is the common 5/10 schedule in which sailors are on-duty for 5 h, followed by 10 h off (15-hour day). The 5/10 schedule results in different clock hours throughout the day–night cycle over a 72-hour period, resulting in de-synchrony between the sleep homeostatic drive and the internal circadian clock. The 5/10 shift schedule is associated with low psychological resilience and psychomotor performance (Campbell et al., 2017).

Treatment implication

Molecules like benzodiazepines, Z drugs like zolpidem (for the short term), and like ramelteon (for chronic usage) can be used for insomnia as per the recommendations. Nonpharmacological

interventions, specifically cognitive behavioral therapy for insomnia (CBT-I), is the recommended first-line treatment for insomnia (Schutte-Rodin, Broch, Buysse, Dorsey & Sateia, 2008).

Neuropsychological issues in military

Neuropsychology is the science and study of brain-behavior relationships and the clinical application of that knowledge. The primary goal of neuropsychology in armed forces is to help service members who have experienced neurological disorders or injuries by providing assessment, diagnosis, and treatment plans to foster effective recovery (Green, Jacobson, Waggoner & Armistead-Jehle, 2017).

Traumatic brain injury (TBI) is one of the most commonly occurring neurological conditions. Other neurological morbidity includes seizure disorders, cerebral vascular accidents, neoplasms, neurodegenerative conditions, hypoxia, etc. TBI can result in a variety of cognitive, emotional, behavioral, and physical sequelae, depending on the severity and the location of the cerebral damage. The cognitive deficits often involve attention and concentration, executive functioning, memory, and expressive language. Emotional and behavioral problems include apathy, irritability, disinhibition, depression, anxiety, and mood lability. The physical symptoms include dizziness, balance problems, vision changes, hearing changes, and headaches (Ryan, Zazeckis, French & Harvey, 2006; Green et al., 2017).

There have been two types of TBI: blunt force and blast wave (bTBI). Few of the neurobiological mechanisms have been elucidated to explain blast wave TBI (Courtney & Courtney, 2015):

1. *Acceleration mechanism*: Rotational accelerations of the brain caused by exposure to a blast wave may result in bTBI.
2. *Direct cranial entry*: A pressure transient traverses the skull and directly injures brain tissue. The pressure transient may result from direct transmission due to bulk motion of the skull. Localized peak pressures may result from constructive interference of internally reflected waves.
3. *Thoracic mechanism*: Combination of a pressure transient reaching the brain via the thorax and a vagally mediated reflex result in bTBI. Transient increased intracranial pressures in the cerebral vasculature may result from high speed propagation of a pressure transient without significant bulk motion.

Concussion injury. Concussion or mTBI has been called the "signature injury" in military circles. Approximately 80% of concussions occur in garrison, calling into question whether it is truly a deployment-related issue that has a higher representation than other battle-related injuries. Concussive symptoms such as headache, photophobia, phonophobia, sleep difficulties, dizziness, and psychiatric conditions after three months of recovery is known as persistent postconcussive symptoms (PPCS) and is prevalent in around 15% of individuals. PPCS needs detailed evaluation which includes neuropsychological testing, a neurological examination, an audiology examination, a psychiatric interview, an EEG, and neuroimaging to look for evidence of diffuse axonal injury or hemorrhage (Ryan et al., 2006; Green et al., 2017).

Gulf War Syndrome. Gulf War syndrome constitutes a wide range of physical (e.g., fatigue, pain, sleep disturbance, fever, rashes, tremor, and sexual dysfunction), cognitive (e.g., attention and memory problems), and psychological (e.g., depression and anxiety) symptoms (Hom, Haley & Kurt, 1997; Ryan et al., 2006). Various theories of the origin of Gulf War syndrome exist. These range from the influence of preexisting conditions or exposure to toxins or inoculations to psychological problems such as posttraumatic stress disorder or depression.

Malingering. World War II saw the publication of multiple articles on malingering in order to avoid military service or discipline. Common methods of malingering purported at this time were consuming alcohol, epinephrine, sugar, and cathartics; claims of pain or other sensory problems (e.g., blindness); claims of motor dysfunction; feigning of insanity; self-mutilation or exaggeration of real symptoms; etc.

Malingering in a military context (as per Uniform Code of Military Justice) refers to any service member who for the express purpose of avoiding a military duty feigns illness, physical disability, or a mental health issue or intentionally inflicts an injury to oneself (Joint Service Committee on Military Justice, 2005). The malingerer may simulate depressive symptoms, make suicidal comments, or adopt nonexistent psychotic symptoms to avoid a specific military duty, to be relieved from a specific duty (e.g., deployment), or to attempt to leave military service prematurely. Malingering could pose difficult diagnostic dilemmas for a military psychologist. There is no litmus test for malingering. The following red flags should raise the suspicion: the member keeps adding symptoms during the interview, the symptoms do not make diagnostic sense, there seems to be clear secondary gain in avoiding duty, elevations on psychological testing suggest faking a bad profile, and/or a resolution of symptoms occurs immediately after the desired outcome (Ryan et al., 2006).

The phenomenon of denial of actual symptoms which are present is known as reverse malingering, faking good, or dissimulation. Individual are evaluated for depression, suicidal or homicidal ideation, an alcohol incident, or domestic abuse. The secondary gain is to avoid the stigma of a mental health diagnosis or a negative career impact. In cases of suspected simulation and dissimulation, information must be collected from at least four sources. The first source is companions or supervisors. The second source is provided by the clinical interview. The third source is the medical record, and the fourth is psychological testing (Ryan et al., 2006).

Conclusion and future directions

A neurobiological approach or perspective has important implications for military-related mental health morbidities. Studying neurobiology via the help of neuroimaging, either structural or functional, would help us to better delineate brain regions and networks, e.g., for those involved in suicide risk, and could be a way to track the effectiveness of interventions targeting such specific neural networks and brain regions. Future imaging–genetic approaches which combine neuroimaging and genomic study would help us to study the association between circuitry-related changes and gene (with epigenetic modification) function and behavioral neuroscience. Neurobiological models using state-of-the-art high-throughput resting-state functional MRI could help us to enumerate quantitative phenotypes for molecular genetic studies and biomarkers of pathological processes in the brain. These biomarkers in future would encourage more sophisticated and personalized treatments like transcranial magnetic stimulation in the military context.

References

Anton, R. (1999). What is craving: Models and implications for treatment. *Alcohol Research and Health*, *23*, 165–173.

Armed Forces Health Surveillance Center (AFHSC). (2014). Suicides and suicide attempts among active component members of the U.S. Armed Forces, 2010–2012: Methods of self-harm vary by major geographic region of assessment. *Medical Surveillance Monthly Report*, *21*, 2–5.

Caldwell, J. A., Mallis, M. M., Caldwell, J. L., Paul, M. A., Miller, J. C., & Neri, D. F. (2009). Fatigue countermeasures in aviation. *Aviation, Space, and Environmental Medicine*, *80*, 29–59.

Campbell, J. S., Markwald, R., Chinoy, E. D., Germain, A., Griese, R. E., Lim, I., & Bowles, S.V. (2017). A sleep primer for military psychologists. In S.V. Bowles, & P.T. Bartone (Eds.), *Handbook of Military Psychology* (pp. 239–260). Washington, DC, USA, Springer.

Campise, R. L., Geller, S. K., & Campise, M. E. (2006). Combat stress. In C.H. Kennedy & E.A. Zillmer (Eds.). *Military Psychology* (pp. 215–240). New York: The Guilford Press.

Courtney, A., & Courtney, M. (2015). The complexity of biomechanics causing primary blast-induced traumatic brain injury: A review of potential mechanisms. *Frontiers in Neurology*, *6*, 1–12.

Falk, B., Hersen, H., & Van Hasselt, V. B. (1994). Assessment of post-traumatic stress disorder in older adults: A critical review. *Clinical Psychology Review*, *14*, 383–416.

Fawcett, J., Busch, K. A., Jacobs, D., Kravitz, H. M., & Fogg, L. (1997). Suicide: A four-pathway clinical biochemical model. *Annals of the New York Academy of Sciences*, *836*, 288–301.

Fisher, C. A., Hoffman, K. J., Austin-Lane, J., & Kao, T. (2000). The relationship between heavy alcohol use and work productivity loss in active duty military personnel: A secondary analysis of the 1995 Department of Defense worldwide survey. *Military Medicine*, *165*, 355–361.

Garg, S., Dharmadhikari, A. S., & Sinha, V. K. (2012) Craving in substance use disorders. *Indian Journal of Social Psychiatry*, *28*(1–2), 43–52.

Ghahramanlou-Holloway, M., Baer, M. M., Neely, L.L., Koltko, V., & Nielsen, M. K. (2017). Suicide prevention in the United States military. In S. V. Bowles, P. T. Bartone (Eds.). *Handbook of Military Psychology* (pp. 73–88). Washington, DC, USA, Springer.

Green, R. R., Jacobson, D. A., Waggoner, J. W., & Armistead-Jehle, P. (2017). Neuropsychology in the Military. In S. V. Bowles & P. T. Bartone (Eds.). *Handbook of Military Psychology* (pp. 137–156). Washington, DC, USA, Springer.

Grunebaum, M. F., Ellis, S. P., Duan, N., Burke, A. K., Oquendo, M. A., & Mann, J. (2012). Pilot randomized clinical trial of an SSRI vs bupropion: Effects on suicidal behavior, ideation, and mood in major depression. *Neuropsychopharmacology*, *37*, 697–706.

Hawton, K., & Heeringen, K. V. (2009). Suicide. *Lancet*, *373*, 1372–1381.

Heeringen, K., & Mann, J. J. (2014). The neurobiology of suicide. *Lancet Psychiatry*, *1*, 63–72.

Hom, J., Haley, R. W., & Kurt, T. L. (1997). Neuropsychological correlates of Gulf War syndrome. *Archives of Clinical Neuropsychology*, *12*, 531–544.

IOM (Institute of Medicine). (2012). *Treatment for Posttraumatic Stress Disorder in Military and Veteran Populations: Initial Assessment*. Washington, DC: The National Academies Press. ISBN: 978-0-309-25421-2, 396 pages. Retrieved from http://www.nap.edu/catalog.php?record_id=13364

Johnston, S. L., Robinson, C., Earles, J. E., Via, J., & Delaney, E. M. (2017). State of psychology in the US armed forces. In S. V. Bowles & P. T. Bartone (Eds.), *Handbook of Military Psychology* (pp. 1–19). Washington, DC, USA, Springer.

Joint Service Committee on Military Justice. (2005). *Manual for Courts-Martial*. Washington, DC: U.S. Government Printing Office.

Kellner, C. H., Fink, M., Knapp, R., Petrides, G., Husain, M., Rummans, T., … Malur, C. (2005). Relief of expressed suicidal intent by ECT: A consortium for research in ECT study. *American Journal of Psychiatry*, *162*, 977–982.

Kennedy, C. H., Jones, D. E., & Grayson, R. (2006). Substance abuse services and gambling treatment in the military. In C. H. Kennedy & E. A. Zillmer (Eds.), *Military Psychology* (pp. 163–192). New York, London: The Guilford Press.

Koob, G. F., & Le Moal, M. (2008). Addiction and the brain antireward system. *Annual Review of Psychology*, *59*, 29–53.

Larkin, G. L., & Beautrais, A. L. (2011). A preliminary naturalistic study of low-dose ketamine for depression and suicide ideation in the emergency department. *International Journal of Neuropsychopharmacology*, *14*, 1127–1131.

Mahan, A. L., & Ressler, K. J. (2012). Fear conditioning, synaptic plasticity and the amygdala: Implications for posttraumatic stress disorder. *Trends in Neurosciences*, *35*, 24–35.

Mann, J. J., & Arango, V. (1992) Integration of neurobiology and psychopathology in a unified model of suicidal behavior. *Journal of Clinical Psychopharmacology*, *12*, 2S–7S.

Mehlum, L., & Weisaeth, L. (2002). Predictors of posttraumatic stress reactions in Norwegian U.N. peacekeepers 7 years after service. *Journal of Trauma and Stress*, *15*(1), 17–26.

Morey, R. A., Gold, A. L., LaBar, K. S., Beall, S. K., Brown, V. M., Haswell, C. C.,… McCarthy, G. (2012). Amygdala volume changes in posttraumatic stress disorder in a large case-controlled veterans group. *Journal of the American Medical Association: Psychiatry*, *69*, 1169–1178.

Mysliwiec, V., Gill, J., Lee, H., Baxter, T., Pierce, R., Barr, T.L.,… Roth, B. J. (2013). Sleep disorders in US military personnel: A high rate of comorbid insomnia and obstructive sleep apnea. *Chest*, *144*(2), 549–557.

Nizamie, S. H., & Garg, S. (2012). Depression and suicide. In N. G. Desai (Ed.), *Dealing with Depression in Medically Ill Patients* (pp. 50–73). New Delhi, India: Elsevier.

Paulus, M. P., Tapert, S. F., & Schulteis, G. (2009) The role of interoception and alliesthesia in addiction. *Pharmacology, Biochemistry and Behaviour*, *94*, 1–7.

Peterson, A.L. (2006). Clinical health psychology and behavioral medicine in military healthcare settings. In C. H. Kennedy & E. A. Zillmer (Eds.), *Military Psychology* (pp. 74–104). New York, London: The Guilford Press.

Redish, A. D. (2004). Addiction as a computational process gone awry. *Science*, *306*, 1944–1947.

Robinson, T., & Berridge, K. (1993). The neural basis of craving: An incentive-sensitization theory of addiction. *The Behavioural and Brain Sciences*, *18*, 247–291.

Ryan, L. M., Zazeckis, T. M., French, L.M., & Harvey, S. (2006). Neuropsychological practice in the military. In C. H. Kennedy & E. A. Zillmer (Eds.), *Military Psychology: Clinical and Operational Applications* (pp. 105–129). New York, London: The Guilford Press.

Scaglione C., & Lockwood, P. (2014). Application of neuroscience research to the understanding and treatment of posttraumatic stress disorder (PTSD). *International Journal of Applied Science and Technology*, *4*, 35–45.

Schutte-Rodin, S., Broch, L., Buysse, D., Dorsey, C., & Sateia, M. (2008). Clinical guideline for the evaluation and management of chronic insomnia in adults. *Journal of Clinical Sleep Medicine*, *4*(5), 487–504.

Smith, M. E. (2005). Bilateral hippocampal volume reduction in adults with post-traumatic stress disorder: A meta-analysis of structural MRI studies. *Hippocampus*, *15*, 798–807.

Watson, N. F., Badr, M. S., Belenky, G., Bliwise, D. L., Buxton, O., Buysse, D., … Tasali, E. (2015). Joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society on the recommended amount of sleep for a healthy adult: Methodology and discussion. *Journal of Clinical Sleep Medicine*, *11*, 931–952.