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### **Journal of Evolution and Health: A joint publication of the Ancestral Health Society and the Society for Evolutionary Medicine and Health**

#### **Title**

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#### **Permalink**

<https://escholarship.org/uc/item/0pf0r8zc>

#### **Journal**

Journal of Evolution and Health: A joint publication of the Ancestral Health Society and the Society for Evolutionary Medicine and Health, 5(1)

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#### **Publication Date**

2020

#### **DOI**

10.15310/J35152613

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Peer reviewed

Can the mammalian dive response override posttraumatic stress disorder?

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**Key words:** Posttraumatic Stress Disorder; Mammalian Dive Response; Fight or Flight; Vagal Tone; Vagus Nerve; Cold Facial Immersion; Evolution; Mammalian Dive Response Therapy

### **Abstract**

Posttraumatic stress disorder (PTSD) is a burdensome condition that has been made worse by the Covid-19 pandemic and that can lead to suicide, especially among childhood trauma victims, rape victims, military service members, and law enforcement. Physiological responses characterized by PTSD are thought to be evolutionary mechanisms that function to promote survival by heightening the stress response in an anticipatory fashion, allowing the organism to react quickly to recurring stressors or threats, even when stressors or threats are not present in the immediate environment. Frequent and sudden activation of the stress response can be uncomfortable and deleterious to health. The mammalian dive response (MDR) is another evolutionary adaptation that functions to promote survival by conserving oxygen when submerged underwater, and like the stress response, it disrupts the homeostatic environment. However, the physiological reactions induced by the MDR are mostly mediated parasympathetically and promote dramatic slowing of the heart rate, while the fight or flight response has opposite physiological effects. Frequent activation of the MDR and its parasympathetic components, such as bradycardia and vagus nerve stimulation, might inhibit sympathetic nervous system (SNS) factors involved with stress and may condition lasting beneficial changes of parasympathetic physiology that protect against the deleterious SNS mechanisms involved in prolonged stress and PTSD.

## **Background**

Posttraumatic stress disorder (PTSD) is a major mental health concern that affects approximately 7-8 % of people during their lifetime, and about 20% of military veterans [1]. PTSD manifests after individuals experience traumatic stimuli or life-threatening events [1-3]. The usual symptoms of PTSD involve physiological hyperarousal, recurrent distressing dreams, negative emotional alterations, and avoidance of thinking about the causative traumatic experiences [1-4]. Traditional treatment modalities consist of serotonin reuptake inhibitors [3] and cognitive behavioral therapy [5]. The non-response rates for these modalities are as high as 20-40% [3] and 50% [5], respectively. Combination therapy consisting of pharmacotherapy and psychological therapy has not been found to be effective [6]. Interestingly, yoga and stimulation of the vagus nerve have been shown to be promising therapeutic interventions for PTSD [7].

While PTSD itself is a disorder, the stress physiology involved in PTSD is considered to be an evolutionary mechanism that makes use of the fight or flight response and functions by reinforcing organismal fear, hypervigilance, and threat avoidance, after experiencing a real or perceived threat [8-11]. Pertinent to the scope of the hypothesis that will be proposed in this article, the stress response is partially characterized by hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis, amygdala, and sympathetic nervous system (SNS)[4,12]. Physiological changes brought on by the stress response and in PTSD include increased heart rate, increased blood pressure, and changes in blood flow to organs needed for the fight or flight response [4]. Upon experiencing a threat, amygdala processing of fear typically stimulates the HPA axis and the release of catecholamines that lead to increased heart rate [4]. Innervation of the sympathetic nervous system constricts intestinal sphincters, reduces blood flow to the gut, and can affect the microbiota-brain-gut-axis [13], which can influence anxiety-like behaviors [7].

Vagal tone, a measure of parasympathetic control over heart rate, is an important variable involved in stress and PTSD [7,14,15]. Low resting vagal tone has been associated with deficits in numerous aspects of emotion regulation [15], while high resting vagal tone has been found to protect against internalizing psychopathology [14]. High vagal tone is also associated with improved physiological recovery following stressful events [15]. The vagus nerve is another important variable involved in PTSD, and is considered to be the primary component of the parasympathetic nervous system. Symptoms of PTSD are partially mediated by the vagus nerve and diminished parasympathetic activity among PTSD patients has been noted [7]. Stimulation of the vagus nerve has been shown to enhance the extinction of conditioned fear in rat models of PTSD. It has been suggested that enhanced fear extinction through vagus nerve stimulation in rats is robust and long lasting [16]. Some evidence shows that vagus nerve stimulation not only reduces anxiety in rats, but in humans as well [16]. The adaptive nature of the stress response can be beneficial for overcoming

short-term threats [17], but when activation of the stress response is prolonged or manifests as PTSD, it is linked to a plethora of deleterious health outcomes, such as heart disease, stroke [1], autoimmune disorders [17], depression [18], increased risk of suicide [3], and addiction [7].

The mammalian dive response (MDR) is also a well-documented evolutionary adaptation that is shared by all vertebrates [19] and mammals [20] studied to date. When the human face is submerged in water and apneic, receptors in the nostrils and on the face elicit biological responses to conserve oxygen [19-22]. These responses consist of parasympathetically mediated bradycardia (slowing of the heart rate), vasoconstriction, and the redirection of blood flow to the brain and core [19-22]. Interestingly, water temperature is important for eliciting a strong MDR. Temperatures from 0-10°C elicit more pronounced bradycardia, while temperatures from 15-35°C have little effect [23]. Cold facial immersion (CFI) exacerbates the MDR through stimulation of cold receptors on the upper facial region [23,24] that are connected to the trigeminal nerve [25]. When stimulated, the trigeminal nerve inhibits respiration and activates vasomotor centers and cardiac vagal motor neurons [25]. In addition, a number of physiological differences have been observed in trained free divers compared to non-trained individuals, both during [26], and after [27,28] apneic submersion activities.

It has been observed that trained free divers showed a more pronounced slowing of the heart rate during MDR compared to non-trained divers, where the heart rate of trained breath hold divers can decrease by more than 50% [23,29]. Increased size of heart chambers [30] has been observed in breath hold divers and higher cardiac parasympathetic tone was exhibited among free dive athletes compared to an untrained control group, even four days after the last dive [27]. Some long term adaptations noted among trained breath hold divers are reduced blood acidosis, increased hemoglobin, larger lungs, increased vital capacity, and increased blood flow to the brain [28]. The physiological adaptations observed among apnea trained divers compared to non-apnea trained divers are considered to be the result of hypoxic conditioning during MDR [28]. Furthermore, an interesting study that investigated the effects of diving on arterial elasticity, function, and structure of female Japanese pearl divers (Ama divers), found that the Ama divers displayed lower arterial stiffness, lower arterial wave reflection, and higher subendocardial perfusion compared to non-Ama [31]. The adaptations mentioned above offer benefits to breath hold divers by allowing them to increase the amount of time they can spend underwater. Although extensive research into the long-term cardiovascular effects of freediving has yet to be produced, available research suggests that the adaptive changes in physiology induced by MDR and CFI training can be conditioned and can offer favorable health outcomes.

In summary, many of the physiological responses involved in the MDR are counteractive to the physiological responses induced by the fight or flight response and that are seen in PTSD. The function of the SNS is to arouse the body for fight or flight [4], while the parasympathetic nervous

system, induced to a dramatic effect by CFI and MDR, functions to inhibit the SNS, reduce heart rate, and promote recovery after experiencing stressors [15].

### **Presentation of hypothesis and implications**

I propose that because the MDR is an evolutionarily potent adaptation that utilizes parasympathetic nervous system components that have opposite physiological effects compared to the SNS pathways that mediate the fight or flight response, repeated induction of the MDR might condition aspects of the parasympathetic nervous system (via vagus nerve stimulation) to become more pronounced, such as lower heart rate, and high vagal tone, thus overriding symptoms of PTSD. Consistent with this hypothesis, are the findings that vagus nerve stimulation in PTSD patients can decrease activity in neural fear processing circuits [7], that high resting vagal tone is associated with adaptive emotion regulation[15], and that trained free divers exhibit high vagal tones[27]. The hypothesis that these changes can be conditioned is based on studies that report lingering physiological adaptations among trained breath hold divers [24,27,28,30]. Activation of the MDR and CFI have been shown to be effective at thwarting supraventricular tachycardia [21], and promoting calming effects in individuals with panic disorder [23], however, the study involving panic disorder was not longitudinal. Furthermore, CFI and the MDR have not been tested with PTSD or other anxiety-like conditions.

This hypothesis can be tested by recruiting participants diagnosed with PTSD and having them follow a regimen of sessions that induce the MDR by CFI in 10°C water while holding their breath until they reach the “struggle stage” [32] of apnea, where breath holders experience involuntary breathing movements, followed by the “breaking point” [32], where the urge to breath can no longer be resisted. The rationale behind this recommended duration for breath holding is based on prior experimental studies [32] and on evidence that suggests longer durations of apnea are correlated with heart rate reduction [33]. Furthermore, trained free divers who practice frequently and who regularly hold their breath into the “struggle stage” and beyond, show lasting physiological adaptations [27,28,30] that are presumed to be the result of hypoxic conditioning[28], and that may be beneficial to those suffering from PTSD. It has been reported that intensive breath hold divers train from 5-6 hours per day, 6 days a week, for 6 months [34]. With the goal of invoking MDR in order to achieve conditioned changes in PTSD patients similar to the adaptations exhibited by trained free divers, and taking into consideration time constraints of volunteers, sessions should be done 1-2 hours per day, 5 days per week, for 6 months. Deep inhale and slow exhale diaphragmatic breathing should be practiced from 2-3 minutes between breath hold sessions to promote recovery and further stimulate the vagus nerve. Longitudinal study designs with follow up occurring 6 months and 1 year after cessation of MDR therapy is recommended due to incidences of relapse that are common among PTSD patients.

It is important to note that activation of the sympathetic and parasympathetic nervous systems at the same time could result in what is known as autonomic conflict, and can produce cardiac arrhythmias [20]. While these arrhythmias are generally benign, participants should be screened for any conditions that might lead to complications, such as water phobias, breath holding phobias, claustrophobia, heart disease, and cardiac rhythm abnormalities.

This hypothesis might also be tested by investigating resilience to PTSD and anxiety disorders among groups of people who frequently submerge their faces underwater and hold their breath for extended periods of time, such as sport free-divers, sport spearfisherman, and maritime based ethnic minorities who rely on free-diving for subsistence. Furthermore, the neuroendocrinology of the MDR remains not fully understood. It would be useful to examine if stress hormones characterized by the stress response and PTSD are inhibited while mammalian dive response is activated. This could be done by comparing hormone levels pre, during, and post MDR therapy.

If MDR therapy can thwart symptoms of PTSD in the manner proposed in this hypothesis, not only would a low- cost treatment for PTSD be discovered, but the non-response rate would likely be low due to the strength and reliability of evolved autonomic functions to react to evolutionarily familiar stimuli, such as submersing the face in water. Based on findings from the Ama divers study, MDR could also play a role in protecting against common comorbidities of PTSD, such as hypertension and heart disease.

**Funding:** None to declare.

**Author Contributions:** The author was responsible for conceptualization, writing, editing, and searching for relevant peer reviewed research.

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