EXHIBIT 4

Expert Report
of
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Geisel School of Medicine at Dartmouth College

Suleiman Abdullah Salim, et al. v.
James E. Mitchell* and *John Jessen
E.D. Wash. No. 15-0286-JLQ

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TABLE OF CONTENTS

I. BACKGROUND AND QUALIFICATIONS

I am a Medical Doctor and am Board Certified in Psychiatry. I also have a Ph.D. in Pharmacology. For more than 24 years, I served as the Executive Director of the United States Department of Veterans Affairs' National Center for Posttraumatic Stress Disorder ("PTSD"). In December 2013, I stepped down from this position to become a Senior Advisor to the Center. Currently, I am a Professor of Psychiatry at the Geisel School of Medicine at Dartmouth College, where I have taught for 43 years. I have over 40 years' experience as a practicing clinician in psychiatry. I am an accomplished researcher with approximately 300 publications and 27 books including textbooks. In addition, I am a Distinguished Lifetime Fellow of the American Psychiatric Association ("APA"), past-president of the International Society for Traumatic Stress Studies ("ISTSS"), past-chair of the APA's DSM-5 PTSD Work Group, and past chair of the Scientific Advisory Board of the Anxiety Disorders Association of America. I have served on many national research, education, and policy committees and boards of peer-reviewed journals. My past honors include the ISTSS Lifetime Achievement Award in 1999 and ISTSS Public Advocacy Award in 2009. I was a finalist for the 2011 Samuel J. Heyman Service to America Medal. My current curriculum vitae, containing a full list of my qualifications, is attached as Appendix A.

During the past four years, I have been deposed twice and testified once at trial. In *Rogers v. Boeing Aerospace Operations, Inc.*, No. 13-1448 (E.D. Mo.), I testified both in a deposition and at the trial. In *Linde v. Arab Bank*, Nos. 04-2799 (E.D.N.Y.), I testified in a deposition, but the case never went to trial.

My fee is $500 for each hour of work (*e.g.*, document review, consultation, independent medical examination, or preparation of this report). I also charge $2,000 for each half day in which I testify in court or for a deposition.

II. OPINIONS

I have been asked to draft a report and potentially testify in the civil action entitled *Salim, et al. v. Michell, et al.*, No. 2:15-cv-286-JLQ in the United States District Court for the Eastern District of Washington regarding particular issues that are within my expertise. Specifically, I have been asked to address:

A) the history of the psychological effects of trauma including the recognition of posttraumatic stress disorder (PTSD) as a discrete mental disorder;

B) the general state of knowledge in 2002 within the mental health community regarding the psychological effects, including but not limited to the risk of PTSD, of intentionally subjecting individuals to traumas or uncontrollable stress; and

C) what the mental health community knew in 2002 about the psychological effects of intentionally inducing and sustaining a state of "learned helplessness.

I will provide a succinct but comprehensive discussion of these issues in which I will rely upon the latest scientific evidence, which can be further detailed in my testimony. I also will rely upon my professional training, clinical practice, and original and peer reviewed research.

A. The history and psychological effects of trauma with specific reference to PTSD

That exposure to trauma may produce enduring psychological consequences has long been recognized. For example, various literary works, dating back to antiquity, such as Homer's *Iliad*, Shakespeare's *Henry IV*, and Dickens's *Tale of Two Cities*, present characters whose psychological transformations and symptoms related to trauma.

In the late 19th century, medical practitioners joined literary artists in beginning to recognize the profound and debilitating psychological impact of military combat among veterans of the U.S. Civil War and the Franco-Prussian War. They were especially impressed by the frequent observation that exposure to war trauma could produce major suffering and incapacitation despite the absence of blood, broken bones or any other overt anatomic or physiological abnormality. As a result, a number of theories were postulated both in the United States and abroad to explain such invisible wounds of war that focused on disturbances of cardiovascular or neuropsychiatric systems. Thus, from the 1860s through the 1980s, various post-traumatic syndromes were identified. Some of these clinical formulations postulated cardiovascular irregularities (*e.g.*, "Soldier's Heart", "Da Costa's Syndrome", "Neurocirculatory Asthenia") while others emphasized neuropsychiatric abnormalities (*e.g.*, "Nostalgia", "Shell Shock", "Combat Fatigue", "War Neurosis") (Cohen et al, 1948; Trimble,, 1985). Similar clinical presentations among 19th-century civilian survivors of train accidents were called "Railway Spine."(Trimble, 1985) (a malady affecting Charles Dickens, among many others). Throughout this period, clinicians providing treatment for survivors of military or civilian trauma were struck by the physiological as well as the psychological symptoms exhibited. Indeed, by the 1940s, Abram Kardiner, an American psychiatrist who worked extensively with World War I veterans suffering from "War Neurosis," was so impressed by their excessive anxiety and jumpiness (i.e., "startle reactions") that he called it a "*physioneurosis*"[11] to capture both the physiological as well as psychological symptoms that he considered key components of the "War Neurosis" syndrome (Kardiner, 1941). More recently, we have identified major biological abnormalities that are central to this disorder, including alterations in brain structure and brain function, described below.

In the United States, approximately half of all Americans will be exposed to at least one traumatic event, such as assault, military combat, an industrial or vehicular accident, rape, domestic violence, or a natural disaster (*e.g.*, an earthquake) (Kessler, *et al.*, 1995). Traumatic exposure is higher for individuals who engage in professions where their work places them in traumatic situations on a regular basis; this includes military personnel, police, firefighters, emergency medical technicians, journalists and others. Exposure to extreme stress is also much higher for people who live in nations subjected to war, state terrorism, torture, or forced migration (as Syria, Algeria, Cambodia, Palestine, Israel, or Iraq). In other words, the greater the likelihood of exposure to traumatic events, the greater the likelihood of developing PTSD. For example, in the U.S where trauma exposure for American adults is 50-60%, approximately 8 percent of Americans develop PTSD. On the other hand, in areas of conflict such as Syria or

Iraq where the likelihood of trauma exposure is 90-100%, PTSD prevalence is 20 to 30 percent (de Jong, et al., 2001; Kessler, et al., 1995).

The strong relationship whereby greater trauma severity (which includes intensity, frequency and duration of trauma) predicts a greater likelihood of developing PTSD, is a fundamental finding in traumatic studies research. The more severe the trauma, the more frequently PTSD occurs (Kulka, *et al.*, 1990). For example, in situations of prolonged severe trauma (such as protracted domestic violence, repeated torture or other abusive treatment during a long incarceration, captivity as a prisoner of war (or other armed conflict), and frequent episodes of child abuse), the risk of developing PTSD is the highest. Also interpersonal trauma or trauma perpetrated against one individual by another (such as attempted murder, rape or torture) is much more likely to produce adverse psychological consequences than non-interpersonal trauma such as a hurricane, earthquake or other natural disaster (Brewin, *et al.*, 2000).

PTSD is the most characteristic psychiatric disorder among individuals who have been exposed to traumatic events. Affected individuals exhibit a characteristic pattern of trauma-related symptoms (discussed below) as well as impairments in everyday functioning that last for at least a month and sometimes for a lifetime. As noted above, this pattern of symptoms has been recognized, albeit by many other names from ancient times through the late twentieth century. It has been called PTSD since the 1980 Revision (the third) of the American Psychiatric Association's (APA) official diagnostic catalog, the Diagnostic and Statistical Manual (DSM-III) Although some of the specific symptoms included in the APA's original 1980 PTSD diagnostic criteria (APA, 1980) have been partially modified over the years (APA, 1987; APA, 1994; APA, 2000; APA 2013), the fundamental PTSD construct – that exposure to catastrophic psychological stress can produce severe, debilitating and long-lasting psychological distress and impairment – has clearly withstood the test of time. As a result, clinicians have by now had at least 36 years with which to utilize PTSD as a diagnostic tool and to develop effective treatments. By 2002, any qualified mental health professional would have been expected to know not only that PTSD existed, but the diagnostic criteria for PTSD, the definition of a traumatic event, the circumstances under which PTSD is most likely to develop, the circumstances that would reliably exacerbate PTSD symptoms, and how to treat the disorder. In the following discussion, we will focus on the DSM-IV-TR PTSD diagnostic criteria (APA, 2000) because they were in effect in 2002. The current DSM-5 criteria (APA, 2013) are the latest revision of all American psychiatric diagnostic criteria. With regard to PTSD, all 17 DSM-IV-TR symptoms re-appear in DSM-5 although some have been modified due to new scientific findings between 2000 and 2013. As stated previously, however, the basic PTSD construct has been preserved since the DSM-III was first published in 1980 (APA, 1980). .

1. Definition of a traumatic event in DSM-IV-TR

Trauma is defined as a catastrophic event (or series of events) in which an individual has been personally threatened, exposed to, or witnessed death, physical harm, or sexual violence. Traumatic events also include indirect exposure in which an individual, who is never in personal danger, is confronted by the news that a loved one has been exposed to trauma (e.g., murder, assault, rape, torture, combat, etc.). These three manifestations of a traumatic event are set forth as Criterion A of the PTSD diagnostic criteria in the DSM-IV-TR edition of APA's diagnostic catalog (APA, 2000).

In addition to exposure to such life-threatening or potentially harmful events,(Criterion A1) individuals who met DSM-IV-TR's Criterion A must also have experienced severe psychological distress, defined as "fear, helplessness or horror" (Criterion A2). In other words, both of Criteria A1 and A2 had to be met for exposure to a catastrophic event to be considered a Criterion A "traumatic event" according to DSM-IV-TR.

2. The DSM- IV-TR criteria for PTSD

All medical diagnoses are revised periodically with advances in science and clinical experience and the same has been true with regard to PTSD. Thus, ever since PTSD first appeared in the DSM-III in 1980, its diagnostic criteria have been carefully considered in each subsequent revision of the DSM, which is recognized by the American mental health community as the authoritative source for diagnosing mental disorders. In this process, which is well known to all practitioners in the field, published scientific research concerning the PTSD criteria are rigorously evaluated and field trials carried out to obtain pertinent data. The DSM-III-Revised (DSM-III-R was published in 1987,(APA, 1987), DSM-IV was published in 1994 (APA, 1994), DSM-IV-TR in 2000 (APA, 2000) and the DSM-5 in 2013 (APA, 2013). In all DSM revisions, strong evidence was required for any change in any diagnostic criterion. Although there have been changes in the PTSD diagnostic criteria from one DSM to the next, the basic construct, that exposure to a psychologically traumatic event can produce a reliable pattern of symptoms that cause extreme distress and severe incapacity, has withstood the test of time and each iteration of APA's rigorous DSM review process. In 2002, the DSM-IV-TR was in effect. By that time, PTSD had been an official diagnosis for 22 years and American mental health practitioners had had extensive experience with its diagnosis and treatment.

Indeed, as of 2002, PTSD was (as it remains today) the most common psychiatric disorder following exposure to a traumatic event. As noted previously, it is caused by exposure to a severely stressful traumatic event. In addition, the person must exhibit a pattern of symptoms included within three symptom clusters: re-experiencing; avoidance/numbing; and hyperarousal — as well as meeting the duration and functional impairment criteria. The Fourth edition-text revision of Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), which was published by the American Psychiatric Association (APA) in 2000, provided the criteria for PTSD in 2002 (APA, 2000). More specifically, an individual diagnosed with PTSD under DSM-IV-TR PTSD is described utilizing criteria in six clusters, Criteria A-F, which are summarized as follows:

> **Criterion A**, which includes A1 **(**Exposure to actual or threatened death, serious injury, or sexual violence**,** which, as discussed, might include direct exposure/ witnessing or indirect exposure by being "confronted" by news that a loved one was thus exposed), and A2 (That this exposure is associated with severe emotional distress, *i.e.*, "fear helplessness or horror,")
>
> **Criterion B (**The traumatic event is persistently experienced, in that the individual experiences at least one of the following 5 symptoms: intrusive recollections; traumatic nightmares; PTSD flashbacks; psychological stress when exposed to traumatic memories/reminders; or physiological arousal when exposed to traumatic memories or reminders).

Criterion C (Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness, whereby the individual experiences 3 of the following 7 symptoms: avoiding trauma-related thoughts or feelings; avoiding trauma-related activities, places or people; amnesia for trauma-related memories; diminished interest or participation in significant activities; feeling of detachment or estrangement from others; restricted range of affect, *i.e.*, loss of the normal range of emotional expression, also characterized as "emotional numbing"; or sense of foreshortened future, *i.e.*, the belief that one has no future to look forward to with regard to family, career, and lifespan.

Criterion D (Persistent symptoms of increased arousal, whereby the individual experiences 2 of the following 5 symptoms: insomnia; irritability; **d**ifficulty concentrating; hypervigilance or hyperarousal due to fears about personal safety, *i.e.* being watchful and "on guard" all the time; or exaggerated startle reaction, *i.e.*, being "jumpy" and exquisitely sensitive to unexpected noises.

Criterion E (Duration of the disturbance, whereby the symptoms in Criteria B, C and D last more than 1 month).

Criterion F (The disturbance causes clinically significant distress or impairment in social, occupation, or other important areas of functioning).

Criterion G (The disturbance is not attributable to the physiological effects of a substance (*e.g.*, medication, alcohol) or another medical condition.

It should be noted that PTSD can occur "with delayed expression," *i.e.*, it may be that, although the onset and expression of some symptoms may be immediate, the full diagnostic criteria are not met until at least 6 months after the event. It should also be noted that, by 2002, there was growing interest in people who lack one or two of the three mandatory DSM symptoms, especially after publication of the National Vietnam veterans Readjustment Study in 1990 (Kulka, *et al.*, 1990). A number of studies have shown that such individuals have clinically significant, post-traumatic symptoms and functional impairment even though they do not meet diagnostic criteria for PTSD (Schnurr, *et al.*, 1993; Weiss, *et al.*, 1993). In 2002, under DSM-IV-TR, the appropriate diagnosis for subthreshold PTSD would have been Anxiety Disorder Not Otherwise Specified (NOS).

A brief word about DSM-5 is in order. I have focused on DSM-IV-TR since it sets forth the diagnostic criteria in effect on 2002. I was very involved in the next DSM revision for PTSD, including that I chaired the DSM-5 Trauma/Stress Workgroup between 2008 and 2013 when DSM-5 was finalized and published (APA, 2013). As stated previously, the basic DSM-III construct for PTSD was preserved, validating that exposure to catastrophic and uncontrollable stress can precipitate a characteristic pattern of severe and incapacitation symptoms that may persist for decades or even a lifetime. That said, DSM-5 reformulated PTSD as a syndrome that can produce a broad spectrum of symptoms, in addition to the stress-based anxiety syndrome explicated in DSM-III and IV. Indeed, DSM-5 recognized that posttraumatic psychopathology can also exhibit prominent depressive, behavioral and cognitive symptoms that do not conform to the narrow anxiety disorder described in DSM-III and DSM-IV. As a result, PTSD was

moved out of the Anxiety Disorders diagnostic cluster and into a category of its own, Trauma and Stress-Related Disorders. Nonetheless, the 17 DSM-IV symptoms remained in DSM-5, though a few other symptoms regarding negative emotions, self-blame and reckless behavior were added (Friedman, 2013). These changes, while reflecting our greater understanding of PTSD, do not in any way undermine my opinions set forth here, including that a mental health professional would have understood the causes and effects of PTSD in 2002, discussed in this report.

3. What is it like to have PTSD?

To put all of this into perhaps more understandable terms, people with PTSD are consumed by concerns about personal safety. They persistently scan the environment for dangerous stimuli. When in doubt, they are more likely to assume that a threat is present and will react accordingly (and sometimes inappropriately). Intrusion and Arousal and Reactivity symptoms, described below, can be understood within this context. The primacy of traumatic over other memories (*e.g.*, the Intrusion symptoms) can also be understood as a pathological exaggeration of the normal human response to dangerous encounters.

People with PTSD exhibit sustained anxiety about potential threats to life and limb, a pervasive and uncontrollable sense of danger, and a preoccupation with concerns about the personal safety of oneself and one's family. The intensity of this emotional reaction provokes avoidant or protective behaviors in an effort to reduce the emotional impact of the stimulus. Stimuli reminiscent of such traumatic events (*e.g.*, seeing someone who resembles the original assailant, confronting warzone reminders, or being exposed to high winds or torrential downpours reminiscent of a hurricane, etc.) evoke similar conditioned responses manifested as fear-induced avoidant and protective behaviors. (*See* DSM IV-TR and DSM-5 (APA 2000, 2013).

These symptoms reflect the persistence of thoughts, feelings, and behaviors specifically related to the traumatic event. Such recollections are intrusive because they are not only unwanted but are also powerful enough to negate consideration of anything else. Daytime recollections and traumatic nightmares often evoke panic, terror, dread, grief, or despair. Traumatic reminders can also evoke intolerable trauma-related emotions such as fear, anger, guilt, and shame and physiological reactions such as racing pulse, rapid breathing, or sweating.

Sometimes when people with PTSD are exposed to traumatic stimuli they are suddenly thrust into a psychological state — the PTSD flashback — in which they imagine or relive a traumatic experience, losing all connection with the present.. It appears that PTSD flashbacks are an expression of an extremely heightened level of arousal associated with excessive release of adrenaline (*e.g.*, norepinephrine) throughout the body, as is discussed below.

Avoidance/Numbing symptoms can be understood as behavioral or cognitive strategies used to ward off the terror and distress caused by Re-experiencing and Hyperrousal symptoms. Avoidance symptoms include efforts to avoid thoughts, feelings, activities, places, and people related to the original traumatic event. For example, if a man who works on the 34th floor of a high-rise office building developed PTSD after he was severely assaulted in an elevator, he may be unable to enter an elevator again. Or a woman who was severely raped may find herself unable to have sexual relations with her husband.

The five numbing symptoms in the Avoidance/Numbing cluster are, in part, psychological mechanisms through which PTSD sufferers anesthetize themselves against the intolerable panic, terror, and pain evoked by Intrusion symptoms. These include constricted emotional states, in which the person is unable to experience positive emotions or feelings such as love, enjoyment or friendship. Marriages, family attachments and friendships are often a major casualty of PTSD because of such anesthetizing of loving feelings necessary to sustain an intimate, loving relationship. Other symptoms in this cluster cause reduced engagement with the world and social isolation because of amnesia, diminished interest or participation in significant activities and feeling detached or estranged from others. People with PTSD have lost the bright future they once expected (*e.g.*, relationships, career, lifespan); they feel that their life has been ruined, that they have nothing to look forward to, that the world is a dangerous place, and that they are unable to cope with the normal demands of life with which they were very competent in dealing with in the (pre-traumatic) past. Perhaps most devastatingly, they believe that they are no longer the people they used to be and will never again have the life they enjoyed before the trauma. People with PTSD are usually very unhappy. They see themselves as forever changed, weak and powerless. They are full of self-blame and self-doubt. Not only are they unable to experience positive emotions but they continually experience negative emotions such as sadness, guilt, shame, fear and anger. In some cases, the combination of negative mood and cognitions, social isolation and lack of hope for the future may lead to suicide.

Arousal and Reactivity symptoms are the most apparent manifestations of the excessive physiologic arousal that is part of the PTSD syndrome, and include insomnia, irritability, startle reactions and hypervigilance. Hypervigilance is the excessive preoccupation with danger and perceived vulnerability mentioned previously. Hyperarousal symptoms make up a hyper-reactive psychophysiological state that makes it very difficult for people with PTSD to concentrate or perform other cognitive tasks. For example, PTSD may impair the capacity to focus on intellectual tasks at school, work, or home. This excessive arousal and reactivity can also, sometimes, lead to irritable behavior.

PTSD is no different from other medical or psychiatric disorders in that its severity may range from mild to severe. As with diabetes, heart disease, and depression, some people with PTSD can lead full and rewarding lives despite the disorder. Although there are no current statistics, it appears that a significant minority of patients may develop a persistent, incapacitating mental illness marked by severe and intolerable symptoms; marital, social, and vocational disability; and extensive use of psychiatric and community services. Such people can typically be found on the fringes of society, in homeless shelters, or enrolled in public sector programs designed for people with persistent mental illnesses, such as schizophrenia (a major psychiatric disorder characterized by disorganization and fragmentation of thought, delusions, hallucinations, apathy, disturbance of language and communication, and withdrawal from social interaction) (Friedman & Rosenheck, 1996).

The long-term course for most people with chronic PTSD is marked by remissions and relapses. Some people fully recover, others partially improve, and others never improve. Our expectation, of course, is that with the development of effective treatments for people with PTSD (or for preventing the development of PTSD among recently traumatized individuals) we can significantly reduce the prevalence of chronic PTSD.

In addition to PTSD per se, there are a number of associated symptoms and conditions that may significantly affect severity and chronicity of this disorder as well as quality of life. This was all well known by 2002. These include:

> Danger to self
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> As noted previously, there is evidence of a positive association between the number of previous traumatic events and the likelihood of a suicide attempt. Furthermore, PTSD often co-occurs with other conditions that are associated with suicidal behavior such as depression, substance use, panic attacks, and severe anxiety (APA, 2000; Kessler, *et al.*, 1995)
>
> Intolerance of ongoing stressors
>
> PTSD patients often exhibit a reduced capacity to cope with the ordinary and predictable challenges of life. Such stressors include marital, familial, workplace or social stressors. Ongoing or secondary stressors are risk factors for the persistence of PTSD (APA, 2000; Brewin, *et al.*, 2000).
>
> Social support
>
> Social support is a powerful protective factor (Brewin, *et al.*, 2000) that includes the capacity of an individual to accept or utilize social support when it is made available. This may be especially problematic in PTSD where symptoms such as avoidance, alienation and detachment impair affected individuals from benefiting from available marital, familial and social support.

PTSD rarely occurs alone. Indeed, 80% of individuals also have at least one other psychiatric disorder. The five disorders most frequently associated with PTSD include: Major Depressive Disorder, Generalized Anxiety Disorder, Social Anxiety Disorder and Simple Phobias, Panic Disorder, and Alcohol/Substance Use Disorder (Kessler, *et al.*, 1995). The best research has been on co-occurring PTSD and major depressive disorder , where it has been shown that symptom severity, chronicity and functional impairment are worse than for either disorder alone. (Brown, *et al*., 2001; Friedman & Yehuda, 1995; Kessler, *et al.*, 1995; Kulka, *et al.*, 1990). Also, treatment is much more complicated when two or more disorders are present rather than PTSD alone. Finally, as noted above, there are a growing number of research findings indicating that PTSD also increases the risk for potentially incapacitating medical problems, as set forth in Schnurr and Green's book, "Trauma and Health" (2004); most if not all of these studies had, it should be noted, been published by 2002.

Among combat veterans, especially from recent wars in Afghanistan and Iraq, it is not uncommon to see individuals with co-occurring PTSD and traumatic brain injuries (TBIs). The latter are usually due to blast injuries from bombs, for example. As discussed previously, combined PTSD and TBI is much more complicated than either one separately, and the combination has a poorer prognosis (Stein & McAllister, 2009).

Again, as noted, with the possible exception of some new findings on TBIs, all of the clinical problems associated with PTSD were well known in 2002. As noted previously, although there

are some differences between DSM-IV-TR (which defined PTSD diagnostic criteria in 2002) and the current DSM-5 criteria, all 17 DSM-IV criteria were retained in DSM-5.

4. What happens to the brain and the body of people with PTSD after trauma?

In the normal human stress response (Friedman, 2015), the amygdala, the brain nucleus that has evolved to detect danger and to rapidly mobilize protective behaviors, becomes acutely activated in reaction to threat. It sets off neuronal alarms that mobilize the many key neurobiological systems that have evolved for preservation of the species, including the most famous threat-related behavior, the Fight or Flight Response, first described by Walter Cannon in the 1920s (Cannon, 1932), in which the organism flees to safety or fights to fend off the aggressor. Later, when the danger has passed, the amygdala in a healthy brain returns to its normal baseline functioning and resets brain activation to the same levels as those seen during the safe period preceding the emergence of the dangerous threat. A major problem in PTSD, however, is that. neither the amygdala nor any other part of the brain can properly recognize safety signals indicating that the danger has passed – it cannot tell the difference between *bona fide* danger signals and neutral or safety signals. Therefore the brain remains in a state of excessive arousal, hypervigilence or high alert (sometimes for decades) that produces many of the emotional, cognitive and behavioral suffering and disability caused by PTSD. All of this was well understood in 2002. (*See* Friedman, Charney & Deutch, 1995; Kolb, 1989; Le Doux, 1995).

Since the human stress response plays a key role in the preservation of the species, it should come as no surprise that many psychobiological systems are involved. Given, that PTSD is fundamentally a dysregulation or abnormality of this complex response, it is also not surprising that abnormal function of many key biological systems has been detected in PTSD. A review of this massive emerging literature is far beyond the scope of this discussion. Although new discoveries keep getting published, it was well known in 2002 that key components of the human stress response were altered in PTSD patients. For example, it was well known that the adrenergic system (characterized by biobehavioral mechanisms facilitated by the neurotransmitters norepinephrine and epinephrine which mediate the Fight or Flight Response) was greatly disrupted in PTSD patients. Also the neurohormal stress response system involving brain and glandular hormones such as corticotropin releasing factor (CRF), adrencorticotrophic hormone (ACTH) and the adrenal hormaone, cortisol, were well known to function abnormally in PTSD. A good summary of all of these scientific findings can be found in Friedman, Charney and Deutch's, 1995 book, "Neurobiological and Clinical Consequences of Stress: from Normal Adaptation to PTSD" which was the first comprehensive synthesis of psychobiological findings that were relevant to PTSD. Finally, brain imagers in the mid-1990s had shown that PTSD patients exhibited structural anatomic abnormalities in a key brain region, the hippocampus (Bremner, et al., 1995). They also demonstrated major abnormalities in the activation of the several brain regions that comprise the brain's fear circuit (*e.g.*, amygdala, hippocampus, cingulate cortex and prefrontal cortex) (Rauch, *et al.*, 1996). In short, these abnormalities in brain structure and function were so well understood by 2002 that the National Association for Mental Illness (NAMI) recognized PTSD as a major mental illness along with depression and schizophrenia (*See* <www.nami.org>).

Finally, evidence had begun to accumulate by 2002 indicating that exposure to catastrophic

events and the occurrence of PTSD risk factors for many medical disorders affecting the cardiovascular, gastrointestinal, endocrinological, musculoskeletal, and other bodily systems (Friedman and Schnurr, 1995; Schnurr and Jankowski, 1989; Schnurr & Green, 2004).

5. Other posttraumatic mental health symptoms include anxiety, depression, dissociation, hypervigilance, flashbacks, substance abuse, etc.

As noted above, there is growing evidence that PTSD may not be the only clinically significant consequence of exposure to a catastrophic event. Other types of post-traumatic outcomes potentially include depression, alcoholism, other psychiatric disorders and medical problems, as noted above.

6. Research on PTSD: The relationship between inescapable stress/learned helplessness and traumatic events.

All traumatic events are not the same. They range from a brief moment of helplessness , fear or horror (as being in dangerously close proximity to a homicide attempt, suddenly finding oneself in the path of a life-threatening tornado, or being a passenger in a vehicular head-on collision) to a protracted experience in which an individual is repeatedly traumatized and from which there is no escape. There are many examples of this scenario such as long-standing domestic violence, hostage situations, torture and other prolonged or repeated abuse during a long incarceration, and being caught in a war zone (as either a combatant or bystander). What all of these traumatic (*i.e.*, PTSD Criterion A) events have in common is that the individual has no control over such life-threatening and potentially harmful events and that there is no way to escape from such situations.

Scientists attempting to create traumatic situations in the laboratory have come to agree that the essential common element in a PTSD Criterion A event is that the individual is helpless to control such life-threatening and potentially harmful events. Indeed, since 1980, the inability to successfully cope with uncontrollable traumatic events has been understood to be a fundamental scenario in which individuals develop PTSD. Examples of animal laboratory paradigms for uncontrollable stress include inescapable shock where animals in an enclosed space receive painful shocks whenever the experimenter chooses to shock them. Another example is a forced swim task where animals are thrown into a pool from which escape is impossible. Animals subjected to these inescapable laboratory situations exhibit fearful, immobilization/freezing, struggling, and/or apathetic behaviors. Neurobiologically they exhibit initial outpourings of norepinephrine, cortisol, and other neurohormones/neurotransmitters/neuropeptides often followed by depletion of these key components of the stress response (Anisman & Zacharko, 1986; Abercrombie & Jacobs, 1987; Charney, *et al.*, 1995; Davis, 1986; Weiss, *et al*., 1981). Therefore, research in which uncontrollable stress parameters are manipulated by the experimenter has been a major laboratory model that has influenced our theoretical understanding of PTSD and has also provided a major experimental paradigm for preclinical and clinical research that has advanced our understanding of this disorder (see below).

Research on uncontrollable stress dates back to the mid-twentieth century (Masserman, 1943; Mowrer, 1960). In one particularly notable paradigm, Martin Seligman subjected beagles to an

uncontrollable stress situation (*e.g.*, repeated, inescapable electric shocks, with no ability to escape). He observed that the beagles would sit passively, and not attempt to escape, when subsequently shocked in circumstances, even when there was a chance to escape. This phenomenon was called "learned helplessness" (Seligman & Mair, 1967).

By the 1980s, theoretical papers began to appear suggesting that learned helplessness due to inescapable and/or unpredictable stress, was an excellent animal model for the traumatic stress experienced by PTSD patients (Foa, Steketee & Rothbaum, 1989; Keane, Zimmerling & Caddell, 1985). Peterson & Seligman (1983) drew a parallel between learned helplessness and victimization (*i.e.*, feelings of adversity –including fear, helplessness and horror - due to poor treatment, fraud, cheating or power inequality) since both are precipitated by uncontrollable aversive events. Indeed, victimization is a well-recognized traumatic scenario known to precede the onset of PTSD among affected individuals. Finally, Seligman and co-workers extrapolating from laboratory-induced responses to learned helplessness in humans, suggested that humans can develop similar patterns of helplessness including loss of mastery, social isolation passivity and dysphoric affect – a profound unhappiness, unease or dissatisfaction. (Garber and Seligman, 1980). As noted previously, uncontrollable stress is an excellent characterization of many traumatic events among humans such as rape, torture or other prolonged abuse, such as child abuse, war and natural disasters. The theoretical and experimental work based on uncontrollable stress/learned helplessness paradigms has contributed greatly to advancing our understanding about how exposure to these types of traumatic events will result in PTSD.

A seminal paper in 1985 (van der Kolk, *et al.*, 1985) extended such psychological theory regarding learned helplessness to psychobiology and suggested that the biological alterations observed among laboratory animals exhibiting learned helplessness might explain alterations in brain mechanisms associated with PTSD. Indeed, in 1991 an entire issue of the National Center for PTSD's periodical, the PTSD Research Quarterly (Bremner, Southwick & Charney, 1991), reviewed the burgeoning literature on biological alterations associated with learned helplessness and concluded that psychobiological changes observed in laboratory animals, mirrored those observed among patients with PTSD. In particular, with the extension of these studies to the rat, there originated an extensive area of research on the neurochemical and behavioral consequences of uncontrollable stress (Seligman and Beagley, 1975).

"Originally considered as an animal model for depression, inescapable stress has proven to be a useful model for the study of reactions to traumatic stress (Telner & Singhal, 1984). … Inescapable stress … [is associated with] a massive output of norepinephrine, dopamine and opiates in specific brain regions. Inescapable stress is associated with impairments in learning and memory manifested by deficits in maze escape behaviors. These deficits appear to be related to alterations in brain structures involved in memory including temporal lobe, amygdala, and hippocampus (Squire & Zola-Morgan, 1991). Inescapable stress is also associated with conditioned fear or the development of fear responses following exposure to the source of trauma (Davis, 1986). A clinical example would be the anxiety and fear invoked in a rape vicim upon returning to the site of the rape. These changes may persist for extended periods beyond the initial stress, resulting in long-term behavioral disturbances and an inability to respond adequately to subsequent stressors." (Bremner, Southwick & Charney, 1991, page 1).

The above example of the intense fear and anxiety evoked in a rape victim exposed to the

original site of the rape, is observed as explicit symptoms of PTSD (B4 and B5) where exposure to a traumatic reminder evokes intense psychological distress and physiological reactivity. (*See* above).

Finally in the National Institute of Mental Health's (NIMH) classic book, The Mental Health Consequences of Torture (Gerrity, *et al.,* 2001), learned helplessness is cited as a major psychosocial model to explain the psychololological impact of torture. These include dysphoria, passivity, decrements in performance on basic tasks, and highly generalized beliefs about personal lack of control over future events (Fairbank, Friedman & Basoglu, 2001).

B. The general state of knowledge in 2002 within the mental health community regarding the psychological effects, including but not limited to the risk of PTSD, of intentionally subjecting individuals to traumas or uncontrollable stress.

By 2002, mental health professionals had been treating patients and conducting intensive research on PTSD for over 20 years. Consequently, there was a great deal known about the cognitive, emotional and behavioral consequences of PTSD. Furthermore, all of the aforementioned literature on learned helplessness/uncontrollable stress, the utilization of learned helplessness experimental paradigms in PTSD research, and the applicability of learned helplessness laboratory paradigms to human traumatic experiences were well known and well accepted within both the scientific and clinical communities.

Torture had been specifically identified as a traumatic event in the original 1980 DSM-III PTSD Criterion A diagnostic criteria and had remained categorized as such in all subsequent revisions. Torture is actually an excellent exemplar of an uncontrollable stressor with a high likelihood of inducing PTSD. Let's consider the variety of torture methods relevant to the current litigation (based upon the Complaint, which is the only document that I have considered, other than my research, cited here, in the preparation of this Report). They include: solitary confinement; extreme darkness, cold, and noise; repeated beatings; starvation; excruciatingly painful stress positions; prolonged sleep deprivation; confinement in coffin-like boxes; and water torture.

For example waterboarding was first used in Spain in the 1500s. It is a form of water torture in which water is poured over a cloth covering the face and breathing passages of an immobilized captive causing the sensation of drowning. Waterboarding can cause extreme pain, dry drowning, damage to lungs, brain damage from oxygen deprivation, and other physical injuries including broken bones due to struggling against restraints, lasting psychological damage and death. Waterboarding induces panic and suffering by forcing a person feel that they are drowning due to the sensation of water inhaled into the sinuses, pharynx, larynx and trachea. Adverse physical consequences due to waterboarding can last for months after the event while psychological effects can last for years.

Given, our previous discussion, being forcibly restrained during waterboarding is clearly an uncontrollably stressful situation. The captive is compelled to experience a frightening, potentially life-threatening or harmful event in which he is powerless to prevent the behavior of his captors. There can be no question that such an event easily meets the DSM definition of a traumatic event that has the potential to induce PTSD. Furthermore, the likelihood that PTSD

will be a consequence of waterboarding, increases each time a captive is subjected to a subsequent waterboarding episode. This was well known in 2002 and was explicitly addressed in the NIMH book on the psychological consequences of torture that was published the previous year (Gerrity, Keane & Tuma, 2001).

What applies to waterboarding also applies, in great measure not only to water dousing, that approximates water boarding, but also to solitary confinement; extreme darkness, cold, and noise; repeated beatings; starvation; excruciatingly painful stress positions; prolonged sleep deprivation; confinement in coffin-like boxes: they all are different forms of uncontrollable stress that meet PTSD's Criterion A (in all DSM editions, since DSM-III was first approved in 1980). In other words all of these methods of torture, as described in the Complaint are potentially traumatic events with a high risk of precipitating PTSD (Gerrity, *et al.*, 2001).

C. The psychological effects of intentionally inducing and sustaining a state of "learned helplessness" as they were known by the mental health community in 2002.

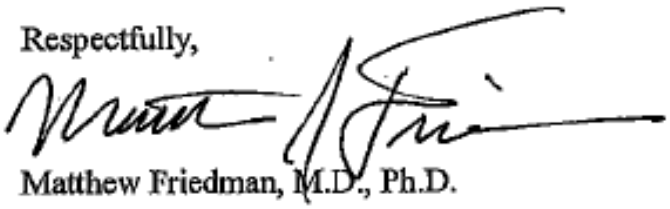
There is little to add to the answer to the previous question in this regard except that by 2002, the PTSD diagnosis had been used in everyday clinical practice and been the subject of intense research for 22 years. There had never been any controversy regarding torture as a traumatic event. Torture had been stipulated as a traumatic event since 1980 and as such, was well recognized as a PTSD Criterion A event with the potential to produce PTSD.

Whether or not uncontrollable stress might produce learned helplessness was originally an inference from research with rats, cats and dogs that seemed to apply to humans who had been subjected to uncontrollable traumatic circumstances (as in Nazi concentration camps, Japanese prisoner of war camps, hostage situations, and situations of domestic violence). (See previous discussion). Although more applicable to PTSD, learned helplessness had also been considered an animal model for depression.

From my perspective, the question of whether torture and captivity produces learned helplessness is a theoretical issue that has not been completely settled. To me the more important question is whether the uncontrollable stress produced by torture and captivity – which one can imagine is only worse when the purpose is to produce learned helplessness, in the way that the Complaint here describes – was likely to precipitate PTSD. Here there is ample evidence from both animal research and human traumatic experiences that such traumatic episodes were extremely likely to produce PTSD. Furthermore, with repeated episodes of torture that likelihood increased towards certainty. In other words, any mental health professional, in 2002, should have known that one or more episodes of torture were very likely to cause PTSD as well as severe physical pain to any individual subjected to such a procedure. This is stated clearly in every edition of the DSM PTSD criteria since 1980. As a psychiatrist who has sworn to "Do no harm", as the first tenet of the Hippocratic Oath, using professional knowledge to amplify the adverse impact of human torture is a clear violation of this oath. I recognize that although psychologists have no equivalent to the Hippocratic Oath to guide ethical and

professional practice, the American Psychological Association's official policy is to condemn participation in torture as a mental health professional. I strongly support that position.

Respectfully,



Matthew Friedman, M.D., Ph.D.

Dated: November 21, 2016

Attachments Included:

Appendix A: Curriculum Vitae
Appendix B: References