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EXHIBIT 6

1

Expert Report of Charles A. Morgan, III, M.D., M.A.

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

November 21, 2016



Introduction:

The organization of this report is such that it provides information about my background and experience and then is sectioned according to the four (4) questions that were asked of me. Prior to preparing this report, I read the Complaint.¹ I have attached my CV in the Appendix to this report. In it you will find my education and work experience as well as a list of my scientific publications. In addition, I have included in the Appendix a copy of my fee schedule and cases in which I have provided testimony within the past 4 years.

Brief Summary of Experience:

I am a Board Certified Psychiatrist (i.e., a Diplomat of the American Academy of Psychiatry & Neurology). I have specialized training in Post Traumatic Stress Disorder and also in the special area of Forensic Psychiatry. I initiated my career on the faculty of medicine at Yale University and the National Center for Post Traumatic Stress Disorder (PTSD) in 1990. I have over 25 years of clinical experience working with victims of trauma who suffer from PTSD, as well as 25 years of experience conducting research that is relevant to PTSD. More specifically, I have conducted research in the following areas: the neurobiology of human fear and alarm systems; the neurobiology of stress and PTSD; the psycho neurobiological responses to extreme stress in humans; the nature and impact of Survival School (SERE) training on human cognition, memory and performance; and in eye-witness memory in response to highly stressful events. I have conducted research at the U.S. SERE schools since 1998 and have served on the Pentagon's panel for SERE curriculum. I have over 100 peer reviewed scientific papers related to this body of research and have been recognized as an expert in my field by my scientific peers, and by courts (national and international).

In addition to my work at Yale University, I was a government service (GS) Medical Intelligence Officer with the Central Intelligence Agency from 2003-2010. In this capacity, I worked in the Directorate of Intelligence and in the Directorate of Science and Technology (DS&T). During my years in the DS&T (2004-2010), I was a Senior Research Scientist and a member of the Government Expert Committee on Educing Information. Our Committee was directly involved with the creation of the Intelligence Science Board's report *Educing Information: Interrogation: Science and Art.*² that was prepared for the U.S. Congress regarding the state, art and practice of interrogation.

¹ www.aclu.org/legal-document/salim-v-mitchell-complaint

² Educing Information. Interrogation: Science and Art. Foundations for the Future. Phase 1 Report. National Defense Intelligence College, Washington DC. December 2006. NDIC Press.

It is based on my experience as a clinician with expertise in PTSD, a researcher with expertise on stress and on SERE, and my experience as an intelligence officer that I have prepared this report.

Question 1:

What was known in 2002 about the physical and psychological effects of inducing and sustaining a state of uncontrollable stress in humans, including through the use of techniques used on U.S. military recruits during Survival, Evasion, Resistance and Escape ("SERE") training?

Answer:

It is within a reasonable degree of medical certainty that a professional in the fields of psychology, and the relevant behavioral and medical sciences would have known by the time frame 2002, that the effect of inducing and sustaining a state of uncontrollable stress in humans (to include the use of techniques used to create stress in U.S. military personnel participating in Survival, Evasion, Resistance and Escape ("SERE") training) would not enhance, but instead, would degrade cognition and memory performance.³

When I use the term "techniques" used to create stress in students at SERE, I am referring to a sub-set of training methods – scenarios – designed to create acute stress in students. These are used during the "confinement" or "detention" phase of the course. Before describing them, a few comments are in order. These "scenarios" are designed to give the SERE students an opportunity to briefly experience the different types of stress that POWs have reported experiencing when held in captivity. The educational value of these stressful "scenarios" is directly related to the training model of SERE, which is one of "stress inoculation." The idea is similar to that used in infectious disease medicine where doctors use vaccinations and give people a "mini dose" of a virus so as to protect them in the future from becoming more sick when exposed to the real virus.

At SERE it is believed that by exposing students to stressful scenarios and helping them work through the scenarios, the students will be better prepared to survive if they should at some point in the future become detained or taken captive. The work for students during these scenarios is to try to put into practice what they have been taught in the classroom phase of the course. The "scenarios" are designed to elicit strong feelings of uncertainty, of alarm, of fear, and of concern on the part of the students so that they have the chance to put into practice what they have learned while feeling genuinely distressed. This act – of putting into practice what one has

³ It is not my intent in this report to review in detail the overall findings of the scientific literature on the negative medical and physical consequences of prolonged exposure to uncontrollable stress. It has been my intent to focus on the cognitive and psychological aspects of exposure to such stress.

learned in spite of the distress being experienced – is thought to result in improved stress tolerance in the future.

As noted in the SERE Psychology handbook, Survival School is designed to:

"Provide realistic training that includes vicarious, simulated, or actual exposure to traumatic stimuli that may be encountered. Examples of application of this principle in military training include exposure to live weapons fire, survival training, or, for subgroups of military personnel, mock captivity training. This principle can be applied to many work roles-for example, those likely to be involved in body handling might be trained in mortuary environments. It is consistent with classical conditioning theories, in that this can help reduce arousal or anxiety associated with particular traumatic stimuli. Strengthen perceived ability to cope during the trauma and with the aftermath. Realistic training contributes to this goal. Instruction and practice in the use of a variety of coping skills (e.g., stress inoculation training, problem-solving, assertion, and cognitive restructuring) may be helpful in enabling workers to tolerate stressful work environments. In addition, individuals can be trained to cope with acute stress reactions that are common following trauma exposure. Such training experiences help to maximize expectations of mastery of traumatic situations and their physical and emotional sequelae. Use of positive role models (leaders and peers) is also an effective tool for building up the sense of ability to cope. The training must include specific, practical actions to change the threatening or horrifying situation for the better. Without such positive action learning, "simulated" terrifying or horrifying situations and stimuli can induce feelings of helplessness that make the training itself traumatizing."4

As noted in the manual, there is an explicit acknowledgement that without positive action learning, the "simulated" scenarios at SERE produce feelings that would make the training itself traumatizing, or capable of causing Post Traumatic Stress Disorder.

Several key elements are integral to the process of ensuring that the scenarios create genuine distress and fear in the SERE students. First, it is necessary to create a perception, on the part of the students, that they are helpless. This is difficult, however, because the students at SERE know that they have enrolled in a training course. They know that the SERE course runs for a specific number of days and that whatever discomfort they may feel, it will come to an end at some point, indeed, by a date certain. At the same time, the students are aware that if they do not complete

⁴ SERE Psychology Handbook, Chapter Two: "Why We do the "Voodoo" that We do: Ethical Practices in Survival, Evation Resistance and Escape (SERE) Psychology. Dr. J.F. Orgrisseg, Ph.D. Provided to me via email by Dr. Gary Percival of the Joint Personnel Recovery Association (JPRA), September 29, 2016...

the SERE course there are significant implications for their ability to deploy – which is something they desire. Thus, as set forth below, SERE stress conditions do not match those used in laboratory animals or those that apply to people who are detained in real world settings. However, in 2002, as reflected by our peerreviewed, published, scientific papers, the relevant scientific community accepted SERE as a valid venue for understanding, with limitations, the impact of acute stress in humans.⁵,⁶,^{7,8,9}

That said, it is extremely important to emphasize the fact that there are *significant differences* between the stress experienced by students in training at SERE and the stress experienced by animals in laboratory experiments or by humans detained in real world situations. First, the stress exposure at SERE is brief in duration and paired with "in role" or "out of role"¹⁰ feedback by an instructor who is actively helping the student correctly "resist" the demands of said "enemy" instructor. The brief duration of the stress limits the potential psycho-biological harm to the student; the active support from the instructor promotes positive coping skills and limits the development of negative self-appraisals.¹¹

Second, the stress experienced by the student at SERE occurs because the student voluntarily agreed to participate in a legitimate, authorized, military class. Although it may be extremely stressful to them, participation in the course is something the individual soldier, sailor, marine or airman has agreed to do. They may withdraw from training, and obviously may withdraw from the SERE course. This situation is, again, *distinctly different* than the conditions of laboratory animals or humans detained in the real world. Laboratory animals do not have a choice about their exposure to uncontrollable stress. Humans detained in the real world certainly do not volunteer to be subjected to such stress and are not in control of when their detention will come to an end, as SERE students are – perhaps the most significant difference of all. Thus, extrapolating from the research at SERE to humans has

⁵ Morgan III CA, Wang S, Mason J, Hazlett G, Fox P, Southwick SM, Charney DS, Greenfield G: Hormone Profiles in Humans Experiencing Military Survival Training. Biol Psychiatry, 2000; 47: 891-901.

⁶ Morgan III CA. Wang S. Southwick SM. Rasmusson A. Hauger R. Charney DS: Plasma Neuropeptide-Y in Humans Exposed to Military Survival Training. Biol Psychiatry 2000; 47: 902-909.

⁷ <u>Morgan III CA</u>, Wang S, Hazlett G, Rassmusson A, Anderson G, Charney DS: Relationships among Cortisol, Catecholamines, Neuropeptide Y and Human Performance During Uncontrollable Stress. Psychosomatic Med. 63: 412-42; 2001.

⁸ Morgan III CA, Hazlett G, Wang S, Richardson G, Schnurr P, Southwick SM: Symptoms of Dissociation in Humans Experiencing Acute Uncontrollable Stress: A Prospective Investigation. Am J psychiatry, 158:8; 1239-1247. 2001.

⁹ Morgan III CA. Rassmusson A. Wang S, Hauger R, Hazlett G: Neuropeptide-Y, Cortisol and Subjective Distress in Humans Exposed to Acute Stress: Replication and Extension of a Previous Report. Biol Psychiatry. 52: (2) 136-142, 2002.

¹⁰ Instructors help students by making explicit verbal comments. They may make these comments while still "in role" and acting as an enemy interrogator. They may also make these comments after calling a "time out" showing their genuine US Government or Military ID to the student, and speaking directly and openly (i.e. "out of role") about how the student is behaving or responding in the training program.

¹¹ See section on Learned Helplessness for the role of negative self-appraisals in the development of the condition of Learned Helplessness.

limitations: although we are able to legitimately comment on the negative impact of SERE stress, we recognize that it necessarily *underestimates* the damaging effects on humans exposed to the kind of uncontrollable stress, under conditions set forth, as described in the Complaint. Yet, in spite of the fact the stress at SERE cannot possibly equal the stress in animal experiments or the stress of real world detained persons, SERE stress elicits at least some of the most robust acute stress responses noted in humans to date and thus provides some insight into the impact of uncontrollable stress in humans. But the differences are obvious and must never be forgotten.

A number of factors contribute to the efficacy of the SERE scenarios in creating genuine stress and feelings of helplessness in students. These include: exposure to the elements, sleep deprivation, food deprivation, physical discomfort in confinement cells, exposure to cold water immersion, cold water hosing, isolation stress, interrogation stress, social stress or water-boarding stress. It should be noted that there are a number of techniques set forth in the Complaint that are not among those I have ever seen in the curriculum for SERE (e.g., a person being covered with ice and wrapped in plastic; suspended from the ceiling; the wearing of diapers; physical insertion of objects into the anus; being chained to a wall; injections).

With respect to exposure to the elements, during the detention phase, SERE students experience genuine physical discomfort in terms of heat or cold exposure. In spite of the active measures taken by the medical SERE staff, students may experience significant cold injury (to feet and fingers) or significant heat injury (heat exhaustion). In addition, in response to the stress of this phase, students may exhibit immunosuppression – and so they are monitored closely by staff for any signs of infection. Similarly the medical staff ensure that direct measures are taken to prevent dehydration, hypo or hyperthermia and electrolyte imbalances in students during the detention phase of the course.¹² The medical staff at SERE have the authority to determine whether a student continues in training.

Although many measures are used by the SERE training staff to create feelings of uncertainty and stress in the students during the detention phase at SERE, certain stressors (i.e., cold/heat stress, sleep deprivation stress, isolation stress, interrogation stress) are similar to the types of stressors used in non-human animal laboratory experiments as well as to some of the stressors described in the Complaint.

I comment in more detail on these stressors because: A) these have been studied in controlled laboratory research environments and provide a reasonable basis for understanding how these factors affect human beings in non-laboratory settings; B) these factors have been assessed in my own research conducted at the US Army,

¹² This is done is all phases of the course. Issues related to hydration, body temperature control and electrolyte imbalances can escalate more quickly during the detention phase and the monitoring is more frequent.

Navy and Marine SERE schools; and C) because these types of stressors are relevant to those in the Complaint.

Heat/Cold Stress:

It is within a reasonable degree of medical and scientific certainty that by the time frame of 2002, a doctoral level professional in psychology or the behavioral sciences would know that the stressors of heat and cold were capable of producing distress¹³, inducing uncontrollable stress¹⁴, and/or impairing performance and cognitive function.¹⁵,¹⁶,¹⁷ From a scientific perspective, by the time period in question (2002), it was established fact that heat and cold stressors were highly capable of activating mammalian neuro-hormonal systems associated with alarm, sympathetic arousal and Hypothalamic-Pituitary-Adrenal Axis activity.

As noted above, it had been well established in the scientific community, by the year 2002, that exposure to significant heat and cold stress was capable of conferring significant negative effects on mammalian functioning. The primary scientific questions, over the past decades, have focused on the specific mechanisms by which these stressors confer their negative impact. As noted below in my discussion of "Learned Helplessness" the negative impact of *any* stressor is much stronger and more destructive when the stress is perceived as "uncontrollable" by the animal or human that is experiencing the stress.

In that regard, the degree to which stress is perceived as "uncontrollable" is related to a number of factors: the *severity* of the stressor; the *relationship* between the stressor and the stimulus complex associated with it; the *predictability* of the stress onset; the *control* the animal or person has over stress onset and termination; and *how prepared* the organism/animal or human is in terms of dealing with stress. Any of these elements can result in a stressor being "uncontrollable" and create the kind of damage discussed below.

Isolation Stress:

¹³ Thermal Stress: Physiological, comfort, performance, and social effects of hot and cold environments. PA Bell, TC Greene Environmental Stress. Gary Evans (Ed). Cambridge University Press. 1982.

¹⁴ McEwen Bruce S. Protective and Damaging Effects of Stress Mediators. N England Journal of Medicine. 1998: 338:171-179.

¹⁵ For Review of Cold Stress and Humans see: Human Physiological Responses to Cold Exposure. Jodle Stocks, et al. 2004. Aviation Space Environmental Medicine. The research cited in the review is data that was generally accepted prior to that point in time. The time frame covered in the data referenced is from 1946 through the 2002 time frame.

¹⁶ For example, see: Task Categorization and the limits of Human Performance in Extreme Heat by Peter Hancock. In: Aviation Space Environmental Med. 53 (8): 778-784, 1982.

¹⁷ For larger overview of the field: Human Performance: Cognition, stress and individual differences. Gerald Matthews, D. Roy Davies, Steven J. Westerman, Rob B. Stammers. (Eds.). 2000. Psychology Press. East Sussex, BN3 2FA; In USA: Taylor & Francis Group.

It is within a reasonable degree of medical and scientific certainty that by the time frame of 2002, a doctoral level professional in psychology or the behavioral sciences would know that the stressor of isolation stress was very capable of causing significant distress and impairment in mammals^{18,19,20} and significant psychological distress and psychiatric illness in humans.^{21,22} Thus, the incidence of psychiatric disorders was significantly higher in prisoners placed in solitary confinement as compared to those in non-solitary confinement (28% vs. 15%). Clearly, within the scientific community, and under the regulations of U.S law regarding scientific research involving humans,²³ there are legal and ethical barriers to studying the impact of isolation stress of the type described in the Complaint. The data that exist regarding its impact on humans are, as a result, largely derived from prison populations. The scientific literature also indicates that although some humans may respond negatively to isolation stress, others may not.^{24,25}

In our research at Survival School, I did evaluate the psychological and neuroendocrine responses to isolation stress. During the years 1998-2002 we found that while some students at Survival School exhibited stress (as measured by, for example, increases in the stress hormone cortisol and reductions in testosterone), others did not. Retrospectively, when we examined our data it appears that the students who were higher in the psychological trait of introversion were less stressed when in isolation than were those who were extroverts. This makes some sense in that individuals who are generally energized by interactions with others (i.e., extroverts) might find the isolation experience more distressing.

The strength of this scientific finding was limited by the number of students in that particular phase of the study. Perhaps one day we may have the opportunity to do a study designed to replicate the finding. To date, we have not and are focused on

¹⁸ Wiberg GS, Grice HC: Isolation Stress in Rats. Science. 1963 Oct 25; 142 (3591):507. An early prominent scientific paper showing that isolation stress significantly impacts the fear and alarm systems of the rodent.

¹⁹ Blanc G, Herve D, Simon H et al. Response to Stress of mesocortico-frontal dopaminergic neurons in rats after long-term isolation. Nature. 284 (5753): 265-7. April 1980. This paper conveys our understanding about how long term exposure to isolation stress specifically leads to an alteration in normal brain functioning.

²⁰ Johnson EO, Kamilaris TC, Carter CS, Calogero AE, Gold PE, Chrousos GP. The Biobehavioral consequences of psychogenic stress in a small, social primate (Callithrix Jacchus Jacchus). Biological Psychiatry Vol. 40 (5) 1 September 1996. Pp 317-337. Isolation stress significantly activates the HPA axis and results in increased ACTH, beta endorphins, plasma cortisol, behavioral arousal.

²¹ Zekind E. Isolation Stress and Medical and Mental Illness. Journal of the American Medical Association (JAMA). 1958: 168 (11): 1427-1431.

²² Andersen HS et al., (2000) A longitudinal study of prisoners on remand: psychiatric prevalence, and incidence and psychopathology in solitary vs. non-solitary confinement. Acta Psychiatrica Scandinavica. July 2000, 10.1034/j.1600-0447.2000.102001019.x.

²³ Code of Federal Regulations Title 45. Public Welfare Department of Health and Human Services Part 46 Protection of Human Subjects.

²⁴ Ecclestone CE, Gendreau P, Knox C: Solitary confinement of prisoners: An assessment of its effects on inmates' personal constructs and adrenocortical activity. Canadian Journal of Behavioral Science. Vol 6 (2) Apr 1974, 178-191.

²⁵ Suedfeld P, Ramirez C, Deaton J, Baker-Brown G: Reactions and Attributes of Prisoners in Solitary Confinement. Criminal Justice and Behavior. 1982 Vol. 9 (3) 303-340.

other projects. This said, it is reasonable to assume that most but not all humans will find the experience of isolation stress, *per se*, to be distressing or "uncontrollable." Based on the scientific evidence, the determining factor is the experience of the individual who is subjected to isolation stress. For example, if the individual's subjective perception of the stressor is that it is outside of his/her control, that it is unpredictable or that it is indefinite, then isolation will be experienced as "uncontrollable stress." That is the case here, based upon the allegations of the Complaint.

Noise Stress/Light Stress:

In our studies at Survival School, we did not specifically evaluate the impact of noise stress on student stress levels. By the year 2002, however, within the scientific literature it was well known that noise stress can be an effective, reliable tool in laboratory research for creating uncontrollable stress conditions. Used in this way, noise stress is known to cause a decline in cognitive functioning²⁶, known to alter mood in a negative manner²⁷and can also negatively affect neuro hormone responses.²⁸

Likewise, one technique that has reported to have been used in the torture of people is forced light exposure.²⁹ Although the scientific literature is complicated due to the purpose and the design of pre-clinical and clinical studies, it is reasonable to say that, in the time frame of 2002, a doctoral level professional in psychology, psychiatry or in behavioral sciences would expect prolonged, unwanted, light exposure to result in deteriorated performance or functioning on the part of the animal or person subjected to the light exposure, perhaps primarily through the disruption of the animal or person's sleep pattern. Moreover, as noted above, the degree to which any stimulus, including noise or light, is perceived by the recipient as aversive, unwanted, distressing , unpredictable or indefinite in duration, that stimulus or stressor can create a condition of "uncontrollable stress," and have the potential to cause traumatic injury.

Sleep Deprivation:

Although study designs have varied, the overall assessment of the issue has remained the same. With respect to the negative effects of sleep deprivation on human cognitive performance:

²⁶ Arnsten AFT, Goldman Rakic PS. Noise Stress Impairs Prefrontal Cortical Cognitive Function in Monkeys. Arch. Gen. Psychiatry, 1998–55 (4): 362-368.

 ²⁷ Brieir A, Margo A, Pickar D, et al. 1987. Controllable and uncontrollable stress in humans: Alterations in mood and neuroendocrine and psychophysiological function. Am J. Psychiatry vol. 144 (11) 1987; 1419-1425.
²⁸ De Boer SF, Van Der Gugten J, Slangen JL. Plasma catecholamine and corticosterone responses to predictable

and unpredictable noise stress in rats. Physiology and Behavior. Vol 45 (4) 1989. Pp 789-795.

²⁹ Roth EF, Lunde I, Boysen G and Genefke IK. Torture and its Treatment. AmJ of Public Health. Vol 77 (11). 1987, 1404-1406.

Neuro-imaging data indicate that the effects of 24 hours of sleep deprivation produces global reductions in brain activity. The decreases in brain activity are greatest in cortico-thalamic networks. These networks are the neural systems that mediate attention and higher order cognitive processing and functioning.³⁰ This means that the overall functioning of human processing of information is generally degraded by sleep deprivation. Sleep deprivation will not, therefore, enhance a person's ability to recall information, nor will it render a person more lucid or cogent.

Neuropsychological and Neuro-imaging studies show that sleep deprivation directly and negatively affect the pre-frontal cortex.^{31,32} Indeed, not only does sleep impairment degrade pre-frontal function, but conditions of sleep deprivation also require the prefrontal cortex to work harder in order to accomplish the same tasks as a non sleep deprived individual.³³ Taken together these data indicate that a person who has suffered significant sleep deprivation is likely to be impaired in his/her ability to perform complex human reasoning and decision-making.³⁴

Sleep-deprivation induced deficits in human cognition have real world consequences. Taken together, by 2003, the scientific literature indicated that sleep deprivation impairs human decision-making for situations that involve a person having to deal with the unexpected, or with something that requires innovation, or that requires a person to revise their plans, or that requires them to deal with competing distractions. It was also well established at that time that sleep deprivation also impaired effective communication between individuals.³⁵ With respect to non-laboratory based findings, the scientific literature had demonstrated at that time that sleep deprivation was significantly associated with accidents,³⁶

³⁰ Neural Basis of alertness and cognitive performance impairments during sleepiness. Effects of 24 h of sleep deprivation on waking human regional brain activity. Thomas, M, Sing H, Belenky G, Wagner Jr. H, Thorne D, Popp K, Rowland L, Welsh A, Balwinski S, and Redmond D. J. Sleep Res. (2000) 9, 335-352.

³¹ The area of the human brain associated with planning complex cognitive behavior, personality expression, decision making and modulating how one behaves in social situations.

³² Harrison Y & Horne JA (1997): Sleep deprivation affects Speech. Sleep, 20 (10), 871-877. Harrison Y & Horne JA (1998): Sleep loss impairs short and novel language tasks having a prefrontal focus. Journal of Sleep Research, 7, 95-100; Harrison Y & Horne JA (1999): One night of sleep loss impairs innovative thinking and flexible decision making. Organizational Behavior and Human Decision Processes. 7, 128-145; Horne, JA (1988a) Sleep loss and Divergent thinking ability. Sleep, 11, 528-536; Horne, JA (1993): Human sleep, sleep loss and behavior: Implications for the prefrontal cortex and psychiatric disorder. British Journal of Psychiatry, 162, 413-419.

³³ Brain Activity is Visibly Altered Following Sleep Deprivation". *UC San Diego Health System*. 3 February 2006. Archived from the original on 2011-03-10.

³⁴ The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation.Van Dongen HP, Maislin G, Mullington JM, Dinges DF, Sleep. 2003 Mar 15; 26(2):117-26.

³⁵ The impact of Sleep Deprivation on Decision Making: A Review. Yvonne Harrison & James A Horne. Journal of Experimental Psychology: Applied. (2000) Vol 6, No. 3, 236-249.

³⁶ Catastrophes, sleep and public policy: consensus report. Mitler, M, Czeisler C, Dement W, Dinges D. Sleep, (1988) 11: 100-109.

military friendly fire incidents,³⁷ and with motor vehicle crashes.³⁸ Thus, it is clear, from a scientific perspective, that the types of impairments measured in the laboratory are not trivial or inconsequential: they are connected to, and elucidate the nature as to why and how sleep deprivation causes problems in real world situations.

With respect to human memory, the negative impact of sleep deprivation on human memory has been well known about and noted in the scientific literature for over 50 years.³⁹ Since the time of those scientific observations in the 1960s, a large body of evidence has been accumulated that has confirmed that sleep deprivation degrades or impairs human memory. Taken together this literature indicates that sleep deprivation affects memory in some very specific ways.

First, human "working memory" is significantly and negatively affected by sleep deprivation. Sleep deprivation results in a significant impairment of temporal memory (although not of memory for a face – facial recognition), and one that is independent of the level of "arousal" or sleepiness.⁴⁰ This suggests that the temporal memories of people who suffer from significant sleep deprivation are likely impaired or degraded. From a practical perspective this means that facts and details associated with temporal sequencing and context are most likely to be disrupted. It is less likely that sleep deprivation, per se, will affect the ability of a human to recognize the face of a person. This said other factors, such as stress exposure, are likely to degrade face recognition as well.⁴¹

Human memory formation, as well as the ability to recall long-term memory are significantly, negatively affected by sleep deprivation.⁴² Sleep deprivation impairs the transformation of working memories into long term memories;⁴³ also, the

³⁷ The effects of sleep deprivation on performance during continuous combat operations. Blenky G, Penetar D, Thorne D, Popp K, Leu J, Thomas M, Sing H, Balkin T, Wesenten N, Redmond D. In B. M. Marriot (Ed) Food components to enhance performance. National Academy Press. Washington DC (1994) 127-135. Artillery teams in simulated sustained combat: performance and other measures. Banderet, LE, Stokes JW. Francesconl R, Kowal DM and Naitoh P. In LC Johnson, DJ Tepas, WP Colguhon, MJ Colligan (EDs). Biological Rythms, Sleep and Shift Work. Spectrum. New York. (1981). 459-477.

³⁸ Sleep related vehicle accidents. Horne JA, and Reyner LA. BMJ (1995) 354: 1179-1194.

³⁹ Misperception and disorientation during sleep. Morris GO, Williams HL and Lubin A. (1960). Archives of General Psychiatry. 2, 247-254; Williams JL, Lubin A & Goodnow J. (1959) Impaired performance with acute sleep loss. Psychological Monographs, 73, (484).

⁴⁰ Sleep Loss and Temporal Memory. Yvonne Harrison and James A Horne. Quarterly Journal of Experimental Psychology, 2000. 53A (1), 271-279.

⁴¹ Morgan III CA, Hazlett GA, Doran T, Garrett S, Hoyt G, et al.: Accuracy of Eyewitness Memory for Persons Encountered during Exposure to Highly Intense Stress. International Journal of Psychiatry & the Law. (2004) vol. 27 (3) p.p. 265-279.

⁴² Oniani, T.N. (1982) Role of sleep in the regulation of learning and memory. Hum. Physiol., 8, 381–391. Wilson, M.A. & McNaughton, B.L. (1994) Reactivation of hippocampal ensemble memories during sleep. Science, 265, 676–679.

⁴³ Linden, E. R.; Bern, D.; Fishbein, W. (1974). "Retrograde amnesia: prolonging the fixation phase of memory consolidation by paradoxical sleep deprivation". Physiology and Behavior. 14: 409–412. <u>doi:10.1016/0031-9384(75)90004-9</u>; Campbell, I.G., Guinan, M.J. & Horowitz, J.M. (2002) Sleep deprivation impairs long-term

processes involved in the retrieval of memories are impaired by sleep deprivation.⁴⁴ Although some of the specific mechanisms by which sleep deprivation causes such deficits in memory functioning had not yet been elucidated by scientists prior to the year 2003, the negative impact of sleep deprivation on memory had been clearly established as an issue in the scientific literature.

Although sleep deprivation has not been the focus, per se, of my published research at Survival School, it was important in the early years of my research work (1997-1999) that I assess the impact of sleep deprivation experienced by students at the school so that I could subsequently measure the impact of other types of stress experienced by students at Survival School (for example, interrogation stress, isolation stress, social stress).

In 1998, my colleagues and I assessed sleep/wake information during all phases of the Army course (classroom, evasion phase, confinement phase, post confinement phase).⁴⁵ During the confinement phase we found (through the use of sleep Actigraphs⁴⁶) that students were obtaining an average of 6 hours of sleep per night. During the evasion phase, sleep was erratic and averaged 2-5 hours (there was a wide range of variance among the students). During the captivity phase, the average number of hours of sleep for students we measured was approximately 4. We also administered psychological questionnaires and found that many students were experiencing cognitive distortions, visual hallucinations, concentration and attention difficulties.

We conducted a study of cognitive distortions in the different phases and found that most students were free of such symptoms in the classroom phase, some experienced them in the evasion phase and all experienced them in the confinement phase. Because the sleep deprivation was more uniform in the confinement phase⁴⁷, the focus of our publication was the degree to which interrogation stress produced cognitive distortions.⁴⁸ However, and consistent with previous scientific literature on sleep deprivation and cognition, we did observe an association between cognitive distortions and sleep deprivation experienced by the students at Survival school. At no time did we ever observe improved cognition due to sleep deprivation.

potentiation in rat hippocampal slices. J. Neurophysiol., 88, 1073–1076. Davis, C.J., Harding, J.W. & Wright, J.W. (2003) REM sleep deprivation induced deficits in the latency-to-peak induction and maintenance of long-term potentiation within the CA1 region of the hippocampus. Brain Res., 973, 293–297

⁴⁴ Effects of stress and REM sleep deprivation on the patterns of avoidance learning and brain acetylcholine in the mouse. *Sagales T, Domino EF, Psychopharmacologia*. 1973; 29(4): 307-15.

⁴⁵ Leu, J. Morgan CA III: Sleep patterns in Survival School students: Evidence from Sleep Acti-graph Data. Unpublished data. 1998.

⁴⁶ A wrist worn watch like device that monitors activity and sleep/wake information.

⁴⁷ As per JPRA regulations, the SERE confinement phase ensures a set number of hours of sleep for students.

⁴⁸ Morgan III CA, Hazlett G, Wang S, Richardson G, Schnurr P, Southwick SM: Symptoms of Dissociation in Humans Experiencing Acute Uncontrollable Stress: A Prospective Investigation. Am J psychiatry, 158:8; 1239-1247. 2001.

The type of sleep deprivation described in the Complaint is not comparable to that experienced at SERE. It is far greater in frequency, in intensity and in duration than the sleep deprivation experienced by students enrolled in Survival School training. Accordingly, one can assume that if the deprivation were greater, we would observe greater cognitive distortions; it is also my opinion that those subjected to such sleep deprivation would experience real trauma and harm.

Interrogation Stress:

Until the US Government gave us unique access to the Survival School venue, there were no controlled, prospective, scientific studies about the impact of acute, realistic and uncontrollable stress in humans. In 1998, our research group at Yale & the VA National Center for PTSD, knew that if it were possible to study healthy people before, during and after exposure to high levels of stress, then we might significantly enhance our understanding as to how and why people differ in their vulnerability to stress. We specifically wanted to understand how stress affects emotional and cognitive functioning as well as human memory functioning.

Prior to studies at Survival School, in the scientific community we knew that high levels of adrenalin (from activation of fear and alarm systems) could significantly and negatively affect human prefrontal cortex operations^{49,50} that high levels of the stress hormone cortisol could damage areas of the human brain associated with memory formation⁵¹, that exposure to uncontrollable stress impairs information processing and learning^{52,53} and that high levels of glutamate turnover in the human brain result in symptoms of dissociation.⁵⁴ We did not know, prior to research at Survival School, how these laboratory based findings connected to human responses in real world highly stressful situations.

⁴⁹ Bremner, J.D., R.B. Innis, C.K. Ng, et al. 1997. Positron emission tomography measurement of cerebral metabolic correlates of yohimbine administration in combat-related posttraumatic stress disorder. Arch. Gen. Psychiatry 54: 246–254.

¹⁰ Southwick SM, Krystal JH, <u>Morgan CA</u>, Johnson DR, Nagy LM, Nicolaou A, Heninger DR, Charney DS: Abnormal Noradrenergic Function in Posttraumatic Stress Disorder, Arch Gen Psychiatry, Vol. 50, 266-274, (1993)

⁵¹ See: Review in: Sapolsky R. Glucocorticoids and Hippocampal Atrophy in Neuropsychiatric Disorders. Arch Gen Psychiatry. 2000; 57 (10): 925-935. Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Southwick SM, Delaney RC, McCarthy G, Charney DS, Innis RB: MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. Am J Psychiatry 1995; 152:973-981; Bremner JD: Does stress damage the brain? Biol Psychiatry 1999; 45:797-805; Sass KJ, Spencer DD, Kim JH, Westerveld M, Novelly RA, Lencz T: Verbal memory impairment correlates with hippocampal pyramidal cell density. Neurology 1990; 40:1694-1697

⁵² Morgan CA, III & Grillon C. Abnormal mismatch negativity in women with sexual assault related PTSD. Biol Psychiatry, (1999) 45: 827-832.

⁵³ Grillon C & Morgan CA: Fear Contextual Startle Conditioning to Explicit and Contextual Cues in Gulf War Veterans with Posttraumatic Stress Disorder. J Abn Psychology, (1999) 108: 134-142.

⁵⁴ Krystal JH, DSouza DC, Karper LP, Bennett A, et al. (1999) interactive effects of subanesthetic ketamine and haloperidol in healthy human subjects. Psychopharmacology, 145, 193-204.

In our research we assessed a number of measures in order to understand stress responding, cognitive processing and memory in Survival School students. We measured hormones in both blood and saliva, we measured physiology, and we measured psychological symptoms and cognitive operations. Put briefly, we were persuaded that Survival School was a valid place to study the real impact of acute stress in humans.

Taken together by the year 2002, our findings indicated that: A) exposure to interrogation stress at Survival School resulted in some of the highest neurohormonal and neurobiological alterations recorded in humans exposed to acute stress^{55,56}; B) that interrogation stress resulted in significant cognitive distortions and alterations in human perception and information processing^{57,59}; and C) that these findings had been replicated.⁵⁹ Finally and although we did not formally publish our data on the negative impact of SERE stress on eyewitness memory in students at SERE until 2004⁶⁰, these data as well as other data on the negative impact of stress on student cognitive performance were presented to the SERE community during the 1999-2001 time frame. One reason for the delay in publication of the eyewitness data was due to having the information about the Survival School (not the scientific data) cleared by the US Government for distribution to the general public.

During the time period of 2002, the research from Survival School was known within the relevant scientific community. Our findings were known to be compatible with the findings from pre-clinical and human laboratory based studies and fully supported the body of scientific evidence showing that the uncontrollable condition of interrogation stress had a significant negative effect on human cognition, perception and memory processing. A reasonable person cognizant of our data at that time would understand that SERE stress had a *significant, negative* impact on human brain and physiological functioning.

⁵⁵ Morgan III CA, Wang S, Mason J, Hazlett G, Fox P, Southwick SM, Charney DS, Greenfield G: Hormone Profiles in Humans Experiencing Military Survival Training. Biol Psychiatry, 2000; 47: 891-901.

⁵⁶ Morgan III CA, Wang S, Hazlett G, Rassmusson A, Anderson G, Charney DS: Relationships among Cortisol, Catecholamines, Neuropeptide Y and Human Performance During Uncontrollable Stress. Psychosomatic Med. 63: 412-42; 2001.

⁵⁷ Morgan III CA, Wang S, Southwick SM, Rasmusson A, Hauger R, Charney DS: Plasma Neuropeptide-Y in Humans Exposed to Military Survival Training. Biol Psychiatry 2000; 47: 902-909.

⁵⁸ Morgan III CA, Hazlett G, Wang S, Richardson G, Schnurr P, Southwick SM: Symptoms of Dissociation in Humans Experiencing Acute Uncontrollable Stress: A Prospective Investigation. Am J psychiatry, 158:8; 1239-1247. 2001.

⁵⁹ Morgan III CA, Rassmusson A, Wang S, Hauger R, Hazlett G: Neuropeptide-Y, Cortisol and Subjective Distress in Humans Exposed to Acute Stress: Replication and Extension of a Previous Report. Biol Psychiatry. 52: (2) 136-142, 2002.

⁶⁰ Morgan III CA, Hazlett GA, Doran T, Garrett S, Hoyt G, Baranoski M, Thomas P, Southwick SM: Accuracy of Eyewitness Memory for Persons Encountered During Exposure to Highly Intense Stress. International Journal of Psychiatry and the Law, 2004 vol 27/3: pp 265-279.

To be clear, the experience of mock detainment and of being interrogated during Survival School is not equal to the experience of a genuine POW or a detainee who is confined and interrogated in the real world. Nor can it reasonably be considered equal to the stress associated with the experiences described in the Complaint. It is well within a reasonable degree of medical and scientific certainty to assume that the distress and stress experienced by a genuine detainee or POW interrogated in real world circumstances such as those described in the Complaint – and who understand that they may die – is significantly greater than the experienced by students who are engaged in military training at SERE.

It is similarly reasonable (and would have been in the year 2002) to conclude that the interrogation stress-induced alterations in cognition, memory, perception, hormones and physiology we observed at SERE cannot be equated with those one would expect to observe in real world interrogations such as the conditions described in the Complaint. Under real world conditions - like those described in the complaint - the degree of uncontrollable stress would be significantly greater, and reasonably expected to cause significant greater psychological, physical and neurobiological injury.

In particular, a doctoral level trained person familiar with our published SERE data in the year 2002 would not conclude that our studies provided evidence that the SERE techniques were "harmless." To conclude that our data indicated that SERE techniques were harmless would reflect a *profound* misunderstanding of our studies, a reasoning error, or both.

By the year 2002, we had completed publication of our primary data regarding the magnitude of stress responses (psychological, physiological, hormonal) in students at SERE. Our initial goal was to establish whether SERE was a valid venue for the study of acute uncontrollable stress in humans. We had accomplished that goal. We had not designed our initial studies to assess whether SERE training had longer term consequences – such as "stress sensitization".

A second goal of the early research at SERE was to assess whether specific neurohormonal factors known to be associated with stress hardiness and stress vulnerability would distinguish which students experienced greater or fewer stress induced deficits when exposed to the stress of the detainment/confinement phase. By 2002 we had published our data regarding specific factors (e.g. Neuropeptide-Y⁶¹; propensity to dissociation⁶²) that affected how well a person tolerated the stress of SERE. Although neither of these data sets spoke directly to the issue of "stress sensitization," our data on the levels of dissociation in students at SERE did raise the possibility that some students were more at risk to the negative impact of the stress than others.

⁶¹ See footnote #5.

⁶² See footnote #7.

We had not detected specific evidence of stress sensitization in our psychological or neuro-endocrine data – which would have suggested that training stress might make a student more vulnerable rather than more resilient to stress. Clearly, the absence of evidence is not evidence, so the question remained an open one. That is, with respect to SERE data in the 2002, the absence of evidence about stress sensitization could not be called evidence *for* the safety of the SERE techniques. Indeed, there is no basis to conclude that SERE techniques are safe, let alone that the techniques deployed here, as described in the Complaint. To the contrary, and given the degree to which we know that the techniques are able to activate fear and alarm systems in student, dramatically alter neuroendocrine responses and psychological processing, it would be reasonable to conclude that – in the absence of all the safeguards that are put in place to assist students during the training experience, the SERE techniques in and of themselves are harmful in terms of their traumatic nature and potential to cause PTSD.

Question 2: What is the reasonableness of using findings of research conducted on U.S. military recruits during Survival School (SERE) training to predict the outcome of the kind of treatment alleged in this case, as described in the Complaint ?

It is within a reasonable degree of medical certainty to conclude:

1) that a person with doctoral level training in the relevant behavioral sciences would not believe that the findings of our research at SERE (and more specifically the type of SERE stress that resulted in our published findings) constituted proof that SERE stress techniques could be used as a valid means for the acquisition of accurate information in a real world interrogation setting. A person who did hold such a belief would be: A) misinformed about the nature of our SERE findings that were available in the 2002; B) ignorant about the robust relationship between high stress exposure and the degradation of information and reduced cognitive functioning; C) deliberately indifferent to the known scientific literature in 2002 (both non-human and human studies) regarding the relationship between stress, cognitive dysfunction, and memory dysfunction; or D) some combination of the above.

2) that at that time, a doctoral level professional in the relevant field, would anticipate that prolonged exposure to SERE type stressors would result in psychological harm and in symptoms similar to many of those described in the Complaint.

I will now explain.

First, with respect to my statement that that SERE stress techniques could be used as a valid means for the acquisition of accurate information in a real world interrogation setting would reflect a state of being misinformed or misunderstanding of our SERE research findings, by the year 2002, our research findings from SERE had consistently shown that exposure to highly realistic uncontrollable SERE stress reliably degraded human performance with respect to cognition and memory. We found that the greater the stress intensity, the more marked the deficits became. We also consistently found that although the stress of SERE impaired cognitive functioning in all students, some students were more vulnerable to stress than others and that their cognitive functioning declined even more rapidly and more profoundly than that of other students. We had no data showing that exposure to high stress sustains or improves cognitive or memory function.

Second, our research findings from SERE in the year 2002 were compatible with what had been consistently found in the scientific literature regarding uncontrollable stress in non-human animals: it resulted in a degradation of the animal. It bears emphasizing that whether the science data were about measures of performance; about the ability to process information; about memory, mood, or motivation; or about neurophysiological functioning, the end results were similar: acute (and chronic) exposure to uncontrollable stress was reliably detrimental.

Third, the stress at SERE is significant and the stress conditions at SERE are sufficient to cause long-term retention of memory and behavioral responses in the form of "classical conditioning." Many students who have attended the course have reported that, from months to years after the course, they have experienced increased symptoms of "startle" and memories of SERE upon hearing specific sounds, when smelling specific odors or feeling heat or cold. These cues remind them of what they experienced while in the detention phase at SERE. Their reports are consistent with both the philosophy at SERE (i.e., that a student will have memories and information from the training that come back to their mind when they find themselves in a stressful detainment type situation in the future) and with what is known about how classical conditioning forms under conditions of stress. The elevated stress hormone levels we have documented at SERE are well within the range necessary for the creation of long-term classical conditioning associations in humans.

Fourth, in 2002, a person with doctoral level experience would reasonably anticipate that, in the absence of the positive therapeutic and educational interventions that students were given at SERE, the specific SERE type stressors experienced by student would have the potential to cause significant psychological harm or psychological illness. It is also reasonable to conclude that in the year 2002, a doctoral level person with knowledge in the field of psychology would know that, a person subjected to the type of stress that is described in the Complaint would likely experience significant psychological distress and likely develop a psychological illness as a result of experiences similar to those described in the Complaint.

Finally, once again, the intensity and the duration of the stress as described in the Complaint are significantly greater than what is experienced by students enrolled at SERE. It is not reasonable to conclude that the techniques as described in the

Complaint are benign or equivalent to those used by instructors with students enrolled at SERE. Although we have scientific evidence that SERE training does not appear to induce psychological injury in most SERE students, there is evidence that some SERE students developed psychiatric illnesses, such as PTSD, from exposure to the training stress at SERE.⁶³ It follows that the psychological/psychiatric consequences of the stress described in the Complaint would be significantly worse.

Question #3: What is your opinion about the relationship between SERE training and the model allegedly developed by the defendants in this case, which sought to induce and sustain a state of "learned helplessness."

Answer: My brief answer is this: There is no legitimate relationship between the SERE training model and the "applied" model of Learned Helplessness allegedly described by the defendants in this case. The SERE training model is based on a "stress inoculation⁶⁴." The model to create "Learned Helplessness" is "stress sensitization".⁶⁵,⁶⁶

The defendants in this case appear to believe that by deliberately, repeatedly methodically, distressing human beings in a very specific manner – subjecting them to experiences of "uncontrollable stress" – the psychological health of these humans will deteriorate and reflect the condition of "Learned Helplessness."

They also appear to believe: A) that they will know when they have adequately created this condition in a human being; B) that once this state of "Learned Helplessness" is achieved the person will willingly provide useful information that is relevant to the interrogator; and "apparently" C) that this process will not cause harm to the individual (I have put quotation marks here because I do not and cannot know whether the defendants believe that they have inflicted damage that is reparable or irreparable, or whether they care. Given their direct claim of seeking to create a state of "Learned Helplessness" it is clear, however, that they sought to significantly deteriorate the psychological health and functioning of the person to whom they subjected the techniques.).

This approach differs significantly from (indeed, one could say it is the antithesis of) the training model at SERE where the focus is helping healthy people stay healthy when exposed to stress and to improve their abilities to perform at a high standard of behavior should they find themselves detained by a hostile government or terrorist group. The entire detention phase of the SERE program is explicitly

⁶³ The Department of Veterans Affairs finally granted disability for PTSD to a veteran who claimed his PTSD was caused in 1975 by water-boarding that was experienced during Survival School training. Houston Chronicle, March 22, 2008.

⁶⁴ Meichenbaum, D. (1985). Stress inoculation training. New York: Pergamon

⁶⁵ Petty F, Chae Y, Kramer G, Jordan S, Wilson L. 1994. Learned helplessness sensitizes hippocampal norepinephrine to mild restress. Biological Psychiatry. June 15. Vol 35 (12) pp 903-908.

⁶⁶ Servatius RJ, Ottenweller JE, Bergen MT, Soldan S, Natelson BH. Persistent stress-induced sensitization of adrenocortical and startle responses. Physiology & Behavior. Vol 56 (5) November 1994. pp 945-954.

designed with the over-arching goal to improve the psychological health and stress resilience capacities of each student.

Creating a state of "Learned Helplessness," however, is decidedly not part of SERE training. That is, learned helplessness is not something that is designed to occur, or that likely occurs, in students while they are participating in SERE training.⁶⁷ During the SERE experience – and especially during the detainment phase – some students may initially appear tentative or hesitant when given the opportunity to demonstrate what they have learned in the classroom phase of the training. Through feedback from the training cadre, during their time in the detained phase students begin to demonstrate the skills that they have been taught. The initial "hesitancy" or lack of demonstrating what they have learned is not Learned Helplessness. It is related to the surprise/shock of being "captured." The fact that students make progress and learn from their mistakes while in the detainment phase of SERE shows that they are not developing Learned Helplessness.

On the debrief day (the day following their release from the confinement phase of SERE training), students are able to get feedback on their performance and gain an understanding of what they found challenging. They learn about their successes and some of their vulnerabilities and what they can work on in the future. Students complete surveys about their experience. I have seen the results of thousands of these surveys and the data from these SERE surveys supports the view that the overwhelming majority of students view the training as positive and as helpful. Some feel less positive about the experience and those generally feel that what they have learned at SERE is that it is a good idea "to never get caught by the enemy." However one might view this feedback, it is not evidence of a state of Learned Helplessness. Evidence for the idea that SERE students have developed a state of Learned Helplessness would have to come from studying the behavior of students when they are reinserted into SERE training or into a similar situation of stress. If when inserted into stressful situations, these military personal simply give up and fail to exhibit any adaptive skills that they have been taught then one might have evidence of Learned Helplessness. But there is no evidence that that has ever occurred, and I do not believe, based upon my observations that it would. In sum, SERE does not seek to create, and does not create Learned Helplessness.

Nor could it. Although operational psychologists who work in the SERE environment may have learned about the concept of Learned Helplessness during their academic education, none have clinical training in how to create states of Learned Helplessness in humans. And even if they could do so, they should not: the deliberate creation of Learned Helplessness in humans would be unethical and is not part of any credentialed training program for psychologists or psychiatrists.

⁶⁷ By definition, if learned helplessness was observed during a student's time in the detention phase at SERE, it would likely be an expression of a learned pattern of helpless responding created by events that occurred prior to the time a student arrived at SERE.

The term "Learned Helplessness" was coined by psychologist Martin Seligman in the 1960s. The term reflected the way in which scientists have characterized the behavior of dogs that were put through a series of experiments involving exposure to repeated electric shocks. For some of the dogs in the experiment, the shock was "escapable" - meaning that the animal could press a lever and avoid the shockwhereas for other dogs, the shock was "inescapable" - meaning that the animal was unable to attenuate or avoid the shocks by pressing a lever. In a subsequent phase of testing the dogs were again exposed to shocks but had the opportunity to escape the shocks. Scientists observed that the dogs for which the shocks had been "inescapable" in the first phase of the experiment, lay down and did not try to escape the shocks in phase two - even though there was a possibility of escaping the shocks. The dogs behaved as if they had "learned" that they were helpless and unable to do anything that would ameliorate their situation. They appeared to have given up.⁶⁸ Originally the model of "learned helplessness" was thought by scientists to provide insight into the human psychiatric condition of depression.⁶⁹ Over the years, however, the model has been thought to perhaps explain some self-defeating behaviors observed in humans who have been traumatized.70 Put broadly, the state of learned helplessness is understood to be the result of significant exposure to uncontrollable, inescapable stress.

But there are no known psychological or biological advantages for an animal or person who has developed a state of learned helplessness. Indeed, understood correctly, an animal (or person) who is in a state of "learned helplessness" will not engage in an action or activity that might ameliorate the situation. If one believed, as the defendants apparently believed, that the act of subjecting a person to uncontrollable stress will create Learned Helplessness and force the person to finally admit what they are hiding from the interrogator, this admission would not be the result of the person being in a state of "Learned Helplessness." Instead, it would represent the person taking an action to help their situation. That is, it would reflect a rational choice on the part of the person who would have decided that saying what they know is better than continuing to refuse the interrogators requests. However, the very fact that the person decided to tell the interrogator what he knows - an act that is to result in an improved outcome for that person because it satisfies the interrogator - would be counter to the very concept of learned helplessness in that the person is taking action, making an effort to improve the situation that they find themselves in. Their action of revealing what they have been withholding reflects a belief on their part that by telling the interrogator what

⁶⁸ Sellgman, M. E. P. (1972). "Learned helplessness". Annual Review of Medicine. 23 (1): 407-412.

⁶⁹ Peterson, C.; Seligman, M.E.P. (1984). "Causal explanations as a risk factor for depression: Theory and evidence". *Psychological Review*. **91**: 347–74.

⁷⁰ Wolfe VV, Gentile C, Wolfe D. The Impact of Sexual Abuse on Children: A PTSD formulation. Behavioral Therapy Vol. 20; (2) Spring 1989. Pp 215-228; Herman JL. Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. Journal of Traumatic Stress (1992) 10.1002/jts.2490050305

Hammack, Sayamwong; Cooper, Matthew; Lezak, Kimberly. "Overlapping neurobiology of learned helplessness and conditioned defeat: Implications for PTSD and mood disorders". Neuropharmacology. 2012 Feb 62(2): 565-575.

they know, the pain and suffering will cease. Whatever that is, it is not "learned helplessness."

In the end, "learned helplessness" is a term that refers to a condition caused when an animal is subjected to uncontrollable stress. This condition is detrimental to nonhuman and human animals. Uncontrollable stress and its consequence, learned helplessness, result in a decline in neuroendocrine functioning, behavioral functioning, cognitive and memory functioning and problem solving.⁷¹ As a result, in the timeframe of 2002, one would not have reasonably expected either the application of uncontrollable stress or the state of "learned helplessness" to be productive methods for the acquisition of information.

Question #4: What is the probability of PTSD or other mental conditions resulting from inducing uncontrollable stress in prisoners who did not consent to such treatment through the treatment alleged in this case or otherwise.

Answer: Post Traumatic Stress Disorder (PTSD) is a mental illness that has been recognized by the American Psychiatric Association and Psychological Association since 1980.⁷² PTSD is caused by exposure to either a single traumatic event or to multiple traumatic events.⁷³

As noted in the Diagnostic and Statistical Manual (DSM-IV TR) in effect in 2002, traumatic events are those in which a person has experienced, witnessed or been confronted with an event or events that involve actual or threatened death or serious injury, or other threat to the physical integrity or oneself or others.⁷⁴ Also required (until 2014) was the criterion that the event the survivor experienced be associated with feelings of "intense fear, helplessness or horror,⁷⁵" in other words, events that any normal person would find shocking, scary⁷⁶, uncontrollable, or

⁷¹ Learned helplessness: Theory and evidence. Maier, Steven F.; Seligman, Martin E. Journal of Experimental Psychology: General, Vol 105(1), Mar 1976, 3-46; Learned helplessness and animal models of depression. Maier, S. (1984) Progress in Neuro-Psychopharmacology and Biological Psychiatry. Vol. 8 (3) p.p. 435-446; Lehner H,

Reinstein, DK, Strowbridge BW, Wurtman RJ. Neurochemical and behavioral consequences of acute uncontrollable stress: Effects of dietary tyrosine. Brain Research. (1984) Vol 303 (2) p.p. 215-223.; Kim J, Diamone DM. The Stressed hippocampus, synaptic plasticity and lost memories. Nature Reviews Neuroscience (June 2002) 3, 453-462; Peters ML, Godaert GLR, Ballieux et al. Cardiovascular and endocrine responses to experimental stress: Effects of mental effort and controllability. Psychoneuroendocrinology. (1998) Vol. 23 (1) Jan. p.p. 1-17. Arnsten, A. Stress signaling pathways that impair prefrontal cortex structure and function. Nature Reviews Neuroscience (June 2009) 10, 410-422. Arnsten A. The Biology of being Frazzled. Science. 12 June 1998. Vol. 280, Issue 5370, p.p. 1711-1712.

⁷² American Psychiatric Association. (1980). Diagnostic and statistical manual of mental disorders. (3rd ed.). Washington, DC: Author

⁷³ Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, 1994 (DSM-IV)

⁷⁴ APA, 2000 pp 463. (American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). WashIngton, DC)

⁷⁵ Ibid pp 467.

⁷⁶ Mineka SKelly K The relationship between anxiety, lack of control and loss of control. Steptoe A Appels Aeds Stress, Personal Control and Health. Brussels, Belgium Wiley1989;163-191.

dangerous. With respect to this brief review, it bears noting specifically that the types of experiences noted in the Complaint would be considered traumatic events.

It is within a reasonable degree of medical certainty that in the time frame of 2002, any reasonable professional who possessed training in psychology or psychiatry would know that experiences designed to produce a perception in the mind of a person being interrogated that said individual was at risk for serious injury harm or death would be capable of causing injury in the form of PTSD.

It is also within a reasonable degree of medical certainty that a person with doctoral level training in psychology or psychiatry would know, in the time frame of 2002 that causing physical harm per se is not required in order to create psychological distress sufficient to create psychological harm in the form of PTSD. Indeed, at that time, it was well known that an essential component of a traumatic event was that the person had to experience a subjective sense of fear, or helplessness or horror, and that there be only a threat of injury or bodily harm.

By that time there was no requirement that the person actually be physically harmed in order to cause PTSD. All that was required was that there was a threat of harm to the individual. The scientific data support the idea that the subjective appraisal of a person is directly linked to the degree of to which that person's fear and alarm systems are activated when they are exposed to a traumatic event. It is well within a reasonable degree of medical certainty that exposure to the types of events detailed in the complaint⁷⁷ would be viewed by a community of mental health experts as "traumatic events." It is also well within a reasonable degree of medical certainty that exposure to the types are activated to the events detailed in the complaint would have a high likelihood of causing PTSD.⁷⁸

The events described in the Complaint have a high likelihood of causing PTSD because they were uncontrollable stressors and traumatic events to the victims. The victims had no genuine control over the timing, the frequency, the duration, the intensity or the quality and type of stressors they experienced. The onset and offset of the stressor was determined exclusively by the interrogator. IF and WHEN the interrogator decided that the responses given by the victim satisfied/did not satisfy the interrogator, THEN the interrogator would modulate the stressor according to his desire.

The events as described in the Complaint represent both threatened and/or inflicted genuine harm to a person's physical integrity and they elicited feelings of fear, helplessness and/or horror. Thus they had the potential to cause the symptoms of PTSD and the other symptoms described in the Complaint.

⁷⁷ www.aclu.org/legal-document/salim-v-mitchell-complaint

⁷⁸ The Mental Health Consequences of Torture. Ellen Gerrity, Terence Keane, Farris Tuma (Eds). 2001. More specifically see Chapter 3: Psychological Effects of Torture.

As discussed above, the stress at SERE is robust and is also capable of creating significant psychological distress and also capable of creating psychological injury. But the training staff at SERE (like the training stress in many military courses such as skydiving, combat diver training etc.) actively work to mitigate the negative impact of SERE stress and to promote healthy, psychologically adaptive responses during stress exposure. This active and positive training environment - in concert with the students' voluntary participation in the training and ability to withdraw therefrom whenever they wish - makes the SERE environment and the impact of SERE stress qualitatively different from the conditions described in the Complaint. The focus of any military training program is to prepare men and women to fight for their country. The focus at SERE - to strengthen these men and women - is the same. But that focus includes preventing harm to these men and women. The focus of the program designed by the defendants is then unlike, indeed it is the opposite, of the SERE program. Its goal appears to have been to break people psychologically and to induce psychological and neurobiological states of distress which, in the year 2002, were known by the scientific community to have no positive benefits and confer significant damaging effects on psychological and neurobiological functioning.

Sincerely,

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