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Psychiatric Effects of Solitary Confinement

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Psychiatric Effects of Solitary Confinement[†]

Stuart Grassian^{*}

TABLE OF CONTENTS

PREFACE	327
I. OVERVIEW	327
II. SOLITARY CONFINEMENT CAN CAUSE SEVERE PSYCHIATRIC HARM	333
<i>A. Solitary Confinement Can Cause a Specific Psychiatric Syndrome</i>	333
1. The Specific Psychiatric Syndrome Associated with Solitary Confinement	335
2. This Syndrome has the Characteristics of an Acute Organic Brain Syndrome—A Delirium	337
<i>B. The Historical Experience with Solitary Confinement: The Nineteenth Century Experience</i>	338
1. The Origin of the American Penitentiary: The Nineteenth Century German Experience	338
2. Psychological Effects of Severe Isolation	341
<i>C. The Twentieth Century Experience: Prisoners of War, “Brain Washing,” and Experimental Research</i>	343
1. Prisoners of War and “Brain Washing”	343
2. Experimental Research on Sensory Deprivation	345
<i>D. Factors Effecting Response to Sensory Restriction and Solitary Confinement</i>	346
1. Differing Conditions of Isolation	346
2. The Perceived Intent of the Isolation Experience	347
3. Individual Differences in Response	347

[†] This article was prepared from a statement given to the Commission on Safety and Abuse in America's Prisons. As the article is an overview of the psychiatric effects of confinement throughout history it is not fully footnoted

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a.	Findings at Pelican Bay State Prison	349
b.	Attention Deficit and Antisocial Personality Disorders.....	350
c.	<i>Langley v. Coughlin</i>	351
d.	Effects on Psychologically More Resilient Inmates: <i>Baraldini v. Meese and Hameed v. Coughlin</i>	352
E.	<i>Long Term Effects of Solitary and Small Group Confinement</i>	353
III.	CONCLUSIONS	354
APPENDIX A: REPORTS OF PSYCHIATRIC DISTURBANCES IN OTHER CONDITIONS OF RESTRICTED ENVIRONMENTAL STIMULATION.....		
		356
I.	AVIATION	356
II.	SMALL GROUP CONFINEMENT.....	357
III.	POLAR HABITATION.....	358
IV.	EXPLORERS: SOLO VOYAGES.....	361
V.	MEDICAL CONDITIONS.....	362
A.	<i>Eye Patched Patients</i>	362
B.	<i>Poliomyelitis</i>	363
C.	<i>Cardiac Patients</i>	363
D.	<i>Hearing-Impaired Individuals</i>	364
E.	<i>Other Medical Patients</i>	365
VI.	OCCUPATIONAL SITUATIONS	365
VII.	ANIMAL STUDIES.....	365
APPENDIX B: THE NINETEENTH CENTURY GERMAN EXPERIENCE WITH SOLITARY CONFINEMENT.....		
		367
APPENDIX C: EXPERIMENTAL RESEARCH ON THE PSYCHIATRIC EFFECT OF PROFOUND SENSORY DEPRIVATION: FACTORS INFLUENCING VULNERABILITY TO PSYCHIATRIC HARM		
		373
I.	THE INFLUENCE OF EXPECTATION.....	373
II.	INDIVIDUAL DIFFERENCES IN RESPONSE	374
A.	<i>Effects of Sensory Deprivation on Antisocial Personality Disorder</i>	376
1.	Aversive Conditioning	376
a.	Ethical Considerations.....	378
b.	SHU Incarceration is not Aversive Conditioning	378

(i) The behavior being changed:.....	379
(ii) The “punishment”:.....	379
APPENDIX D: REPORTS OF THE LONG-TERM EFFECTS OF SOLITARY CONFINEMENT IN FORMER POLITICAL PRISONERS AND IN PRISONERS OF WAR: SOLITARY CONFINEMENT AS A MEANS OF “BRAIN WASHING” AND “INDOCTRINATING”	380

PREFACE

Dr. Grassian is a Board Certified Psychiatrist who was on the faculty of the Harvard Medical School for over twenty-five years. He has had extensive experience in evaluating the psychiatric effects of solitary confinement, and in the course of his professional involvement, has been involved as an expert regarding the psychiatric impact of federal and state segregation and disciplinary units in many settings. His observations and conclusions regarding this issue have been cited in a number of federal court decisions. The following statement is largely a redacted, non-institution and non-inmate specific, version of a declaration which was submitted in September 1993 in *Madrid v. Gomez*.¹ To enhance the readability of this statement, much of the supporting medical literature is described in the appendices to the statement.

I. OVERVIEW

Solitary confinement—that is the confinement of a prisoner alone in a cell for all, or nearly all, of the day with minimal environmental stimulation and minimal opportunity for social interaction—can cause severe psychiatric harm. It has indeed long been known that severe restriction of environmental and social stimulation has a profoundly deleterious effect on mental functioning; this issue has been a major concern for many groups of patients including, for example, patients in intensive care units, spinal patients immobilized by the need for prolonged traction, and patients with impairment of

1. 889 F. Supp. 1146 (N.D. Cal. 1995), *rev'd and remanded*, 150 F.3d 1030 (9th Cir. 1998).

their sensory apparatus (such as eye-patched or hearing-impaired patients). This issue has also been a very significant concern in military situations, polar and submarine expeditions, and in preparations for space travel.

The United States was actually the world leader in introducing prolonged incarceration, and solitary confinement, as a means of dealing with criminal behavior. The “penitentiary system” began in the United States, first in Philadelphia, in the early nineteenth century, a product of a spirit of great social optimism about the possibility of rehabilitation of individuals with socially deviant behavior.² The Americans were quite proud of their “penitentiary system” and they invited and encouraged important visitors from abroad to observe them.³ This system, originally labeled as the “Philadelphia System,” involved almost an exclusive reliance upon solitary confinement as a means of incarceration and also became the predominant mode of incarceration, both for post conviction and also for pretrial detainees, in the several European prison systems which emulated the American model.⁴

The results were, in fact, catastrophic. The incidence of mental disturbances among prisoners so detained, and the severity of such disturbances, was so great that the system fell into disfavor and was ultimately abandoned. During this process a major body of clinical literature developed which documented the psychiatric disturbances created by such stringent conditions of confinement.⁵

The paradigmatic psychiatric disturbance was an agitated confusional state which, in more severe cases, had the characteristics of a florid delirium, characterized by severe confusional, paranoid, and hallucinatory features, and also by intense agitation and random, impulsive, often self-directed violence. Such disturbances were often

2. An excellent history of the Philadelphia System is found in NORMAN JOHNSTON ET AL., *EASTERN STATE PENITENTIARY: CRUCIBLE OF GOOD INTENTIONS* (1994).

3. See DAVID ROTHMAN, *THE DISCOVERY OF THE ASYLUM* 81 (1971); see also GUSTAVE DE BEAUMONT & ALEXIS DE TOCQUEVILLE, *ON THE PENITENTIARY SYSTEM IN THE UNITED STATES AND ITS APPLICATION IN FRANCE*, http://www.law.du.edu/sterling/Content/ALH/Tocqueville_Pen.pdf; CHARLES DICKENS, *AMERICAN NOTES AND PICTURES FROM ITALY* (Leonee Ormond ed., Everymans Library 1997) (1842).

4. ROTHMAN, *supra* note 3, at 96–101.

5. See Appendix D (describing this literature).

observed in individuals who had no prior history of any mental illness. In addition, solitary confinement often resulted in severe exacerbation of a previously existing mental condition. Even among inmates who did not develop overt psychiatric illness as a result of solitary confinement, such confinement almost inevitably imposed significant psychological pain during the period of isolated confinement and often significantly impaired the inmate's capacity to adapt successfully to the broader prison environment.

It is both tragic and highly disturbing that the lessons of the nineteenth century experience with solitary confinement are today being so completely ignored by those responsible for addressing the housing and the mental health needs in the prison setting. For, indeed, the psychiatric harm caused by solitary confinement had become exceedingly apparent well over one hundred years ago. Indeed, by 1890, with *In re Medley*,⁶ the United States Supreme Court explicitly recognized the massive psychiatric harm caused by solitary confinement:

This matter of solitary confinement is not . . . a mere unimportant regulation as to the safe-keeping of the prisoner

. . . [E]xperience [with the penitentiary system of solitary confinement] demonstrated that there were serious objections to it. A considerable number of the prisoners fell, after even a short confinement, into a semi-fatuous condition, from which it was next to impossible to arouse them, and others became violently insane; others, still, committed suicide; while those who stood the ordeal better were not generally reformed, and in most cases did not recover sufficient mental activity to be of any subsequent service to the community.⁷

The consequences of the Supreme Court's holding were quite dramatic for Mr. Medley. Mr. Medley had been convicted of having murdered his wife. Under the Colorado statute in force at the time of the murder he would have been executed after about one additional

6. 134 U.S. 160 (1890).

7. *Id.* at 167–68.

month of incarceration in the county jail. But in the interim between Mr. Medley's crime and his trial the Colorado legislature had passed a new statute which called for the convicted murderer to be, instead, incarcerated in solitary confinement in the state prison during the month prior to his execution.⁸ Unhappily, when the legislature passed the new law it simultaneously rescinded the older law without allowing for a bridging clause which would have allowed for Mr. Medley's sentencing under the older statute.⁹

Mr. Medley appealed his sentencing under the new statute, arguing that punishment under this new law was so substantially more burdensome than punishment under the old law as to render its application to him *ex post facto*.¹⁰ The Supreme Court agreed with him, even though it simultaneously recognized that if Mr. Medley was not sentenced under the new law, he could not be sentenced at all.¹¹ Despite this, the Court held that this additional punishment of one month of solitary confinement was simply too egregious to ignore; the Court declared Mr. Medley a free man, and ordered his release from prison.¹²

Dramatic concerns about the profound psychiatric effects of solitary confinement have continued into the twentieth century, both in the medical literature and in the news. The alarm raised about the "brain washing" of political prisoners of the Soviet Union and of Communist China—and especially of American prisoners of war during the Korean War—gave rise to a major body of medical and scientific literature concerning the effects of sensory deprivation and social isolation, including a substantial body of experimental research.¹³

This literature, as well as my own observations, has demonstrated that, deprived of a sufficient level of environmental and social stimulation, individuals will soon become incapable of maintaining an adequate state of alertness and attention to the environment.

8. *Id.* at 162–63.

9. *Id.* at 166.

10. *Id.* at 162.

11. *Id.* at 166.

12. *Id.* at 174.

13. THE MANIPULATION OF HUMAN BEHAVIOR 2–3, 35 (Albert D. Biderman & Herbert Zimmer eds., 1961).

Indeed, even a few days of solitary confinement will predictably shift the electroencephalogram (EEG) pattern toward an abnormal pattern characteristic of stupor and delirium.

This fact is not surprising. Most individuals have at one time or another experienced, at least briefly, the effects of intense monotony and inadequate environmental stimulation. After even a relatively brief period of time in such a situation an individual is likely to descend into a mental torpor or “fog,” in which alertness, attention, and concentration all become impaired. In such a state, after a time, the individual becomes increasingly incapable of processing external stimuli, and often becomes “hyperresponsive” to such stimulation. For example, a sudden noise or the flashing of a light jars the individual from his stupor and becomes intensely unpleasant. Over time the very absence of stimulation causes whatever stimulation is available to become noxious and irritating. Individuals in such a stupor tend to avoid any stimulation, and withdraw progressively into themselves and their own mental fog.

An adequate state of responsiveness to the environment requires both the ability to achieve and maintain an attentional set and the ability to shift attention. The impairment of alertness and concentration in solitary confinement leads to two related abnormalities: the inability to focus, and the inability to shift attention. The inability to focus (to achieve and maintain attention) is experienced as a kind of dissociative stupor—a mental “fog” in which the individual cannot focus attention, and cannot, for example, grasp or recall when he attempts to read or to think.

The inability to shift attention results in a kind of “tunnel vision” in which the individual’s attention becomes stuck, almost always on something intensely unpleasant, and in which he cannot stop thinking about that matter; instead, he becomes obsessively fixated upon it. These obsessional preoccupations are especially troubling. Individuals in solitary confinement easily become preoccupied with some thought, some perceived slight or irritation, some sound or smell coming from a neighboring cell, or, perhaps most commonly, by some bodily sensation. Tortured by it, such individuals are unable to stop dwelling on it. In solitary confinement ordinary stimuli become intensely unpleasant and small irritations become maddening. Individuals in such confinement brood upon normally

unimportant stimuli and minor irritations become the focus of increasing agitation and paranoia. I have examined countless individuals in solitary confinement who have become obsessively preoccupied with some minor, almost imperceptible bodily sensation, a sensation which grows over time into a worry, and finally into an all-consuming, life-threatening illness.

Individuals experiencing such environmental restriction find it difficult to maintain a normal pattern of daytime alertness and nighttime sleep. They often find themselves incapable of resisting their bed during the day—incapable of resisting the paralyzing effect of their stupor—and yet incapable of any restful sleep at night. The lack of meaningful activity is further compounded by the effect of continual exposure to artificial light and diminished opportunity to experience natural daylight. And the individual's difficulty in maintaining a normal day-night sleep cycle is often far worsened by constant intrusions on nighttime dark and quiet, such as steel doors slamming shut, flashlights shining in their face, and so forth.

There are substantial differences in the effects of solitary confinement upon different individuals. Those most severely affected are often individuals with evidence of subtle neurological or attention deficit disorder, or with some other vulnerability. These individuals suffer from states of florid psychotic delirium, marked by severe hallucinatory confusion, disorientation, and even incoherence, and by intense agitation and paranoia. These psychotic disturbances often have a dissociative character, and individuals so affected often do not recall events which occurred during the course of the confusional psychosis. Generally, individuals with more stable personalities and greater ability to modulate their emotional expression and behavior and individuals with stronger cognitive functioning are less severely affected. However, all of these individuals will still experience a degree of stupor, difficulties with thinking and concentration, obsessional thinking, agitation, irritability, and difficulty tolerating external stimuli (especially noxious stimuli).

Moreover, although many of the acute symptoms suffered by these inmates are likely to subside upon termination of solitary confinement, many—including some who did not become overtly psychiatrically ill during their confinement in solitary—will likely suffer permanent harm as a result of such confinement. This harm is

most commonly manifested by a continued intolerance of social interaction, a handicap which often prevents the inmate from successfully readjusting to the broader social environment of general population in prison and, perhaps more significantly, often severely impairs the inmate's capacity to reintegrate into the broader community upon release from imprisonment.

Many inmates housed in such stringent conditions are extremely fearful of acknowledging the psychological harm or stress they are experiencing as a result of such confinement. This reluctance of inmates in solitary confinement is a response to the perception that such confinement is an overt attempt by authorities to "break them down" psychologically, and in my experience, tends to be more severe when the inmate experiences the stringencies of his confinement as being the product of an arbitrary exercise of power, rather than the fair result of an inherently reasonable process. Furthermore, in solitary confinement settings, mental health screening interviews are often conducted at the cell front, rather than in a private setting, and inmates are generally quite reluctant to disclose psychological distress in the context of such an interview since such conversation would inevitably be heard by other inmates in adjacent cells, exposing them to possible stigma and humiliation in front of their fellow inmates.

II. SOLITARY CONFINEMENT CAN CAUSE SEVERE PSYCHIATRIC HARM

A. Solitary Confinement Can Cause a Specific Psychiatric Syndrome

During the course of my involvement as an expert I have had the opportunity to evaluate the psychiatric effects of solitary confinement in well over two hundred prisoners in various state and federal penitentiaries. I have observed that, for many of the inmates so housed, incarceration in solitary caused either severe exacerbation or recurrence of preexisting illness, or the appearance of an acute mental illness in individuals who had previously been free of any such illness.

I became aware of the particular toxicity of solitary confinement when I first had the opportunity to evaluate prisoners in solitary

confinement as a result of my involvement in a class action lawsuit in Massachusetts, which challenged conditions in solitary confinement at the maximum security state penitentiary in Walpole, Massachusetts.¹⁴ The clinical observations I made in the course of my involvement in that lawsuit, coupled with my research into the medical literature concerning this issue, have formed the basis of two articles I have since published on this topic in peer-reviewed journals.¹⁵ My subsequent professional experience has included observations of similar phenomena in many other solitary confinement settings.

When I initially agreed to evaluate the Walpole prisoners I had not yet reviewed the literature on the psychiatric effects of solitary confinement and I was somewhat skeptical; I expected that inmates would feign illness and exaggerate whatever psychiatric symptomatology they suffered. I discovered, however, something very different. Contrary to my expectations, the prisoners appeared to be extremely defensive about the psychiatric problems they were suffering in Special Housing Unit (SHU); they tended to rationalize away their symptoms, avoid talking about them, or deny or distort their existence all in an apparent effort to minimize the significance of their reactions to isolation. Numerous interviews began with statements such as “solitary doesn’t bother me” or “some of the guys can’t take it—not me,” or even with the mention of a symptom and a simultaneous denial of its significance: “As soon as I got in I started cutting my wrists. I figured it was the only way to get out of here.”

As these interviews progressed the facile accounts gave way to descriptions of experiences that were very worrisome. For example, one inmate was unable to describe the events of the several days surrounding his wrist-slashing, nor could he describe his thoughts or feelings at the time. Similarly, the prisoner who said he could “take it” eventually came to describe panic, fears of suffocation, and paranoid distortions which he suffered while in isolation. Moreover,

14. *Libby v. Comm’r of Corr.*, 432 N.E.2d 486 (Mass. 1982).

15. See Stuart Grassian & Nancy Friedman, *Effects of Sensory Deprivation in Psychiatric Seclusion and Solitary Confinement*, 8 INT’L J.L. & PSYCHIATRY 49 (1986); Stuart Grassian, *Psychopathological Effects of Solitary Confinement*, 140 AM. J. PSYCHIATRY 1450 (1983).

the specific psychiatric symptoms reported were strikingly consistent among the inmates:

1. The Specific Psychiatric Syndrome Associated with Solitary Confinement

- a. Hyperresponsivity to External Stimuli: More than half the prisoners reported a progressive inability to tolerate ordinary stimuli. For example, “You get sensitive to noise, the plumbing system. Someone in the tier above me pushes the button on the faucet . . . It’s too loud, gets on your nerves. I can’t stand it. I start to holler.”

- b. Perceptual Distortions, Illusions, and Hallucinations: Almost a third of the prisoners described hearing voices, often in whispers and often saying frightening things to them. There were also reports of noises taking on increasing meaning and frightening significance. For example, “I hear noises, can’t identify them—starts to sound like sticks beating men, but I’m pretty sure no one is being beaten . . . I’m not sure.” These perceptual changes at times became more complex and personalized:

They come by with four trays; the first has big pancakes. I think I am going to get them. Then someone comes up and gives me tiny ones—they get real small, like silver dollars. I seem to see movements, real fast motions in front of me. Then seems like they are doing things behind your back, can’t quite see them. Did someone just hit me? I dwell on it for hours.

- c. Panic Attacks: Well over half the inmates interviewed described severe panic attacks while in SHU.

- d. Difficulties with Thinking, Concentration, and Memory: Many reported symptoms of difficulty in concentration and memory. One prisoner described his experience, “I can’t concentrate, can’t read . . . Your mind’s narcotized. Sometimes I can’t grasp words in my mind that I know. Get stuck, have to think of another word. Memory’s going. You feel like you are losing something you might not get back.” In some cases this problem was far more severe, leading to acute psychotic, confusional states. One prisoner had slashed his wrists during such a state and his confusion and disorientation had actually been noted in his medical record.

e. **Intrusive Obsessional Thoughts: Emergence of Primitive Aggressive Ruminations:** Almost half the prisoners reported the emergence of primitive aggressive fantasies of revenge, torture, and mutilation of the prison guards. In each case the fantasies were described as entirely unwelcome, frightening, and uncontrollable. For example, one prisoner recounted

I try to sleep sixteen hours a day, block out my thoughts; muscles tense, think of torturing and killing the guards; lasts a couple of hours. I can't stop it. Bothers me. Have to keep control. This makes me think I'm flipping my mind . . . I get panicky, thoughts come back—pictured throwing a guard in lime—eats away at his skin, his flesh—torture him—try to block it out, but I can't.

f. **Overt Paranoia:** Almost half the prisoners interviewed reported paranoid and persecutory fears. Some of these persecutory fears were short of overt psychotic disorganization. For example, one prisoner recalled “sometimes I get paranoid—think they meant something else. Like a remark about Italians. Dwell on it for hours. Get frantic. Like when they push buttons on the sink. Think they did it just to annoy me.” In other cases this paranoia deteriorated into overt psychosis:

Spaced out. Hear singing, people's voices, ‘Cut your wrists and go to Bridgewater and the Celtics are playing tonight.’ I doubt myself. Is it real? . . . I suspect they are putting drugs in my food, they are putting drugs in my cell . . . The Reverend, the priest, even you, you're all in cahoots in the Scared Straight Program.

g. **Problems with Impulse Control:** Slightly less than half of the prisoners reported episodes of loss of impulse control with random violence: “I snap off the handle over absolutely nothing. Have torn up mail and pictures, throw things around. Try to control it. Know it only hurts myself.” Several of these prisoners reported impulsive self-mutilation; “I cut my wrists many times in isolation. Now it seems crazy. But every time I did it, I wasn't thinking—lost control—cut myself without knowing what I was doing.”

2. This Syndrome has the Characteristics of an Acute Organic Brain Syndrome—A Delirium

Clearly, these symptoms were very dramatic. Moreover, they appeared to form a discreet syndrome—that is, a constellation of symptoms occurring together and with a characteristic course over time, thus suggestive of a discreet illness. Moreover, this syndrome was strikingly unique; some of the symptoms described above are found in virtually no other psychiatric illness. The characteristic acute dissociative, confusional psychoses are a rare phenomenon in psychiatry. Similarly, cases of random, impulsive violence in the context of such confusional state is exceedingly rare. But the most unique symptoms in this cluster are the striking and dramatically extensive perceptual disturbances experienced by the isolated person. Indeed, these disturbances are almost pathognomonic of the syndrome, meaning they are symptoms virtually found nowhere else. For example, loss of perceptual constancy (objects becoming larger and smaller, seeming to “melt” or change form, sounds becoming louder and softer, etc.) is very rare and, when found, is far more commonly associated with neurological illness (especially seizure disorders and brain tumors affecting sensory integration areas of the brain) than with primary psychiatric illness.¹⁶

In addition, functional psychiatric illness very rarely presents with such severe and florid perceptual distortions, illusions, and hallucinations simultaneously affecting multiple perceptual modalities—auditory, visual, olfactory, tactile, and kinesthetic.¹⁷

Similarly, hyperresponsivity to external stimuli with a dysesthetic (subjectively painful) response to such stimuli, is likewise rare. In fact, it is exceedingly rare; so rare that appearance of this symptom also might suggest an organic brain dysfunction etiology.¹⁸

16. When seen in primary psychiatric illness, it is basically only seen in especially severe, insidious, early onset schizophrenia—the kind of schizophrenic illness which has always been thought to clinically “feel” like a fundamentally biological/neurologic disease.

17. In fact, in the more common psychotic illnesses such as schizophrenia and psychotic depression, auditory hallucinations are by far the most common type; visual hallucinations come a distant second; and hallucinations in all other modalities are actually very uncommon. Moreover, combined modality hallucinations (other than the combination of auditory with visual) are exceedingly rare.

18. This symptom is similar, for example, to the experience many people have during a

Thus, the fact that all of these quite unusual symptoms ran together in the same syndrome was itself a clear confirmation of the distinct nature of this syndrome. While this syndrome is strikingly atypical for the functional psychiatric illnesses, it is quite characteristic of an acute organic brain syndrome: delirium, a syndrome characterized by a decreased level of alertness and EEG abnormalities; by the same perceptual and cognitive disturbances, fearfulness, paranoia, and agitation; and random, impulsive, and self-destructive behavior which I observed in the Walpole population.

Moreover, delirium is a syndrome which is known to result from the type of conditions, including restricted environmental stimulation, which are characteristic of solitary confinement. Even the EEG abnormalities characteristic of delirium have been observed in individuals exposed to conditions of sensory deprivation. By now the potentially catastrophic effects of restricted environmental stimulation have been the subject of a voluminous medical literature; annual international symposia are being held on the subject, and the issue has even found its way into the popular media. The literature is summarized in the appendices to this statement.

*B. The Historical Experience with Solitary Confinement: The
Nineteenth Century Experience*

1. The Origin of the American Penitentiary: The Nineteenth
Century German Experience

Preindustrial societies had often not made any fundamental distinction between deviant behavior seen as the product of “criminal intent” as opposed to behavior seen as stemming from “mental illness.”¹⁹ For such societies, deviant behavior—whatever its origins—was a social evil that was deeply feared and cruelly punished.

febrile illness of finding any touching of their body exceedingly unpleasant, or the inability of a patient with a headache to tolerate an even ordinary volume of sound, or the inability of some pregnant women to tolerate even ordinary smells without becoming nauseated.

19. ROTHMAN, *supra* note 3, at 4–5, 62–65.

In Colonial America the Salem witch trials were but one example of a continuing tendency to equate “lunacy” with “demonic possession” and, ultimately, with “evil.”²⁰ Deviant behavior was naturally feared and hated; the instinctive response was to punish it cruelly, lock it away, banish it, or kill its perpetrator. Thus, in Colonial America generally, the social response to deviant behavior was relatively simple: the protection of the larger society was paramount, while the distinction between “illness” and “evil” was far less critical. Indeed, the social response to deviance largely stemmed from the severe puritanical belief in innate human evil that deserved violent retaliation such as whipping, pillories, stockades, brandings, and, ultimately, the gallows. At times, when there was a more “humane” response to persons viewed as suffering from lunacy this response consisted simply of keeping the individual caged under lock and key, often for the rest of his life.

But in the early nineteenth century, a surge of great social optimism swept over America, and along with this grew a belief in the possibility of social reform, perhaps an overly optimistic faith in the possibility of rehabilitation of persons whose behavior was deviant.²¹ Not coincidentally, this spirit gave rise virtually simultaneously to two great social reform movements in the United States: the development of large mental hospitals and the construction of the first large penitentiaries.

Both of these institutions were founded upon a similar premise—namely, that psychological and social deviance was largely a result of the evils and stresses of “modern society,” and both held a fundamental belief that healing would naturally occur if the deviant individual was removed from the evils of the larger society, and thus enabled to know his own true nature.²²

In the case of the mental hospital this belief gave rise to the concept of a healing, pastoral, therapeutic community.²³ But, in the case of the penitentiary, an additional safeguard was obviously

20. GEORGE IVES, A HISTORY OF PENAL METHODS: CRIMINALS, WITCHES, LUNATICS 58–59, 68–73 (reprint 1970) (1914).

21. ROTHMAN, *supra* note 3, at 57–58, 79.

22. *Id.* at 82.

23. *Id.* at 133.

required: the inmates clearly had to be protected, not only from the evil influences of the broader society, but also from the evil influences of each other.²⁴ The proper approach thus appeared to be to give each inmate the opportunity to live a life alone, like a penitent monk in his own monastic cell.

Thus, the earliest American penitentiaries were, generally, systems of rigid solitary confinement.²⁵ Extravagant attention was paid to the design of these institutions, to ensure the absolute and total isolation of the offender from any evil and corrupting influences.²⁶ The Philadelphia Prison, completed in 1829, was particularly conscientious in this regard:

The arrangements . . . guaranteed that convicts would avoid all contamination and follow a path to reform. Inmates remained in solitary cells for eating, sleeping, and working No precaution against contamination was excessive. Officials placed hoods over the head of a new prisoner when marching him to his cell so he would not see or be seen by other inmates.

. . . Thrown upon his own innate sentiments, with no evil example to lead him astray, . . . the criminal would start his rehabilitation. Then, after a period of total isolation, without companions, books, or tools, . . . [h]e would return to the community cured of vice and idleness, to take his place as a responsible citizen.²⁷

The American penitentiary, and the Philadelphia System, became world-famous; no important visitor to the United States neglected to tour its penitentiaries and to bring back their principles for emulation in Europe. Some such as Alexis de Tocqueville of France and Nicholas Julius from Prussia came specifically for that purpose.²⁸ Tocqueville wrote of the utter, “perfect” desolation of the American

24. *Id.* at 83.

25. *Id.*

26. *Id.* at 82–83.

27. *Id.* at 85–86.

28. *Id.* at 81.

penitentiary, of the “profound silence” within its “vast walls,” likening it to the silence of death.²⁹

2. Psychological Effects of Severe Isolation

The openness with which these institutions were held up to public scrutiny led in time to open concern about the psychological effects of such confinement. During a tour of the United States in 1842, Charles Dickens wrote with pathos of the Philadelphia Prison:

The system here is rigid, strict, and hopeless solitary confinement. . . . Over the head and face of every prisoner who comes into the melancholy house, a black hood is drawn, and in this dark shroud, . . . he is led to the cell from which he never again comes forth, until his whole term of imprisonment has expired. He is a man buried alive dead to everything but torturing anxieties and horrible despair.

. . . .

The first man I saw . . . answered . . . always with a strange kind of pause He gazed about him and in the act of doing so fell into a strange stare as if he had forgotten something.

In another cell was a German, . . . a more dejected, broken-hearted, wretched creature, it would be difficult to imagine. . . .

There was a sailor [w]hy does he stare at his hands and pick the flesh open, upon the fingers, and raise his eyes for an instant . . . to those bare walls . . . ?³⁰

American concern about the effects of rigid solitary confinement began as early as the 1830s.³¹ Statistical comparisons began to be made between the Philadelphia system and its chief competitor: the Auburn system prevailing in New York State at the Auburn and Sing-Sing penitentiaries.³² The latter system also utilized solitary

29. *Id.* at 97.

30. P. Herbert Liederman, *Man Alone: Sensory Deprivation and Behavioral Change*, 8 CORRECTIONAL PSYCHIATRY & J. SOC. THERAPY 64, 66 (1962).

31. ROTHMAN, *supra* note 3, at 87–88.

32. *Id.* at 88.

confinement, but less rigidly; inmates left their cells to work together in workshops and exercise in a common courtyard, although here, too, absolute and strict silence was maintained at all times.³³ Statistical comparisons began to generate evidence that “[i]t was unnatural . . . to leave men in solitary, day after day, year after year; indeed, it was so unnatural that it bred insanity.”³⁴ The Philadelphia Prison system appeared to have a higher incidence not only of insanity but also of physical disease and death than its New York State system counterpart.³⁵

Meanwhile, the American system had been emulated in many major European prisons, such as at Halle, Germany.³⁶ Although the Americans had been the world leaders in instituting rigid solitary confinement in their penitentiary system, German clinicians eventually assumed the task of documenting its demise. Between 1854 and 1909, thirty-seven articles appeared in German scientific journals on the subject of psychotic disturbances among prisoners, summarizing years of work and hundreds of cases. A major review of this literature was published in 1912.³⁷ A summary and synthesis of this rather large body of work appears as an appendix to this article.³⁸

But it should be noted that interest in the problem was not purely academic; psychotic disturbances among prisoners were of such frequency in these prisons that they attracted administrative as well as clinical concern, and great effort was made to explain this disturbing incidence. Thus, the literature covered a variety of issues: speculation, for example, on the “moral degeneracy” of the prison population; comparison of the psychopathology of those who committed “crimes of passion” with those who committed “crimes against property”; or documentation of the incidence of the major diagnostic categories of the time (for example, “circular insanity,” “alcoholic psychoses,” epilepsy, and general paresis) among the prison population.

33. *Id.* at 95, 97.

34. *Id.* at 87.

35. *Id.* at 87–88.

36. See PAUL NITSCHKE & KARL WILMANN, THE HISTORY OF THE PRISON PSYCHOSES (Francis M. Barnes, Jr. & Bernard Glueck trans., 1912).

37. See *id.*

38. See Appendix B.

However, multiple reports based on careful clinical observation suggested that a substantial majority of these prison psychoses were direct reactions to the conditions of imprisonment itself. Gradually, a clinically distinguishable syndrome of acute reactive prison psychoses began to be defined. Different variables were considered in attempting to explain the etiology of these reactive prison psychoses, including long versus short durations of imprisonment, or imprisonment of those already convicted versus imprisonment while awaiting trial. However, the most consistent factor described, reported in over half the total literature, was solitary confinement.

C. The Twentieth Century Experience: Prisoners of War, "Brain Washing," and Experimental Research

1. Prisoners of War and "Brain Washing"

Unfortunately, other than some anecdotal reports, there was little discussion of the psychological effects of solitary confinement in the medical literature during the first half of the twentieth century. Undoubtedly, this was in part a consequence of the disastrous earlier experience with such confinement. As statistical evidence accumulated during the nineteenth century that solitary confinement produced a very disturbing incidence of insanity, physical disease, and death the system fell into disrepute and, with this, it had changed from an open, optimistic experiment in social reform into a hidden, secretive place of punishment and control.

Its devastating psychological impact, however, did not change, a fact which became suddenly and very painfully evident in the 1950s as the American public began hearing the frightening and dramatic reports of "brain washing" of American prisoners of war in Korea—reports that alterations in the sensory environment were being intentionally imposed upon these prisoners in a seemingly Orwellian attempt to profoundly disrupt their psychological equilibrium.³⁹

By the 1950s, reports had already appeared of major psychiatric disturbances among survivors of prolonged solitary confinement in

39. Lawrence E. Hinkle, Jr., *The Physiological State of the Interrogation Subject as It Affects Brain Function*, in *THE MANIPULATION OF HUMAN BEHAVIOR*, *supra* note 13, at 35.

war,⁴⁰ but during the decade of the Korean War major attention was riveted on the occurrence of these disturbances not only in war but in a variety of other settings as well. In 1956 the Group for the Advancement of Psychiatry (GAP) held a symposium, “Factors Used to Increase the Susceptibility of Individuals to Forceful Indoctrination,” to study methods used by the Chinese and Russian Communists to “indoctrinate” and “break the will” of political prisoners and prisoners of war.⁴¹ Dr. Milton Meltzer, former Chief Medical Officer at Alcatraz Federal Penitentiary, contributed his observations of psychiatric disturbances among prisoners exposed to punitive solitary confinement at Alcatraz.⁴² These prisoners were rarely confined for periods beyond one week.⁴³ Despite this, Dr. Meltzer described acute psychotic breakdowns among prisoners so confined; his descriptions closely paralleled the observations at Walpole:

The motor effects ranged from occasional tense pacing, restlessness and sense of inner tension with noise making, yelling, banging and assaultiveness at one extreme, to a kind of regressed, dissociated, withdrawn, hypnoid and reverie-like state at the other. . . .

... [T]he sense of self, the ego and ego boundary phenomena are profoundly affected by the isolation.⁴⁴

In the same symposium Dr. John Lilly of the National Institute of Mental Health noted that despite the importance of other factors which tended to “weaken personalities and make them more susceptible to [forced indoctrination]”—such as semi-starvation, physical pain and injury, and sleep deprivation—social and sensory isolation was still the central pathogenic factor in such confinement.⁴⁵

40. See, e.g., CHRISTOPHER BURNEY, SOLITARY CONFINEMENT (1952).

41. See GROUP FOR THE ADVANCEMENT OF PSYCHIATRY, FACTORS USED TO INCREASE THE SUSCEPTIBILITY OF INDIVIDUALS TO FORCEFUL INDOCTRINATION (1956).

42. *Id.* at 96–103.

43. *Id.* at 98.

44. *Id.*

45. *Id.* at 89.

2. Experimental Research on Sensory Deprivation

An experimental model was therefore designed to study the effect of such sensory deprivation; this research, conducted during the 1950s and early 1960s, primarily at Harvard and McGill University Medical Centers, was in fact funded in large part by the United States government—and especially by the Department of Defense and the Central Intelligence Agency. This research is described in an appendix to this article.⁴⁶ Its relevant conclusions can, however, be described relatively briefly:

In these studies subjects were placed in a situation designed for maximum reduction perceptually informative external stimuli (light-proof, sound-proof rooms; cardboard tubes surrounding the arms and hands to reduce proprioceptive and tactile sensation; and so on).⁴⁷ The research revealed that characteristic symptoms generally developed in such settings. These symptoms included perceptual distortions and illusions in multiple spheres (visual, auditory, tactile, olfactory); vivid fantasies, often accompanied by strikingly vivid hallucinations in multiple spheres; derealization experiences; and hyperresponsivity to external stimuli. What was also clear, however, was that while some subjects tolerated such experiences well, many did not, and characteristic syndromes were observed, including the above symptoms and cognitive impairment; massive free-floating anxiety; extreme motor restlessness; emergence of primitive aggressive fantasies which were often accompanied by fearful hallucinations; and a decreased capacity to maintain an observing, reality-testing ego function. In some cases an overt psychosis supervened with persecutory delusions and, in other cases, a marked dissociative, catatonic-like stupor (delirium) with mutism developed. EEG recordings confirmed the presence of abnormalities typical of stupor and delirium.

These findings clearly demonstrated that this experimental model did reproduce the findings in the non-experimental situations,

46. See Appendix C.

47. See, e.g., CHARLES A. BROWNFIELD, *ISOLATION: CLINICAL AND EXPERIMENTAL APPROACHES* (1965); *SENSORY DEPRIVATION: A SYMPOSIUM HELD AT HARVARD MEDICAL SCHOOL* (Philip Solomon et al. eds., 1961) [hereinafter *SENSORY DEPRIVATION—HARVARD*].

including the findings among prisoners of war held in solitary confinement.

D. Factors Effecting Response to Sensory Restriction and Solitary Confinement

Much of the subsequent research in this area attempted to delineate variables which might explain these differing outcomes. These variables can be divided into two categories: i) differences among various conditions of perceptual deprivation, and ii) differences in preexisting personality functioning among individuals experiencing such conditions.

1. Differing Conditions of Isolation

One of the factors that was commonly cited in the research was the intensity and duration of the sensory deprivation. More severe sensory restriction, the presence of noxious stimulation, and longer duration of the sensory deprivation experience have all been associated with an increased risk of adverse psychiatric consequences.

In my experience, conditions experienced by inmates in various prison solitary confinement settings generally bear some similarities (a cell of roughly fifty to eighty square feet; approximately twenty-two and one-half hours per day locked in the cell; about one hour per day of yard exercise, five out of the seven days each week), in other respects the conditions are fairly variable. For example, some cells have barred doors, which allow better ventilation, sound transmission, and visual connection with the outside environment than do mesh steel doors; solid steel doors are the most restrictive—especially when they are either hinged or slide shut with almost no air gap from the wall. Moreover, administrative conditions regarding the amount and circumstances of visitation, the availability of reading material and television, and so forth are all factors which vary from institution to institution, and even from time to time within a given institution.

2. The Perceived Intent of the Isolation Experience

In addition to the factors described above, another critical factor in determining the effect of isolation appears to be the perceived intent of the isolation. Experimental research has demonstrated that an individual who receives clues which cause him to experience the isolation situation as potentially threatening is far more likely to develop adverse psychiatric reactions to the isolation experience.⁴⁸ Conversely, if the subject has reason to believe the situation is likely to be benign he will be far more likely to tolerate or even enjoy it.⁴⁹ Among the latter group of subjects who tolerated isolation well, many reported pleasant or at least non-threatening visual imagery, fantasy, and hallucinatory experiences.⁵⁰ “His mind may begin to wander, engage in daydreams, slip off into hypnagogic reveries with their attendant vivid pictorial images . . . he may be quietly having sexual or other pleasurable thoughts.”⁵¹

This finding is perhaps not surprising. It appears that sensory restriction produces perceptual disturbances and illusions which are analogous to those produced by hallucinogenic drugs, and clearly, while there are some individuals who could be said to have volunteered to undergo such hallucinatory, psychotic-like experiences it must be almost uniformly terrifying to be forced to undergo an experience similar to that induced by hallucinogenic drugs.

3. Individual Differences in Response

Many studies have demonstrated that there is great variability among individuals in regard to their capacity to tolerate a given condition of sensory restriction. This variability helps to provide further insight into the nature of the toxic effect of such isolation conditions, and provides striking corroboration of the fact that such

48. See Nancy A. Wright & David S. Abbey, *Perceptual Deprivation Tolerance and Adequacy of Defenses*, 20 PERCEPTUAL & MOTOR SKILLS 35 (1965).

49. Leo Goldberger, *Experimental Isolation: An Overview*, 122 AM. J. PSYCHIATRY 774, 777 (1966).

50. *Id.*

51. *Id.*

deprivation of environmental stimulation, especially when of prolonged duration, is toxic to brain functioning and causes symptoms characteristic of stupor and delirium.

Generally, individuals with mature, healthy personality functioning and of at least average intelligence are most able to tolerate the regressive pull and perceptual intrusions of such isolation situations. On the other hand, individuals with primitive or psychopathic functioning or borderline cognitive capacities, impulse-ridden individuals, and individuals whose internal emotional life is chaotic or fearful are especially at risk for severe psychopathologic reactions to such isolation.⁵²

Moreover, there is clear evidence that, in a situation of restricted environmental stimulation, preexisting central nervous system dysfunction is a major predisposing factor to the development of adverse psychiatric reactions and of overt delirium. For example, in one study of patients suffering visual deprivation following eye surgery (eye-patched patients), those patients with preexisting central nervous system dysfunction were found to be at especially high risk to develop symptoms of delirium.⁵³ Further, the presence of a preexisting personality disorder or impairment of psychosocial functioning was associated with increased risk of incapacitating fearfulness, paranoia, agitation, and irrational aggression toward staff.⁵⁴

In addition, individuals may at times be exposed to situations which cause impairment of central nervous system functioning. Such situations—especially if they impair the individual's state of alertness (for example, sleep deprivation, abnormal sleep-wake cycles, or the use of sedating medication) will substantially increase the individual's vulnerability to the development of delirium. Delirium among post-surgical patients and the so-called "ICU psychoses" are examples of this phenomenon.⁵⁵ One of the characteristic difficulties

52. See Appendix C (describing these studies in more detail).

53. Eugene Ziskind, *Isolation Stress in Medical and Mental Illness*, 168 J. AM. MED. ASS'N 1427, 1428 (1958).

54. Hillel Klein & Rafael Moses, *Psychological Reaction to Sensory Deprivation in Patients with Ablatio Retinae*, 24 PSYCHOTHERAPY & PSYCHOSOMATICS 41, 49–51 (1974). A more extensive review of this literature is contained in Appendix A to this declaration.

55. Appendix A discusses this issue in more detail.

experienced by inmates in solitary confinement is abnormal sleep-wake cycles and impaired sleep.

a. Findings at Pelican Bay State Prison

These findings received further corroboration in my observations of inmates at Pelican Bay State Prison, California. In 1991–1992, as part of my participation in *Madrid v. Gomez*—a class-action lawsuit challenging conditions at Pelican Bay State Prison, a new “supermax” facility in California⁵⁶—I evaluated forty-nine inmates housed in the SHU at the institution and prepared a lengthy report to the federal court of my findings.⁵⁷ Many of the inmates I evaluated there suffered severe psychiatric disturbances while housed in Pelican Bay SHU, either springing up *de novo* while so incarcerated or representing a recurrence or severe exacerbation of preexisting illness. Of the forty-nine inmates I evaluated, at least seventeen were actively psychotic and/or acutely suicidal and urgently in need of acute hospital treatment, and twenty-three others suffered serious psychopathological reactions to solitary confinement, including (in several cases) periods of psychotic disorganization.

The clinical data at Pelican Bay also added striking corroboration to the conclusion that the severe and prolonged restriction of environmental stimulation in solitary confinement is toxic to brain functioning. The data demonstrated that the most severe, florid psychiatric illnesses resulting from solitary confinement tend to be suffered by those individuals with preexisting brain dysfunction. As noted before, I have observed a high incidence of preexisting central nervous system dysfunction among the inmates I evaluated in solitary confinement settings. This was also the case at Pelican Bay, and statistical analysis of the Pelican Bay data quite dramatically demonstrated that inmates with such preexisting vulnerability were the most likely to develop overt confusional, agitated, hallucinatory psychoses as a result of SHU confinement.

56. *Madrid v. Gomez*, 889 F. Supp. 1146 (N.D. Cal. 1995), *rev'd and remanded*, 150 F.3d 1030 (9th Cir. 1998).

57. Much of the literature review and historical material in the present declaration is taken from my *Madrid* declaration.

b. Attention Deficit and Antisocial Personality Disorders

In addition, research regarding Attention Deficit Hyperactivity Disorder and Antisocial Personality Disorder demonstrated that these conditions are similarly associated with a particular inability to tolerate restricted environmental stimulation. There is increasing evidence that childhood impulsivity and Attention Deficit Hyperactivity Disorder bear some relationship to Antisocial Personality Disorder, in that both are characterized by impulsivity and stimulation-seeking behavior, and both involve biologically based abnormalities in central nervous system functioning. Moreover, the clinical literature demonstrates that individuals with Antisocial Personality Disorder are especially intolerant of restricted environmental stimulation. For example, the psychopathic individual has been characterized as pathologically “stimulation seeking,” “impulsive,” and “unable to tolerate routine and boredom.”⁵⁸

Given the exigencies of conducting clinical observations of inmates in solitary confinement it is not surprising that little systematic attempt has been made to elucidate the underlying psychological characteristics of those most at risk for developing severe psychopathological reactions to such isolation. However, among the clinical reports on Ganser’s Syndrome, a related condition, in non-prison populations are several studies of patients in psychiatric hospitals.⁵⁹ These patients were, of course, available for extensive psychological assessment and observation, and these reports described the majority of these patients as suffering long-standing hysterical character disorders, having problems with severe impulsivity, childhood truancy, and antisocial behavior patterns.⁶⁰

Thus, the medical literature demonstrates that individuals whose internal emotional life is chaotic and impulse-ridden and individuals with central nervous system dysfunction may be especially prone to

58. Herbert C. Quay, *Psychopathic Personality as Pathological Stimulation-Seeking*, 122 AM. J. PSYCHIATRY 180, 180 (1965). Appendix B contains a more detailed discussion.

59. See, e.g., Merle R. Ingraham & David M. Moriarty, *A Contribution to the Understanding of the Ganser Syndrome*, 8 COMPREHENSIVE PSYCHIATRY 35 (1967); Rupert H. May et al., *The Ganser Syndrome: A Report of Three Cases*, 130 J. NERVOUS & MENTAL DISEASES 331 (1960).

60. May et al., *supra* note 59, at 331–36.

psychopathologic reactions to restricted environmental stimulation in a variety of settings. Yet, among the prison population, it is quite likely that these are the very individuals who are especially prone to committing infractions that result in stricter incarceration, including severe isolation and solitary confinement.

c. *Langley v. Coughlin*⁶¹

In the late 1980s I interviewed and reviewed the medical records of several dozen inmates confined in maximum security prisons in New York State, including a large group of women incarcerated at the maximum security women's prison for the state of New York at Bedford Hills. During the process of these evaluations it became clear that a very high percentage of these women had a history of serious emotional or organic mental difficulties. Many had severe cognitive limitations, were highly emotionally labile, impulse ridden, and prone to psychotic disorganization. In many cases the infraction which led to their original incarceration was an act which had been committed impulsively and chaotically. Under the stress of imprisonment these inmates became even more unable to conform their behavior to the requirements of their situation.

Inevitably, this resulted in their being sentenced to terms in the SHU, and once in the SHU their subsequent course was often a nightmare. Many became grossly disorganized and psychotic, smearing themselves with feces, mumbling and screaming incoherently all day and night, some even descending to the horror of eating parts of their own bodies.

The resulting lawsuit was ultimately settled by consent decree. The settlement provided injunctive relief as well as monetary damages both for the mentally ill inmates whose emotional condition had deteriorated during their incarceration in the SHU, and also for the non-mentally ill women who had been subjected to the bedlam of mental illness created in their SHU environment. The injunctive relief required the prison to begin to reframe the meaning it gave to

61. There are two companion cases: *Langley v. Coughlin*, 715 F. Supp. 522 (S.D.N.Y. 1989); and *Langley v. Coughlin*, 709 F. Supp. 482 (S.D.N.Y. 1989), *aff'd*, 888 F.2d 252 (2d Cir. 1989).

behavioral disturbances which they had previously responded to by further SHU time.⁶² Under the settlement the prison began to actively consider whether such disturbances were the result of organic personality disturbances, affective or impulse disorders, or even of schizophreniform illness. The result of these changes was apparently quite dramatic.

Many of the prisoners who had been in SHU began to be treated in a residential psychiatric unit within the prison. This unit had previously refused to treat such inmates, claiming that their security needs were greater than could be handled. When pressed to provide services as a result of the settlement not only did the unit discover that it was able to provide those services, but moreover discovered that the custodial and security needs of these inmates dramatically decreased when their behavioral disturbances were framed as psychiatric problems rather than as a security issue. Thus, as a result of the settlement of the lawsuit, all parties to the suit benefited—prisoners and the officers of the correctional facility alike. I followed the result of the litigation in my capacity as an expert member of the settlement.

d. Effects on Psychologically More Resilient Inmates:

*Baraldini v. Meese*⁶³ and *Hameed v. Coughlin*⁶⁴

In 1988 in the course of my involvement in *Baraldini v. Meese*, a class-action challenging the confinement of a small group of women in a subterranean security housing unit at the Federal Penitentiary in Lexington, Kentucky, I had the opportunity to interview several women who were in confinement in this facility. These women had been convicted of having committed politically motivated crimes, were all highly educated, and had a history of relatively strong psychological functioning prior to their confinement. None of these women developed the florid confusional psychosis described earlier in this affidavit, yet each of them demonstrated significant

62. *Langley*, 709 F. Supp. 482.

63. 691 F. Supp. 432 (D.D.C. 1988), *rev'd sub nom.*, *Baraldini v. Thornburgh*, 884 F.2d 615 (D.C. Cir. 1989).

64. 57 F.3d 217 (2d Cir. 1995).

psychopathological reactions to their prolonged confinement in a setting of severe environmental and social isolation. These included perceptual disturbances, free-floating anxiety, and panic attacks. These inmates also uniformly described severe difficulties in thinking, concentration, and memory; for example, one inmate reported that she was able to perform tasks requiring some mental effort—such as reading or writing—only for about the first three hours of the morning after she awoke; by then, her mind had become so slowed down, so much “in a fog,” that she was entirely unable to maintain any meaningful attention or expend any meaningful mental effort.

I have since evaluated a number of individuals who evidenced strong psychological adjustment prior to imprisonment. For example, in 1993 I evaluated Bashir Hameed, an inmate who had been incarcerated in the SHU at Shawangunk Correctional Facility and who had brought suit concerning his incarceration there. As I described in my testimony in that case, Mr. Hameed is an individual who evidences strong prior psychological adjustment and no prior psychiatric history, yet became significantly ill as a result of his SHU confinement.

E. Long Term Effects of Solitary and Small Group Confinement

Long-term studies of veterans of prisoner of war camps, and of kidnapping and hostage situations have demonstrated that while many of the acute symptoms I outlined above tend to subside after release from confinement, there are also long-term effects which may persist for decades.⁶⁵ These not only include persistent symptoms of post traumatic stress (such as flashbacks, chronic hypervigilance, and a pervasive sense of hopelessness), but also lasting personality changes—especially including a continuing pattern of intolerance of social interaction, leaving the individual socially impoverished and withdrawn, subtly angry and fearful when forced into social interaction.⁶⁶

65. See LAWRENCE E. HINKLE, JR. & HAROLD G. WOLFF, COMMUNIST INTERROGATION AND INDOCTRINATION OF “ENEMIES OF THE STATES” (1956).

66. This literature is reviewed in Appendix D to this declaration.

In addition, from time to time I have had the opportunity to evaluate individuals who had been incarcerated in solitary confinement several years previously. I have found the same pattern of personality change described above: these individuals had become strikingly socially impoverished and experienced intense irritation with social interaction, patterns dramatically different from their functioning prior to solitary confinement.

III. CONCLUSIONS

The restriction of environmental stimulation and social isolation associated with confinement in solitary are strikingly toxic to mental functioning, producing a stuporous condition associated with perceptual and cognitive impairment and affective disturbances. In more severe cases, inmates so confined have developed florid delirium—a confusional psychosis with intense agitation, fearfulness, and disorganization. But even those inmate who are more psychologically resilient inevitably suffer severe psychological pain as a result of such confinement, especially when the confinement is prolonged, and especially when the individual experiences this confinement as being the product of an arbitrary exercise of power and intimidation. Moreover, the harm caused by such confinement may result in prolonged or permanent psychiatric disability, including impairments which may seriously reduce the inmate's capacity to reintegrate into the broader community upon release from prison.

Many of the prisoners who are housed in long-term solitary confinement are undoubtedly a danger to the community and a danger to the corrections officers charged with their custody. But for many they are a danger not because they are coldly ruthless, but because they are volatile, impulse-ridden, and internally disorganized.

As noted earlier in this statement, modern societies made a fundamental moral division between socially deviant behavior that was seen as a product of evil intent, and such behavior that was seen as a product of illness. Yet this bifurcation has never been as simple as might at first glance appear. Socially deviant behavior can in fact be described along a spectrum of intent. At one end are those whose behavior is entirely “instrumental”—ruthless, carefully planned, and

rational; at the other are individuals whose socially deviant behavior is the product of unchecked emotional impulse, internal chaos, and often of psychiatric or neurological illness.

It is a great irony that as one passes through the levels of incarceration—from the minimum to the moderate to the maximum security institutions, and then to the solitary confinement section of these institutions—one does not pass deeper and deeper into a subpopulation of the most ruthlessly calculating criminals. Instead, ironically and tragically, one comes full circle back to those who are emotionally fragile and, often, severely mentally ill. The laws and practices that have established and perpetuated this tragedy deeply offend any sense of common human decency.

APPENDIX A:

REPORTS OF PSYCHIATRIC DISTURBANCES IN OTHER CONDITIONS OF
RESTRICTED ENVIRONMENTAL STIMULATION

The psychopathologic syndrome which I have described in the body of this article is found in other settings besides isolation in civil prisons. Some of these settings involve small group, rather than solitary isolation, and the studies have demonstrated that isolated groups comprising two individuals may be the most pathogenic of all. These studies also suggest that those individuals with below average intelligence and poor psychosocial adjustment prior to isolation developed more severe psychiatric difficulties during isolation. In some studies, such disturbances persisted at a one year follow-up after reentry.

I. AVIATION

One particular study, by Bennett, has described psychiatric disturbances among pilots of the British Royal Air Force who had been exposed in-flight to periods of restricted auditory and visual stimulation.⁶⁷ All of the groups he described became significantly anxious; many suffered full-blown panic attacks, and many experienced unusual sensations which they were very reluctant to describe. The most severely disturbed groups refused to expose themselves further to the isolation conditions of these flights. At all levels of impairment, however, anxiety was common (both panic and free-floating anxiety). Pilots reported anxiety symptoms such as feeling “hot and tense and powerless” and “nervous and afraid.”⁶⁸ Feelings of derealization, feelings of detachment from reality, and perceptual distortions were described. Some of these perceptual distortions were dangerous—such as having the impression that the aircraft was turning when it was not—and resulted in serious errors in

67. A.M. Hastin Bennett, *Sensory Deprivation in Aviation*, in SENSORY DEPRIVATION—HARVARD, *supra* note 47, at 161–73.

68. *Id.* at 164.

judgment like making the aircraft spiral dangerously downward after attempting to “correct” for what was incorrectly perceived as a turning aircraft.

Another study described strikingly similar symptoms among United States Navy pilots exposed to periods of in-flight isolation.⁶⁹ Among pilots who flew alone at high altitude (meaning in a situation of monotonous visual and sensory stimulation) and flying with a minimum of pilot activity, over one third experienced frightening feelings of unreality and became severely anxious.⁷⁰

II. SMALL GROUP CONFINEMENT

Many studies—both anecdotal and experimental—have been made of individuals confined together in small groups. Groups thus described have ranged in size from two to approximately sixty individuals, the larger groups include reports of men isolated on a Pacific island, in submarines, and on Antarctic expeditions.⁷¹ The most consistent finding was of dramatically increased levels of hostility, interpersonal conflict, and paranoia.⁷² Individuals exposed to such conditions also tend to become irrationally territorial, staking out “areas of exclusive or special use, [and] acting with hostility to trespasses by others.”⁷³

Confined groups comprising just two individuals may be the most pathogenic of all, associated with especially high rates of mutual paranoia and violent hostility. Admiral Byrd believed it to be extremely unsafe to staff an Antarctic base unit with just two men:

69. Brant Clark & Ashton Graybiel, *The Break-off Phenomenon*, 28 J. AVIATION MED. 121 (1957).

70. *Id.* at 122.

71. See Seward Smith, *Studies of Small Groups in Confinement*, in SENSORY DEPRIVATION: FIFTEEN YEARS OF RESEARCH 374–76 (John Peter Zubek ed., 1969) [hereinafter SENSORY DEPRIVATION: FIFTEEN YEARS]. For articles reporting effects in arctic environments, see Jeanette J. Cochrane & S.J.J. Freeman, *Working in Arctic and Sub-Arctic Conditions: Mental Health Issues*, 34 CAN. J. PSYCHIATRY 884 (1989); Eric Gunderson & Paul D. Nelson, *Adaptation of Small Groups to Extreme Environments*, AEROSPACE MED., Dec. 1963, at 1111; Charles S. Mullin & H.J.M. Connery, *Psychological Study at an Antarctic IGY Station*, 10 U.S. ARMED FORCES MED. J. 290 (1959).

72. Smith, *supra* note 71, at 377.

73. *Id.* at 380.

[I]t doesn't take two men long to find each other out. . . . [T]he time comes . . . when even his [campmate's] unformed thoughts can be anticipated, his pet ideas become a meaningless drool, and the way he blows out a pressure lamp or drops his boots on the floor or eats his food becomes a rasping annoyance. . . . Men who have lived in the Canadian bush know well what happens to trappers paired off this way

. . . During my first winter at Little America I walked for hours with a man who was on the verge of murder or suicide over imaginary persecutions by another man who had been his devoted friend.⁷⁴

III. POLAR HABITATION

Psychiatric disturbances have been described in Arctic and Antarctic inhabitants (explorers, researchers, and their support staff), spending varying periods in winter isolation. In these regions, winters last for up to nine months with weather conditions so cold (-100°F) that leaving the confines of the indoors is dangerous.⁷⁵ Typically, teams of work groups have fewer than fifty members who spend up to two years working in small quarters.⁷⁶ Small group isolation conditions at these stations have been compared to life in prisons by at least one researcher: "[T]he isolation imposed by the harsh environment [of the Antarctic] is rarely experienced outside penal conditions."⁷⁷

A review of the literature on the psychological adjustment to Antarctic living described a staff wintering over at a British Antarctic station; those of the staff who adjusted best tended to be socially mature, intelligent, reserved, and trusting individuals.⁷⁸ Similarly,

74. *Id.* at 381.

75. Gunderson & Nelson, *supra* note 71, at 1111.

76. *Id.*

77. Robert J. Biersner & Robert Hogan, *Personality Correlates of Adjustment in Isolated Work Groups*, 18 J. RESEARCH IN PERSONALITY 491, 491 (1989).

78. See Esther D. Rothblum, *Psychological Factors in the Antarctic*, 124 J. PSYCH. 253 (1990).

French, United States, and Australian studies revealed that intelligence and previous social adjustment predicted a decreased risk for psychiatric disturbance among workers at Antarctic stations.⁷⁹ On the other hand, lack of respect for authority and aggression were important markers for poor isolation adjustment.⁸⁰

Similarly, another study correlated outcome measures with psychological testing obtained prior to work station assignment.⁸¹ These researchers found specifically that persons with antisocial and psychotic tendencies were poor risks for efficient functioning in conditions of isolation.⁸²

As a result of these disturbing findings among Antarctic workers, systematic efforts have been made to provide psychological screening of potential station employees and to ameliorate the isolation conditions prevailing in such stations.⁸³ Despite these efforts, significant psychiatric disturbances have continued to be observed.⁸⁴ The fact that these individuals were confined in small groups rather than alone was not found to prevent these disturbances; indeed, one of the central pathogenic factors cited in this literature has been the interpersonal tension and hostility generated by small group confinement.⁸⁵

Studies have described a “winter-over syndrome” including progressively worsening depression, hostility, sleep disturbance, impaired cognitive functioning, and paranoia during small group winter confinement in the Antarctic.⁸⁶ Strikingly similar findings were reported by the United States Navy Medical Neuropsychiatric Research Unit, which found high incidences of sleep disturbance, depression, anxiety, aggression, somatic complaints, and a

79. *Id.* at 256; *see also* Smith, *supra* note 71, at 393–95.

80. Mullin & Connery, *supra* note 71, at 292.

81. *See* Morgan W. Wright et al., *Personality Factors in the Selection of Civilians for Isolated Northern Stations*, 8 CAN. PSYCHOLOGIST 23 (1967).

82. *Id.* at 29.

83. Cochrane & Freeman, *supra* note 71, at 889.

84. K. Natani & J. Shurley, *Sociopsychological Aspects of a Winter Vigil at South Pole Station*, in HUMAN ADAPTABILITY TO ANTARCTIC CONDITIONS 89–114 (Eugene Gunderson ed., Am. Geophysical Union 1974).

85. *See* Biersner & Hogan, *supra* note 77, at 491–96.

86. *See, e.g.,* R. Strange & W. Klein, *Emotional and Social Adjustment of Recent Winter-Over in Isolated Antarctic Stations*, 7 ANTARCTIC BIBLIOGRAPHY 229 (1974).

progressive impoverishment of social relationships as the winter progressed.⁸⁷ Psychiatric problems worsened as the length of time in this confinement increased; in one study of a group of Japanese winter-stationed in the Antarctic, periodic psychological testing revealed increasing levels of anxiety and depression as the winter progressed.⁸⁸ Similar findings have been described among a group of Americans stationed in the Antarctic.⁸⁹

A review of the literature on the psychological adjustment to Arctic life described a syndrome which parallels the Antarctic literature: sleep disturbances, apathy, irritability, cognitive dysfunction, hallucinations, depression, and anxiety were widely reported as a result of the small group isolation endured by inhabitants.⁹⁰ They also reported “depression, irritability, [and] easily provoked anger which may escalate into dramatic and florid acting out and, not surprisingly, a breakdown in relationships with other members of the group. . . . [I]nsomnia, pallor, loss of appetite, loss of interest, psychomotor retardation, paranoid ideation, [and] nonspecific hallucinations of light flashes and sudden movements [were also experienced].”⁹¹ Even when Arctic workers were adequately preselected by psychological screening, trained, and supported sleep difficulties, apathy, and irritability persisted.

Studies on reintegration into the home environment after Antarctic living found persisting problems and symptoms including sleep disturbances, cognitive slowing, emotional withdrawal, resentment of authority, indecisiveness, and poor communication even one year after reintegration.⁹²

Robert J. Biersner and Robert Hogan summarized the findings related to personality variables in the Arctic and Antarctic workers: “Individuals with high needs for novelty and new sensations, . . . who are emotionally unstable, or who are unconcerned with social

87. See E.K. Eric Gunderson, *Emotional Symptoms in Extremely Isolated Groups*, 9 ARCHIVES GEN. PSYCHIATRY 362 (1963); Gunderson & Nelson, *supra* note 71, at 1111–15.

88. Rothblum, *supra* note 78, at 253–73.

89. Gunderson & Nelson, *supra* note 71, at 1114.

90. See Cochrane & Freeman, *supra* note 71, at 889.

91. *Id.* at 887.

92. Rothblum, *supra* note 78, at 267.

approval seem unsuited for . . . such environments The opposite [traits are found in] those who adjust well.”⁹³

IV. EXPLORERS: SOLO VOYAGES

Anecdotal reports of shipwrecked sailors and individuals accomplishing long solo sea voyages have generally described “disturbances in attention and in organization of thought, labile and extreme affect, hallucinations and delusions.”⁹⁴ Dramatic anecdotal reports have appeared from time to time. Some of these were summarized in a review article by Dr. Philip Solomon, one of the lead scientists in the Harvard Medical School/Boston City Hospital group:

Christine Ritter in her very sensitive document *A Woman in the Polar Night*, reported that at times she saw a monster . . . [and] experienced depersonalization to the extent that she thought she and her companions were dissolving in moonlight ‘as though it were eating us up’ The Spitzbergen hunters use the term ran (strangeness) to describe these experiences⁹⁵

Tales of the sea have provided many accounts of hallucinatory phenomena. John Slocum sailed alone around the world . . . [In the South Atlantic] he suddenly saw a man, who at first he thought to be a pirate, take over the tiller

Walter Gibson, a soldier in the British Indian Army, was on a ship torpedoed in the Indian Ocean by the Japanese in World War II [The shipwrecked survivors] reported that “all of us at various stages in that first week became a prey to hallucinations” . . . [As the weeks passed] the feeling of comradeship disappeared and the men began to find themselves “watching our fellows covertly and suspiciously.”⁹⁶

93. Biersner & Hogan, *supra* note 77, at 495.

94. Peter Suedfeld, *Introduction and Historical Background*, in SENSORY DEPRIVATION: FIFTEEN YEARS, *supra* note 71, at 7.

95. Philip Solomon et al., *Sensory Deprivation: A Review*, 114 AM. J. PSYCHIATRY 357, 357–58 (1957).

96. *Id.*

Murder, suicide, and cannibalism followed as social controls dissolved.⁹⁷

V. MEDICAL CONDITIONS

A. Eye Patched Patients

Restricted environmental stimulation conditions also occur post-operatively and in certain medical conditions. In a study of one hundred American patients with macular degeneration of the retina, a high percentage of such patients experienced disturbing visual hallucinations.⁹⁸ Those patients who were relatively cognitively limited, those who were socially isolated, and those with simultaneous sensory impairment in another modality (for example, hearing-impaired patients) fared worst.⁹⁹ But other factors, including the presence of concomitant medical illness, did not appear to affect the incidence of hallucinations.¹⁰⁰

In an especially relevant study of eye patched patients, it was determined that psychologically well-adjusted patients (as assessed prior to surgery) tended not to develop visual hallucinations during the period when their eyes were patched, whereas those suffering preexisting personality disturbances did tend to develop such hallucinations.¹⁰¹ Among those patients who did develop hallucinations, almost half developed complex hallucinations involving human figures and with content suggesting serious preoccupations with themes of depression and anxiety.¹⁰² Moreover, among those patients who had both preexisting personality disturbances and difficulty with their premorbid psychosocial adjustment, eye patching produced severe psychiatric symptomatology, including: paranoid thoughts about being poisoned, physically harmed or attacked; psychomotor agitation; interpersonal

97. *Id.*

98. See Suzanne Holroyd et al., *Visual Hallucinations in Patients with Macular Degeneration*, 149 AM. J. PSYCHIATRY 1701, 1703 (1992).

99. *Id.* at 1703–04.

100. *Id.*

101. Klein & Moses, *supra* note 54, at 49.

102. *Id.*

aggressiveness; inability to comply with staff directives; fearful visual hallucinations; and incapacitating anxiety.¹⁰³ In this most disturbed group, symptoms had not remitted when observed one week after their eye patches were removed.¹⁰⁴

Other studies have also found patients to suffer from perceptual distortions, thinking disturbances, and mood changes following the visual deprivation that is part of postoperative recovery in eye surgery.¹⁰⁵ Furthermore, it was noted that “[i]n patients with . . . brain damage, there were also deliriod symptoms, e.g., confusion, disorientation, memory impairment, vivid hallucinations [and disorganized] hyperkinetic activity”¹⁰⁶ Finally, in C. Wesley Jackson’s extensive literature review of hospitalized eye patched patients, psychiatric disturbance was commonly found.¹⁰⁷ These patients suffered from unusual emotional, cognitive, and sensory-perceptual disturbances similar to those previously described.

B. Poliomyelitis

Polio patients confined to tank-type respirators have become psychotic as a direct result of such confinement; moreover, they became more ill, with more florid hallucinations and delusions, at night when sensory input was diminished.¹⁰⁸ The same florid hallucinatory, delusional psychosis has been found in other patients similarly confined in tank respirators.¹⁰⁹

C. Cardiac Patients

Patients with decompensated heart disease are at times placed on very strict bed rest; some of these patients have developed acute

103. *Id.* at 50.

104. *Id.*

105. See, e.g., Eugene Ziskind et al., *Observations on Mental Symptoms in Eye Patched Patients: Hypnagogic Symptoms in Sensory Deprivation*, 116 AM. J. PSYCHIATRY 893 (1960); Ziskind, *supra* note 53.

106. Ziskind et al., *supra* note 105, at 894.

107. See C. Wesley Jackson, Jr., *Clinical Sensory Deprivation: A Review of Hospitalized Eye-Surgery Patients*, in SENSORY DEPRIVATION: FIFTEEN YEARS, *supra* note 71, at 337–43.

108. Solomon et al., *supra* note 95, at 361.

109. *Id.* at 362.

confusional, paranoid, hallucinatory psychoses, especially at night during periods of decreased sensory input.

Studies of postoperative open heart surgery patients who were bed confined—their visual stimulation restricted to looking up at a white-tiled hospital room ceiling—revealed a high rate of disordered thinking, visual and auditory hallucinations, and disorientation.¹¹⁰ There is an extremely disturbing incidence of psychosis following open heart surgery, ranging in various studies from 14% to 30%.¹¹¹ Upon recovery these patients described their postoperative environment as a major pathogenic factor in producing their psychiatric illness.¹¹² Perceptual disturbances and emotional lability, as well as paranoia, depression, and obsessive-compulsive reactions to the restrictive postoperative environment have been documented in other studies as well.¹¹³

D. Hearing-Impaired Individuals

Another condition of restricted environmental stimulation leading to psychiatric disturbance involves the hearing impaired. Studies of the deaf consistently find significantly higher rates of paranoia in these individuals.¹¹⁴ High rates of paranoia have been reported in both the developmentally hearing impaired as well as those who

110. See, e.g., N. Egerton & J.H. Kay, *Psychological Disturbances Associated with Open Heart Surgery*, 110 BRIT. J. PSYCHIATRY 433 (1964); Donald S. Kornfeld et al., *Psychiatric Complications of Open-Heart Surgery*, 273 NEW ENG. J. MED. 287 (1965); Herbert R. Lazarus & Jerome H. Hagens, *Prevention of Psychosis Following Open-Heart Surgery*, 124 AM. J. PSYCHIATRY 1190 (1968); Larkin M. Wilson, *Intensive Care Delirium*, 130 ARCHIVES INTERNAL MED. 225 (1972).

111. Robert E. Lee & Patricia A. Ball, *Some Thoughts on the Psychology of the Coronary Care Unit Patient*, 75 AM. J. NURSING 1498, 1501 (1975).

112. Kornfeld et al., *supra* note 110, at 290.

113. See, e.g., Rosemary Ellis, *Unusual Sensory and Thought Disturbances After Cardiac Surgery*, 72 AM. J. NURSING 2021 (1972); Alvin G. Goldstein, *Hallucinatory Experience: A Personal Account*, 85 J. ABNORMAL PSYCHOL. 423 (1976); Linda Reckhow Thomson, *Sensory Deprivation: A Personal Experience*, 73 AM. J. NURSING 266 (1973); Lee & Ball, *supra* note 111.

114. See, e.g., Kenneth Z. Altshuler, *Studies of the Deaf: Relevance to Psychiatric Theory*, 127 AM. J. PSYCHIATRY 1521 (1971); F. Houston & A.B. Royse, *Relationship Between Deafness and Psychotic Illness*, 100 J. MENTAL SCI. 990 (1954).

became deaf in later life. Experimentally induced deafness in psychiatrically unimpaired adults also produced paranoia.¹¹⁵

E. Other Medical Patients

Disorientation and delusional psychoses have also been reported among immobilized orthopedic patients and in patients postsurgically bed-confined. Nursing researchers have studied this phenomenon and have concluded that frightening hallucinatory experiences “are probably far more widespread than has been suspected.”¹¹⁶

VI. OCCUPATIONAL SITUATIONS

Researchers reported in the *New England Journal of Medicine* on a study of fifty long-distance truck drivers; of these, thirty experienced vivid visual hallucinations and some became disoriented as if in a dream.¹¹⁷

VII. ANIMAL STUDIES

As noted in the body of this article, many prisoners confined in solitary become intolerant of normal levels of environmental (especially social) stimulation. These reports receive experimental confirmation in laboratory research on animals. Such research demonstrates that sensory deprivation produces an intolerance to normal levels of environmental stimulation; animals exposed to sensory deprivation conditions became overly aroused—“hyperexcitable”—when exposed to normal levels of environmental stimulation, often resulting in severe behavioral disturbances.¹¹⁸

115. See Phil G. Zimbardo et al., *Induced Hearing Deficit Generates Experimental Paranoia*, 212 *SCI.* 1529, 1529–31 (1981).

116. Florence S. Downs, *Bed Rest and Sensory Disturbances*, 74 *AM. J. NURSING* 434, 438 (1974).

117. Ross A. McFarland & Ronald C. Moore, *Human Factors in Highway Safety*, 256 *NEW ENG. J. MED.* 792, 797 (1957).

118. See Austin H. Riesen, *Excessive Arousal Effects of Stimulation After Early Sensory Deprivation*, in *SENSORY DEPRIVATION—HARVARD*, *supra* note 47, at 35–36.

One study produced agitation in mice and rats after a few days of isolation, a report which corroborated previous studies with rats.¹¹⁹ Others have also found isolation-induced aggressive behavior in mice (such as biting attacks).¹²⁰ Further, social isolation has been demonstrated to produce profound and lasting psychological effects in primates. Researchers have noted that over four hundred published investigations of the effects of social isolation on primates show such deleterious effects as self-mutilation and disturbances in perception and learning.¹²¹ They found that in adult rhesus monkeys even brief periods of social isolation produce compromised cognitive processing.¹²² Others have produced symptoms of depression in rhesus monkeys by confining them for thirty days.¹²³ They concluded that solitary “confinement produced greater destructive behavioral effects in less time and with fewer individual differences among subjects than did total social isolation, previously [demonstrated to be] the most powerful technique for producing psychopathological behavior among monkey subjects.”¹²⁴ Induced depression through confinement has been reported in both young and mature monkeys.¹²⁵ Finally, isolation-produced fear in dogs has been clearly demonstrated.¹²⁶

119. See T.C. Barnes, *Isolation Stress in Rats and Mice as a Neuropharmacological Test*, 18 FED’N PROC. 365 (1959).

120. Kinzo Matsumoto et al., *Desipramine Enhances Isolation-Induced Aggressive Behavior in Mice*, 39 PHARMACOLOGY BIOCHEMISTRY & BEHAV. 167, 168 (1991).

121. See David A. Washburn & Duane M. Rumbaugh, *Impaired Performance from Brief Social Isolation of Rhesus Monkeys*, 105 J. COMP. PSYCHOL. 145 (1991).

122. *Id.* at 145.

123. William T. McKinney et al., *Depression in Primates*, 127 AM. J. PSYCHIATRY 1313, 1316 (1971).

124. *Id.* at 1317.

125. See Harry F. Harlow & Steven J. Suomi, *Induced Depression in Monkeys*, 12 BEHAV. BIOLOGY 273 (1974).

126. See W.R. Thompson & R. Melzack, *Early Environment*, 194 SCI. AM. 38 (1956).

APPENDIX B:

THE NINETEENTH CENTURY GERMAN EXPERIENCE WITH SOLITARY
CONFINEMENT

Between 1854 and 1909 thirty-seven articles appeared in the German medical literature on the subject of psychotic disturbances among prisoners, summarizing years of work and many hundreds of cases. A major review of this literature was published in 1912.¹²⁷ Solitary confinement was the single most important factor identified in the etiology of these psychotic illnesses.

Indeed, the first report on the subject of prison psychoses was that of Delbruck, chief physician of the prison at Halle, in which the frequency of mental disturbances was at last so great that it attracted the attention of the authorities.¹²⁸ Delbruck's report concluded that prolonged absolute isolation has a very injurious effect on the body and mind and that it seems to predispose inmates to hallucinations and advised the immediate termination of solitary confinement.¹²⁹

In 1863 Gutsch reported on eighty-four cases of psychosis stemming from solitary confinement and described vivid hallucinations and persecutory delusions, apprehensiveness, psychomotor excitation, sudden onset of the syndrome, and rapid recovery upon termination of solitary confinement.¹³⁰ Many of these individuals developed "suicidal and maniacal outbreaks."¹³¹

In 1871, in a report on fifteen cases of acute reactive psychoses, some of which apparently occurred within hours of incarceration in solitary, Reich described hallucinosis and persecutory delusions in addition to severe anxiety leading to motor excitement—"[t]he patient becomes noisy, screams, runs aimlessly about, destroys and ruins everything that comes in his way."¹³² He also described an acute confusional state accompanying these symptoms, sudden

127. See NITSCHKE & WILMANN, *supra* note 36.

128. *Id.* at 1.

129. *Id.* at 2.

130. *Id.* at 8.

131. *Id.*

132. *Id.* at 31.

cessation of symptoms, recovery, and subsequent amnesia for the events of the psychosis.¹³³

In a statistical summary, Knecht reported in 1891 on the diagnostic assessment of 186 inmates at the “insane department” of the prison at Waldheim and concluded that over half of the total inmates in this department were there due to reactive manifestations to solitary confinement.¹³⁴ The majority of these inmates became insane within two years of confinement in solitary.¹³⁵

In 1884 Sommer reported on 111 cases describing an acute, reactive, hallucinatory, anxious, confusional state associated with solitary confinement, emphasizing the “excited outbursts” and “vicious assaults” of these patients.¹³⁶ His patients’ illness began with difficulty in concentration and hyperresponsivity to minor “inexplicable” external stimuli. These “elementary disturbances of the sensorium (i.e., the five senses)” were seen as leading to “elementary hallucinations” which became more numerous, eventually including auditory, visual, and olfactory hallucinations and eventually becoming incorporated with fearful persecutory delusions.¹³⁷

In 1889 Kirn described 129 cases of psychosis among the inmates at the county jail at Freiburg, concluding that in fifty of those cases, “solitary confinement can be definitely considered as the etiological factor, (and these) show a certain characteristic stamp” including persecutory delusions and hallucinations in multiple spheres (auditory, visual olfactory, tactile).¹³⁸ He also noted that these symptoms often precipitated at night:

[T]he patient is suddenly surprised at night by hallucinatory experiences which bring on an anxious excitement. These manifestations become constant from now on, in many cases occurring only at night, in others also in the daytime. Attentive patients not infrequently hear at first a humming and buzzing

133. *Id.* at 32–33.

134. *Id.*

135. *Id.* at 17.

136. *Id.* at 12, 16.

137. *Id.* at 12–16.

138. *Id.* at 21.

in their ears, unpleasant noises and inarticulate sounds which they cannot understand until finally they hear well differentiated sounds and distinct words and sentences. . . .

. . . The visual hallucinations are very vivid.¹³⁹

In 1888 Moeli contributed a description of “*vorbereiden*”—also known as “the symptom of approximate answers.”¹⁴⁰ Ten years later Ganser contributed to the literature the elucidation of a syndrome which included Moeli’s symptom.¹⁴¹ As Arieti points out, Ganser’s Syndrome became well known—indeed, almost a codification of the whole body of literature on the prison psychoses.¹⁴² Ganser provided a comprehensive and well-elucidated synthesis of symptoms, most of which had been previously described elsewhere. The syndrome he described included (in addition to *vorbereiden*) vivid visual and auditory hallucinations, a distinct clouding of consciousness, sudden cessation of symptoms “as from a dream,” and “a more or less complete amnesia for the events during the period of clouded consciousness.”¹⁴³ Ganser’s most original description was of “hysterical stigmata” within the syndrome, including conversion symptoms, especially total analgesia.¹⁴⁴

Some of the German authors failed to note whether the inmates they were describing were housed in solitary confinement and, unfortunately, Ganser was one of these, stating only that his were prisoners awaiting trial. However, Langard, in 1901, also reporting on observations of accused prisoners awaiting trial, described an acute violent hallucinatory confusion with persecutory delusions and

139. *Id.* at 23–24.

140. *Vorbereiden* is a rather remarkable symptom of deranged and confused thought processes in which the individual’s response to a question suggests that he grasped the gist of the question, and his answer is clearly relevant to the question, and related to the obvious correct answer, yet it still oddly manages to be incorrect. An example would be: Q: “How many colors are there in the flag of the United States” A: “Four”. Q: “What are they?” A: “Yellow”.

141. Ganser, *Ueber Einen Eigenartigen Hysterischen Dämmerzustand*, 30 *ARCHIV FÜR PSYCHIATRIE UND NERVENKRANKHEITEN* [ARCH PSYCH. & NERVENK.] 633 (1898) (F.R.G.).

142. *AMERICAN HANDBOOK OF PSYCHIATRY* 710–12 (Gerald Caplan ed., 2d ed. 1974).

143. *Id.*

144. *Id.*

specifically stated that this syndrome occurred exclusively among those who awaited trial in solitary confinement.¹⁴⁵

Also in 1901 Raecke similarly reported on prisoners awaiting trial and described the full syndrome described by Ganser, including *vorbereiden*; he specifically condemned solitary confinement as responsible for the syndrome.¹⁴⁶ He described his cases as beginning with apathy, progressing to “inability to concentrate, a feeling of incapacity to think,” and even catatonic features, including negativism, stupor, and mutism.¹⁴⁷

In another report, written the same year, Skliar reported on sixty case histories of which he identified twenty-one as acute prison psychoses caused by solitary confinement.¹⁴⁸ While *vorbereiden* was not noted, most of the other symptoms described by Ganser and Raecke were, including massive anxiety and fearful auditory and visual hallucinations; in severe cases, hallucinations of smell, taste, and “general sensation” as well as persecutory delusions, senseless agitation and violence, confusion, and disorientation.¹⁴⁹ The psychosis developed rapidly, at times within hours of incarceration in solitary confinement.¹⁵⁰ Catatonic symptomatology was also noted.¹⁵¹

The German literature reported only on prisoners who suffered gross psychotic symptomatology, some of whom were observed in hospitals or “insane departments” of prisons; thus, these reports generally described only syndromal expressions that rose to the level of overt psychosis. The German reports do, however, powerfully demonstrate the existence of a particular, clinically distinguishable psychiatric syndrome associated with solitary confinement. These multiple reports described a syndrome which included:

1. Massive free-floating anxiety.
2. “Disturbances of the Sensorium,” including—

145. NITSCHKE & WILMANN, *supra* note 36, at 32.

146. *Id.* at 34.

147. *Id.* at 33–35.

148. *Id.* at 40.

149. *Id.* at 41.

150. *Id.*

151. *Id.*

- a. hyperresponsivity to external stimuli; and
 - b. vivid hallucinations in multiple spheres (including auditory, visual, olfactory, gustatory, and tactile modalities); in some reports, these began as simple “elementary” hallucinations and progressed to complex, formed hallucinations.
3. Persecutory delusions, often incorporating coexistent complex hallucinations.
 4. Acute confusional states. In some reports these were seen as beginning with simple inattention and difficulty in concentration. In others, the onset was described as sudden. The confusional state and disorientation was in several reports described as resembling a dissociative, dreamlike state, at times involving features of a catatonic stupor, including negativism and mutism; and, upon recovery, leaving a residual amnesia for the events of the confusional state. Ganser and others observed hysterical conversion symptoms during this confusional state.
 5. Vorbereiden: This was an infrequent finding, mostly described in conjunction with a confusional, hallucinatory state.
 6. Motor excitement, often associated with sudden, violent destructive outbursts.
 7. Characteristic course of the illness:
 - a. onset was described by some authors as sudden, by others as heralded by a progression beginning with sensory disturbances and/or inattention and difficulty in concentration; and
 - b. in many cases, rapid subsidence of acute symptoms upon termination of solitary confinement.

The German reports were generally based upon prisoners who had been hospitalized because of their psychotic illness. In contrast, the population reported upon in the Walpole study was not preselected by overt psychiatric status. Despite this, all of the major symptoms

reported by the German clinicians were observed in the Walpole population, except for *vorbereiden* and hysterical conversion symptoms. In addition, less severe forms of the isolation syndrome were observed in the Walpole population, including:

- Perceptual distortions and loss of perceptual constancy, in some cases without hallucinations.
- Ideas of reference and paranoid ideation short of overt delusions.
- Emergence of primitive aggressive fantasies which remained ego-dystonic and with reality-testing preserved.
- Disturbances of memory and attention short of overt disorientation and confusional state.
- Derealization experiences without massive dissociative regression.

Since Ganser's report has become the twentieth century's clearest memory of a much vaster body of literature, it is also of interest to review the literature describing observations of Ganser's Syndrome in non-prison populations. Several of these reports have been studies of patients in psychiatric hospitals suffering from this syndrome. Since these patients were hospitalized, it was possible to obtain more extensive evaluation and testing of their status. Several reports described a majority of the patients studied as suffering long standing hysterical conversion symptoms; impulsivity, childhood truancy, and antisocial behavior were also commonly described.¹⁵² These findings suggest also that antisocial behavior patterns and psychopathic personality disorder may bear a close relationship to primitive hysterical personality disorder, a relationship which has been described by other authors as well.¹⁵³

152. See, e.g., Ingraham & Moriarty, *supra* note 59; May et al., *supra* note 59; Milo Tyndel, *Some Aspects of the Ganser State*, 102 J. MENTAL SCI. 324 (1956); Herbert Weiner & Alex Braiman, *The Ganser Syndrome*, 111 AM. J. PSYCHIATRY 767 (1955).

153. See ROBERT A. WOODRUFF, JR. ET AL., *PSYCHIATRIC DIAGNOSIS* (1974).

APPENDIX C:

EXPERIMENTAL RESEARCH ON THE PSYCHIATRIC EFFECT OF
PROFOUND SENSORY DEPRIVATION: FACTORS INFLUENCING
VULNERABILITY TO PSYCHIATRIC HARM

As noted in the body of this article, laboratory research has demonstrated that experimentally induced sensory deprivation has major psychological effects and can precipitate severe psychiatric illness. Much of the research in this area attempted to delineate factors in addition to the duration and intensity of sensory restriction which might account for these differing outcomes. The factors which have been elucidated include two which are especially relevant to this discussion and may help to explain the particular malignancy of sensory deprivation in solitary confinement: expectation and individual response.

I. THE INFLUENCE OF EXPECTATION

Research has suggested that a subject's reaction to participation in a sensory deprivation experiment could be profoundly manipulated by external cues imposed by the experimenter:

[These] dramatic effects could be a function of the demand characteristics of the experimental situation. . . .

There is evidence . . . that preparing a subject for probable hallucinations significantly affects the frequency of hallucinations. . . . [S]uch devices as "panic buttons" in experiments are in a sense eloquent "instructions." The use of such a device increases the subject's expectation that something intolerable may occur, and, with it, the likelihood of a bad experience.¹⁵⁴

154. Martin T. Orne & Karl E. Scheibe, *The Contribution of Nondeprivation Factors in the Production of Sensory Deprivation Effects: The Psychology of the "Panic Button,"* 68 J. ABNORMAL & SOC. PSYCHOL. 3, 4 (1964) (citations omitted).

In the experiment, the researchers exposed two groups of subjects to identical conditions of sensory deprivation. The experimental group's introduction to the experiment included the presence of a medical "Emergency Tray," and instructions about a "Panic Button." As predicted, the experimental group became significantly more symptomatic in measures of cognitive impairment and restlessness, and also more symptomatic in every other measure—including perceptual aberrations, anxiety, and spatial disorientation.¹⁵⁵

In a related manner, prisoners in solitary confinement generally view such confinement as threatening and punitive, and often as a deliberate attempt to make them "crack up" or "break my spirit." In light of this, it is not surprising that the only recent report suggesting no major ill effect of solitary confinement utilized prisoners who volunteered to spend four days in solitary confinement.¹⁵⁶

II. INDIVIDUAL DIFFERENCES IN RESPONSE

Several authors have directed attention to the fact that within a given experimental format, massive differences in response can be observed among individual subjects. Often subjects who tolerated the experimental situation well reported pleasant, or at least non-threatening, visual imagery, fantasy, and hallucinatory experiences. The individual's mind may begin to wander, engage in daydreams, slip off into hypnogogic reveries with their attendant vivid pictorial images. The individual may be quietly having sexual and other pleasurable thoughts.¹⁵⁷

On the other hand,

Another subject in the same situation may deal with it in quite another manner. He may soon complain of all manner of things: the bed is causing him a backache, his mind is a blank [He also complains of] intense boredom, tenseness,

155. *Id.* at 3–12.

156. See Richard H. Walters et al., *Effects of Solitary Confinement on Prisoners*, 119 AM. J. PSYCHIATRY 771 (1963).

157. Wright et al., *supra* note 81, at 36.

depressive feelings or of having unpleasant thoughts or picture-like images that disturb him.¹⁵⁸

In response to these concerns about the incidence of psychopathological reactions to sensory deprivation, an important thrust of the experimentation in this area has been, by prescreening, to select as subjects only those persons demonstrating, by some measure, psychological strength and capacity to tolerate regression. The theoretical premise of such work has been:

[I]n the sensory deprivation experiments, it is the ego's autonomy from the drives that is predominately involved Differences in drive-discharge thresholds, phantasy [sic] and daydream capacity, capacity for what [is] . . . termed "regression in the service of the ego" are other theoretically relevant structural dimensions accounting for differences in isolation behavior.¹⁵⁹

These ideas have been subjected to experimental verification, which has corroborated that some individuals tolerate such isolation better than others. For example, two researchers, using the Rohrshach Test for prescreening, concluded that the Rohrshach manifestations of an individual's defense and control mechanisms appear to be a reliable measure for predicting whether an individual will be effective in controlling the drive-dominated responses that might emerge during the individual's period of reduced sensory stimulation.¹⁶⁰

Anecdotal reports in a similar vein appear from time to time in the literature. A subject of one study became panicky during sensory deprivation and stated he had been diagnosed "borderline psychotic."¹⁶¹ Curtis and Zuckerman report on a psychotic paranoid reaction in one subject who suffered delusions for several days afterward, and severe anxiety and depression lasting several weeks;

158. Leo Goldberger, *Experimental Isolation: An Overview*, 122 AM. J. PSYCHIATRY 774, 777 (1966).

159. *Id.* at 778 (footnotes omitted).

160. Wright et al., *supra* note 81, at 37.

161. Sanford J. Freedman & Milton Greenblatt, *Studies in Human Isolation II: Hallucinations and Other Cognitive Findings*, 11 U.S. ARMED FORCES MED. J. 1479, 1486 (1960).

personality test prescreening had suggested poor adjustment, hostility, lack of insight, and insecurity in interpersonal relationships.¹⁶²

Others prescreened forty-three subjects and identified seven as suffering “personality deviations.” Two of these subjects, who were diagnosed as borderline, developed frightening, aggressive fantasies, paranoia, and difficulty in reality testing; one of them prematurely terminated the experiment. Two others were diagnosed as psychopathic; both forced the premature termination of the experiment by disruptive behavior.¹⁶³

Others, using interview techniques and formal psychological test data, studied the effects of two to six days of sensory deprivation on hospitalized psychiatric patients. Among the previously non-psychotic patients they studied, two developed overt paranoid psychoses during the experiment, ultimately necessitating electroshock treatment. These particular individuals appeared to have been unable to tolerate the emergence of aggressive fantasies and images during the sensory deprivation experience.¹⁶⁴

A. Effects of Sensory Deprivation on Antisocial Personality Disorder

1. Aversive Conditioning

Individuals with psychopathic personality disorder are probably among the least tolerant of sensory deprivation. One researcher has described the essential core of psychopathic pathology as a pathological inability to tolerate restricted environmental stimulation:

The psychopath is almost universally characterized as [pathologically stimulus seeking and] highly impulsive He is unable to tolerate routine and boredom. . . . [H]is outbursts frequently appear to be motivated by little more than a need for thrills and excitement. . . .

162. George C. Curtis & Marvin Zuckerman, *A Psychopathological Reaction Precipitated by Sensory Deprivation*, 125 AM. J. PSYCHIATRY 255, 256 (1968).

163. See Henry U. Grunebaum et al., *Sensory Deprivation and Personality*, 116 AM. J. PSYCHIATRY 878 (1960).

164. See H. Azima & Fern J. Cramer, *Effects of Partial Perceptual Isolation in Mentally Disturbed Individuals*, 17 DISEASES NERVOUS SYS. 117 (1956).

It is the impulsivity and lack of even minimal tolerance for sameness which appear to be the primary and distinctive features of the disorder.¹⁶⁵

He goes on to argue that psychopathic individuals may chronically exist in a state of relative stimulus deprivation: “[H]ighly impulsive, psychopathic behavior [may be seen] in terms of stimulation-seeking pathology. If decreased reactivity and/or rapid adaptation [to environmental stimuli] do produce in these persons an affective state of unpleasantness close to that produced by severe sensory deprivation or monotony in the normal individual”¹⁶⁶

He argues that behavioral impulsivity in such individuals may be an effort at coping with this condition of relative sensory deprivation which they experience: “It may be possible . . . to view much of the impulsivity of the psychopath, his need to create excitement and adventure, his thrill-seeking behavior, and his inability to tolerate routine and boredom as a manifestation of an inordinate need for increases or changes in the pattern of stimulation.”¹⁶⁷

A later study, directly comparing psychopathic inmates with non-psychopathic controls, corroborated these findings. The psychopathic inmates scored significantly higher on measures of boredom susceptibility and of impulsivity. The authors concluded that psychopaths are pathologically stimulation seeking and incapable of tolerating isolation conditions.¹⁶⁸

Others, in a large scale study of criminal offenders suffering from mental illness, noted that the prevalence of severe mental illness is higher among incarcerated offenders than among the general population; and that, compared with non-mentally ill inmates, the mentally ill inmates were more likely to be housed in solitary. Moreover many of these mentally ill inmates suffered from a combination of psychiatric disorders predisposing them to both psychotic breakdown and to extreme impulsivity (often including

165. Quay, *supra* note 58, at 80.

166. *Id.* at 182.

167. *Id.* at 181.

168. See Timothy D. Emmons & Warren W. Webb, *Subjective Correlates to Emotional Responsivity and Stimulation Seeking in Psychopaths, Normals, and Acting-Out Neurotics*, 42 J. CONSULTING & CLINICAL PSYCHOL. 620 (1974).

substance abuse). Such individuals tended to be highly impulsive, lacking in internal controls, and tended to engage in self-abusive and self-destructive behavior in the prison setting, and especially so when housed in solitary.¹⁶⁹

Many of the inmates placed in solitary confinement are thus likely to be among the least capable of tolerating the experience, and among the most likely to suffer behavioral deterioration as a consequence of such confinement. Solitary confinement has at times been rationalized as being a form of “aversive conditioning,” intended to extinguish negative inmate behaviors. Yet this assertion ignores many of the most basic tenets of any behavior modification treatment, and would in any case clearly violate the ethical guidelines governing the use of aversive conditioning:

a. Ethical Considerations

First of all, since aversive conditioning—the use of punishment as a means of inducing behavior change—is inherently suspect ethically and creates an inherent risk of harm, very clear outcome variables have to be articulated and systematically measured over time. As a result of these serial measurements, there must be clear evidence that the undesirable behavior is in fact lessening in frequency and intensity. Such measurement will also identify those patients for whom such aversive conditioning is actually harmful, allowing these individuals to be removed from the aversive treatment protocol. Were such measurements done in the prison setting, staff would inevitably be required to acknowledge the behavioral deterioration which many inmates were suffering as a result of placement in solitary, and in such cases, ethical considerations would have required transferring the inmate out of such confinement.

b. SHU Incarceration is not Aversive Conditioning

SHU incarceration does not meet criteria for aversive conditioning. Indeed, any behavior modification scheme must define and describe very explicitly two variables:

169. Curtis & Zuckerman, *supra* note 162, at 271–72.

(i) The behavior being changed:

Behavior researchers have learned that in order for a subject to benefit from aversive (or any other form of) conditioning, the behavior at issue must be a single, very clearly defined behavior. When multiple behaviors are responded to by the same reinforcer or punishment, learning and behavior change does not occur. Thus, placement in SHU, which is “punishment” for a host of different behaviors, is simply not being used in a manner consistent with an intent of behavior modification; there is inadequate linkage of any specific behavior to this “punishment.”

(ii) The “punishment”:

Moreover, SHU confinement is quite clearly not “punishment.” To be effective, a “punishment” must be very closely linked in time to the targeted behavior, and for learning to occur, there must be repeated opportunities to experience this close link between the target behavior and the punishment. Thus, the “punishment” must be brief and immediate. For example, a mild but painful electric shock or a sudden very loud noise would be ideal punishments in aversive conditioning.

Occasionally “time outs,” the brief use of a seclusion room to quickly control disruptive behavior, are used as part of an aversive conditioning program. But when this technique is employed, it is used very quickly and for a very brief period of time—in order for the “time out” to work as a behavior modifier, there must be very clear alternative behaviors which, when manifested, will immediately end the “time out.”

For any behavior modification scheme to work then, there must always be an exquisitely close relationship between behavior and response. Indeterminate or prolonged sentencing to solitary simply has nothing to do with aversive conditioning.

APPENDIX D:

REPORTS OF THE LONG-TERM EFFECTS OF SOLITARY CONFINEMENT
IN FORMER POLITICAL PRISONERS AND IN PRISONERS OF WAR:
SOLITARY CONFINEMENT AS A MEANS OF “BRAIN WASHING” AND
“INDOCTRINATING”

Although concerns about the psychiatric effects of solitary confinement among prisoners of war were raised in the medical literature at least as early as post-World War II, this issue reached massive public exposure only after the fearful news of “brain washing” among American prisoners of war in Korea. As is well known, the 1950’s were an era of tremendous fear of Communism and of the attempts by communist states to “indoctrinate” people into their ideology. As noted in the body of this article, in the 1950s the United States Department of Defense and the Central Intelligence Agency sponsored a great deal of research on these issues. The results of extensive research done for the Department of Defense were subsequently published.¹⁷⁰ The paper documented interrogation techniques of the Soviet KGB in regard to the incarceration of political prisoners, and the Chinese communists’ imprisonment of American prisoners of war in Korea.

The report indicated that the KGB operated detention prisons, many of which were “modern . . . well built and spotlessly clean . . . [with] attached medical facilities and rooms for the care of sick detainees. An exercise yard is a standard facility.”¹⁷¹ Incarceration in these prisons is almost universally in solitary confinement, in a cell approximately ten feet by six feet in size.¹⁷² “An almost invariable feature of the management of any important suspect under detention is a period of total isolation in a detention cell.”¹⁷³

This isolation was seen as a central feature of the imprisonment: “The effects upon prisoners of the regimen in the isolation cell are

170. HINKLE & WOLFF, *supra* note 65.

171. *Id.* at 125.

172. *Id.*

173. *Id.* at 126.

striking. . . . A major aspect of this prison experience is isolation. . . . [In the cells] [h]is internal as well as external life is disrupted” and “he develops a predictable group of symptoms, which might almost be called ‘disease syndrome.’”¹⁷⁴

This syndrome develops over time:

He becomes increasingly anxious and restless, and his sleep is disturbed. . . .

The period of anxiety, hyperactivity, and apparent adjustment to the isolation routine usually continues from one to three weeks. As it continues, the prisoner becomes increasingly dejected and dependent. He gradually gives up all spontaneous activity within his cell and ceases to care about personal appearance and actions. Finally, he sits and stares with a vacant expression, perhaps endlessly twisting a button on his coat. He allows himself to become dirty and disheveled. . . . He goes through the motions of his prison routine automatically, as if he were in a daze. . . . Ultimately he seems to lose many of the restraints of ordinary behavior. He may soil himself. He weeps; he mutters It usually takes from four to six weeks to produce this phenomenon in a newly imprisoned man.¹⁷⁵

Addressing the emotional impact on prisoners of such confinement, the report noted that:

His sleep is disturbed by nightmares. Ultimately he may reach a state of depression in which he ceases to care about his personal appearance and behavior and pays little attention to his surroundings. In this state the prisoner may have illusory experiences. A distant sound in the corridor sounds like someone calling his name. The rattle of a footstep may be interpreted as a key in the lock opening the cell.

174. *Id.* at 127.

175. *Id.* at 128.

Some prisoners may become delirious and have visual hallucinations.¹⁷⁶

However, the report also notes that each individual may respond differently: Not all men who first experience total isolation react in precisely this manner. In some, these symptoms are less conspicuous. In others, dejection and utter despondence set in earlier, or later. Still others, and especially those with pre-existing personality disturbances, may become frankly psychotic.¹⁷⁷

The authors of this report note that the procedures in the Chinese detention camps are somewhat more complex. Prisoners there underwent an initial period of isolation similar to that found in the Soviet prisons.¹⁷⁸ In the second phase, however they were housed in extremely tight quarters within “group cells” comprising approximately eight prisoners.¹⁷⁹ Under the tensions and hostilities created in this environment, brutality of prisoners by other prisoners was almost inevitable and was, according to the authors, apparently an intended result of this “group cell” confinement.¹⁸⁰

There are many long-term studies of American prisoners of war; unfortunately, the factor of solitary confinement has not generally been separated out in these studies. However, one relatively recent study of Korean prisoners of war described long-term effects including interpersonal withdrawal and suspiciousness, confusion, chronic depression, and apathy toward environmental stimuli. Irritability, restlessness, cognitive impairment, and psychosomatic ailments were extremely common in the group, most of whom had suffered periods of incarceration in solitary confinement at the hands of the Chinese. This report also included a case report of one individual exposed to harsh conditions of solitary confinement for more than sixteen months; thirty years after release, he continued suffering sleep disturbances, nightmares, fearfulness, interpersonal suspicion and withdrawal, severe anxiety, and severe depression. These former prisoners also had psychosomatic ailments including

176. *Id.*

177. *Id.* at 129.

178. *Id.* at 153.

179. *Id.* at 156.

180. *Id.* at 159.

gastrointestinal disturbances, chronic headaches, and obsessive ruminations. They tended to become confused and thus cognitively impaired and were emotionally volatile and explosive.¹⁸¹

In former prisoners of war in the Korean conflict, approximately forty years after their release from confinement, solitary confinement was cited as one of the severe stressors in this group. These former prisoners demonstrated persistent anxiety, psychosomatic ailments, suspiciousness, confusion, and depression. They tended to be estranged and detached from social interaction, suffered from obsessional ruminations, and tended to become confused and cognitively impaired, suffering memory and concentration difficulties which affected their cognitive performance on formal testing.¹⁸²

181. See Patricia B. Sutker et al., *Cognitive Deficits and Psychopathology Among Former Prisoners of War and Combat Veterans of the Korean Conflict*, 148 AM. J. PSYCHIATRY 67 (1991).

182. *Id.* at 68.

EXHIBIT 28

INMATE PROPERTY**(REV. 4/1/14)****MATRIX - AUTHORIZED PERSONAL PROPERTY SCHEDULE (APPS)**

Facilities may submit requests to be exempted from the personal property items detailed in these schedules. These Exemption Requests are to be submitted to the Chief, Standardized Procedures Unit (SPU). All requests must include rationale and supporting data such as incident reports, physical plant limitations, etc. In the case of an emergency, where the safety of inmates, staff, or other persons are at immediate risk, the requesting institution may immediately act to restrict an item but must notify the SPU. An exemption request shall be submitted to the SPU within five working days of any locally imposed restriction. Exemption requests will be processed through the appropriate Associate Director and then submitted to the Deputy Director, Division of Adult Institutions (DAI) for approval/disapproval.

Inmates may request to have items added to the APPS through their Inmate Advisory Council (IAC). All IAC requests are subject to review by local institutional administration and may be included with any facility Exemption Requests forwarded to the DAI.

TABLE OF CONTENTS

ITEM DESCRIPTION	PAGE #
Granted Exemption Requests.....	2
Reception Centers Male Inmates	6
General Population Levels I, II, and III Male Inmates	15
Level IV Male Inmates.....	25
Administrative Segregation Unit/Security Housing Unit/ Psychiatric Services Unit Male Inmates	35
Female Inmates.....	45

AVENAL STATE PRISON**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIPATRIA STATE PRISON**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.
- Tennis shoes are not permitted.

CALIFORNIA CORRECTIONAL CENTER

- Personal televisions are not permitted at Level I and II Dormitories.

CALIFORNIA CORRECTIONAL INSTITUTION**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

CENTRAL CALIFORNIA WOMEN'S FACILITY

No Exemptions

CENTINELA STATE PRISON**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIFORNIA INSTITUTION FOR MEN**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIFORNIA INSTITUTION FOR WOMEN (CIW)

No Exemptions

CALIFORNIA MEN'S COLONY**West Facility**

- Fans, lamps, portable typewriters, battery rechargers, alternating current (AC)/direct current (DC) adapters, television sets, rechargeable batteries, and powdered creamer are excluded.
- Extension cords and hot pots are permitted by Warden's discretion.

East Facility

- 9 foot extension cord, as permitted at Warden's discretion.

Administrative Segregation Units

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIFORNIA MEDICAL FACILITY**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

CORCORAN STATE PRISON**Dormitory Facilities**

- Fans, televisions, and musical instruments are not permitted in any dormitory housing.

Administrative Segregation Units

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIFORNIA REHABILITATION CENTER

No Exemptions

GRANTED EXEMPTION REQUESTS (continued)**CORRECTIONAL TRAINING FACILITY****All Facilities**

- Tweezers, non-metal, plastic only, permitted for PG A, B, C, and U.

CHUCKAWALLA VALLEY STATE PRISON**Dormitory Facilities**

- Dormitory housing is excluded from battery rechargers, hot pots, electric AC power lamps, televisions, electric razors, electric typewriters, AC power adapters.

Administrative Segregation Units

- Entertainment appliances are not permitted in units with physical plant limitations.

DEUEL VOCATIONAL INSTITUTION**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

FOLSOM STATE PRISON**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

HIGH DESERT STATE PRISON**Level IV Facilities**

- Disposable razors and manual typewriters are not permitted.
- Clip on fans and lights are not permitted.
- Styrofoam is not permitted.

Administrative Segregation

- Entertainment appliances are not permitted in units with physical plant limitations.

IRONWOOD STATE PRISON

No Exemptions

KERN VALLEY STATE PRISON**Administrative Segregation Unit**

- Entertainment appliances are not permitted in units with physical plant limitations.
- AC appliances are not permitted in ASU B1 intake cells (Correctional Clinical Case Management Services).

CALIFORNIA STATE PRISON, LOS ANGELES COUNTY**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

MULE CREEK STATE PRISON

No Exemptions

NORTH KERN STATE PRISON**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

PELICAN BAY STATE PRISON**All Facilities**

- Personal toothbrushes are excluded from all facilities, State-issue only.

Level I and Gym Facilities

- AC appliances are not permitted.

Level II Facilities

- Hand held mirrors, nail clippers, disposable razors, ballpoint pens, pencil sharpeners, and fans are not permitted.

Level IV Facilities

- Hand held mirrors, nail clippers, ballpoint pens, and fans are not permitted.

Administrative Segregation Unit

- Entertainment appliances are not permitted in units with physical plant limitations.

GRANTED EXEMPTION REQUESTS (continued)**PLEASANT VALLEY STATE PRISON****Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

RICHARD J. DONOVAN CORRECTIONAL FACILITY**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIFORNIA STATE PRISON, SACRAMENTO**All Dormitory Facilities**

- Fans, televisions, and musical instruments are not permitted in dormitory housing.

Gymnasium Dormitory Facilities

- AC appliances are not permitted in gymnasium dormitories.

CALIFORNIA SUBSTANCE ABUSE TREATMENT FACILITY AND STATE PRISON, CORCORAN**All Facilities**

- Pencil sharpeners are not permitted.

Dormitory Facilities

- Televisions operating on AC power are not permitted in dormitories.
- Battery operated televisions are still permitted in dormitories, but must be able to fit into locker.

Level IV Facilities

- Mirrors are not permitted.

SIERRA CONSERVATION CENTER**Level I and II Facilities**

- Televisions are not permitted.
- Hot Pots are not permitted in Conservation Camps, Level I and Level II facilities.

Administrative Segregation Units

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIFORNIA STATE PRISON, SOLANO**Level II Dormitories**

- New televisions are not permitted. Previously existing televisions shall be permitted to remain until removed through attrition.

Administrative Segregation Units

- Entertainment appliances are not permitted in units with physical plant limitations.

SAN QUENTIN STATE PRISON**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

SALINAS VALLEY STATE PRISON**Administrative Segregation Unit**

- Entertainment appliances are not permitted in units with physical plant limitations.

VALLEY STATE PRISON FOR WOMEN

No Exemptions

WASCO STATE PRISON**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

CALIFORNIA OUT-OF-STATE-FACILITIES (COCF)**Administrative Segregation Units**

- Entertainment appliances are not permitted in units with physical plant limitations.

COMMUNITY CORRECTIONAL FACILITIES (CCF) – MALE INMATES

- AC appliances are not permitted.
- Battery operated televisions are not permitted.

CONSERVATION CAMPS (FEMALE)

- Immersion heaters and televisions are not permitted.

COMMUNITY CORRECTIONAL FACILITIES (FEMALE)

- All AC appliances, hot pots and warmers, and extension cords, are not permitted.

COMMUNITY PRISONS MOTHER PROGRAM (CPMP), FEMALE REHABILITATIVE COMMUNITY CORRECTIONAL CENTERS (FRCCC):

- Immersion heaters, televisions, antenna, splitters and coaxial cables are not permitted.
- No food or drinks, bowls, tumblers, can openers, storage containers, or umbrellas.
- No clothing pins, combination locks, foot lockers.
- Colored pattern clothing is permitted.

EXHIBIT 29

UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

DIEGO ABREGO AREVALO, et al.,

Petitioners-Plaintiffs,

v.

THOMAS DECKER, in his official capacity as
Field Office Director, New York City Field
Office, U.S. Immigration & Customs
Enforcement, et al.,

Respondents-Defendants.

Civil Action No. _____

DECLARATION OF ROBERT M. SAPOLSKY

I, Robert M. Sapolsky, declare under penalty of perjury, pursuant to 28 U.S.C. § 1746, that the following is true and correct to the best of my knowledge:

RELEVANT BACKGROUND AND QUALIFICATIONS

1. I am the John A. and Cynthia Fry Gunn Professor at Stanford University. I hold joint appointments in the departments of Biology, Neurology & Neurological Sciences, and Neurosurgery. I graduated summa cum laude, Phi Beta Kappa, from Harvard University in 1978, with a degree in biological anthropology. I received my Ph.D. in neuroendocrinology at Rockefeller University. I have been a professor at Stanford since 1987, and holder of an endowed chair since 2002. I have received a number of honors and awards for my work, including the MacArthur Fellowship Genius Grant, an Alfred P. Sloan Fellowship, the Klingenstein Fellowship in Neuroscience, and the John P. McGovern Award for Behavioral Science, awarded by the American Association for the Advancement of Science. I also received the National Science Foundation Presidential Young Investigator Award, as well as awards for Young Investigator of the Year from the International Society for Psychoneuroendocrinology, the Society for Neuroscience, and the Biological Psychiatry Society.
2. As a neuroendocrinologist, I have decades of experience working in the area of stress and the body. My work has included studies of humans, non-human primates, rats and mice, and while focusing mostly on the brain, has included work on cancer progression and viral infections. My lab was one of the pioneers in the early 1990s of developing gene therapy in the injured nervous system, and this involved extensive work with, and molecular manipulation of, viral vectors derived from herpes simplex virus, adenovirus, and adeno-associated virus.

3. I have published approximately 470 peer-reviewed publications in science and/or medical journals, and have single-authored six books. My work has been cited more than 140,000 times which, by a recent meta-analysis, places me in the top 0.1% of measures of scientific impact.
4. My C.V. includes a full list of my honors, experience, and publications, and it is attached as Exhibit A.
5. I am not being compensated for my time reviewing materials and preparing this report.
6. During the past four years, I have testified six times as an expert witness in criminal trials involving murder or attempted murder, in evaluating the neuropsychiatric status of the defendant. None of these involved anything related to viral infections. The cases are: *People v. Vladimir Sotelo* (Cal. 2018); *People v. Anicacio Garcia* (Cal. 2019); *People v. Pedro Lopez* (Cal. 2019) (once at trial and again at retrial); *People v. Arent Bradt* (Cal. 2019); and *State v. Darryl Oliver* (Ga. 2020).

STRESS AND HEALTH: A BRIEF OVERVIEW

7. The word “stress” entered the medical and psychological literature (borrowed from engineering) approximately a century ago, and tens of thousands of papers have examined the subject since then. Broadly, a “stressor” can be defined as an external event that throws an organism out of homeostatic balance, and the “stress-response” is the array of physiological adaptations meant to re-establish homeostasis.
8. In its original medical sense, a stressor was conceptualized as a physical challenge to homeostasis, such as a prey species sprinting from a predator, or a hungry predator sprinting after its prey. The field then expanded in a critical way to include the concept of psychological stressors, which involves the activation of the stress-response by the *anticipation* (accurate or otherwise) of a physical challenge to homeostasis. Classic physical stressors are typically short-term in nature, whereas psychological stressors are more likely to be chronic; crucially, when stress is chronic, there are increased risks and severity of disease.
9. Humans are obviously the most psychologically and socially sophisticated species on earth; disease in humans can arise from chronic stress spanning days to decades, and such stress is typically psychosocial in nature. A partial list of diseases that can be caused by or worsened by stress include Type II diabetes, obesity, metabolic syndrome, hypertension, cardiovascular disease, inflammatory bowel disorders, impaired fertility, accelerated brain aging and cellular senescence, and an array of cognitive and psychiatric disorders.¹
10. Of greatest relevance to this matter, the longest-recognized pathological consequence of chronic stress (since circa 1930) is suppression of the immune system, worsening of the outcome of immune diseases, and triggering of inflammation. Of critical relevance to

¹ See, e.g., Robert Sapolsky et al., *How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions*, 21 *Endocrine Reviews* 55-89 (2000); Robert Sapolsky, *Why Zebras Don't Get Ulcers: A Guide to Stress, Stress-Related Disease and Coping* (3d. ed. 2004).

COVID-19, this involves impaired immune defenses against viral infection, and compromised function of the lungs.

STRESS IMPAIRS IMMUNE DEFENSES AGAINST VIRAL INFECTIONS

11. An extensive literature demonstrates that stress compromises the ability of the immune system to defend the body against viral infections; such work has included humans, rodents and livestock as study subjects. While there has not been sufficient time to test this with respect to the SARS-CoV-2 virus, this stress effect has been shown for a wide array of classes of viruses. Thus, stress worsens the consequences of infection with herpes simplex virus (HSV),² Epstein-Barr virus (EBV),³ West Nile Virus,⁴ influenza virus,⁵ human papilloma virus,⁶ viral meningitis and viral endocarditis.⁷
12. The links between stress and impaired viral defense have depended on studies of an array of human stressors. These include the stress of various psychiatric and psychological disorders, experimental psychological stressors, low socioeconomic status, being the primary caregiver for a chronically ill patient, or being an astronaut on a prolonged mission. Collectively, these studies have included more than 150,000 human subjects.
13. Though the specific biological pathways vary from condition to condition, mental illnesses such as anxiety, depression, and bipolar disorder will impair immune function and heighten vulnerability to viral infection.
14. Stress has been shown to disrupt anti-viral defenses in laboratory animals as well. Such stressors include social instability, social defeat, social isolation, or exposure to uncontrollable (versus controllable) shocks. The viruses tested have included those listed in paragraph 11 and, in addition, pseudorabies virus,⁸ murine encephalomyelitis virus,⁹ and bovine HSV.¹⁰
15. In some studies, impaired viral defenses are demonstrated by showing impairment of specific constituents of the immune system. This involves decreased levels in the blood

² See, e.g., K. Ashcraft et al., *Psychological stress impairs the local CD8⁺ T cell response to mucosal HSV-1 infection and allows for increased pathogenicity via a glucocorticoid receptor-mediated mechanism*, 33 *Psychoneuroendocrinology* 951-63 (2008).

³ See, e.g., R. Glaser et al., *Stress and the memory T-cell response to the Epstein-Barr virus in healthy medical students*, 12 *Health Psych.* 435 (1993).

⁴ See, e.g., D. Ben-Nathan & G. Feuerstein, *The influence of cold or isolation stress on resistance of mice to West Nile virus encephalitis*, *Experientia* 46, 285-90 (1990).

⁵ See, e.g., J. Tian et al., *A single E627K mutation in the PB2 protein of H9N2 avian influenza virus increases virulence by inducing higher glucocorticoids (GCs) level*, 7(6) *PLoS ONE* e38233 (2012).

⁶ See, e.g., C. Fang et al., *Perceived stress is associated with impaired T-cell response to HPV16 in women with cervical dysplasia*, 35 *Ann. Behav. MED* 87-96 (2008).

⁷ See, e.g., H. Song et al., *Stress related disorders and subsequent risk of life threatening infections: population based sibling controlled cohort study*, 367 *Brit. Med. J.* 15784 (2019).

⁸ See, e.g., J. de Groot et al., *A single social defeat transiently suppresses the anti-viral immune response in mice*, 95 *J. Neuroimmunol.* 143-51 (1999).

⁹ See, e.g., Young E et al., *Chronic social stress impairs virus specific adaptive immunity during acute Theiler's virus infection*, 254 *J. Neuroimmunol.* 254, 19-27 (2013).

¹⁰ See, e.g., P.D. Hodgson, *Effect of stress on viral bacterial synergy in bovine respiratory disease: novel mechanisms to regulate inflammation*, 6(4) *Comp. Func. Genomics* 244-250 (2005).

and in tissue of key chemical mediators of anti-viral infection, damaging levels of cytokines, impaired generation of antibodies that specifically target the virus in question, and impaired recruitment of immune cells to the tissue that is virally infected.¹¹

16. In some studies, stress-induced impairment of viral defenses has been demonstrated with viral outcomes. These include stress-induced increases in viral replication rates, higher viral titers (the concentration of the virus in the body), impaired clearance of virus from infected tissue, more reactivation of latent viruses, and increased epidemiological evidence of life-threatening viral infections in a population.¹²
17. Finally, in some studies, the outcome has been a pathological one. These include demonstrations of stress worsening virally-induced nerve damage, slower healing of HSV-induced lesions, a variety of pathological effects on the lungs (see below), and lower survival rates. For example, one study demonstrated that physical or psychological stressors in mice cause an approximate 50% increase in mortality rates induced by West Nile Virus.¹³
18. Collectively, these studies show that various types of stressors, in various species, worsen the outcome of viral diseases, as measured by immune outcomes, virological outcomes, disease outcomes and survival rates. The evidence also shows that the degree of immunosuppression worsens the more severe and long-lasting the stress has been.

STRESS AND LUNG DISEASES: RELEVANCE TO SARS-COV-2

19. There are two primary ways in which chronic stress will likely leave an individual at significantly higher risk of contracting COVID-19 and at significantly higher risk of serious illness or death if infected. First, as described above, there is the weakening of the immune system's anti-viral capacities, which will leave the body more vulnerable to viral infections and less able to combat them. Second, there is the specific risk caused by inflammation of the lungs and other organs, which I will describe here.
20. The literature reviewed above considers the adverse effects of stress on tissues and organs throughout the body. The core of SARS-CoV-2's ability to sicken and kill arises from it preferentially targeting the lungs; this, of course, is the reason why symptoms of the disease revolve around respiratory distress, and why shortages of ventilators are so devastating. Thus, it is important to focus on the specific issue of what stress does to lungs and respiration, and the pertinence of this to SARS-CoV-2.
21. Study of the impact of stress on disease has been furthered enormously by the appreciation that chronic stress causes inflammation throughout the body, which exacerbates the course of a wide range of diseases.

¹¹ See, e.g., C. Welsh et al., *Effects of stress on the immune response to Theiler's virus--implications for virus-induced autoimmunity*, 17 *Neuroimmunomodulation* 169-72 (2010).

¹² See, e.g., Song, *supra*; M. Elftman et al., *Stress-induced glucocorticoids at the earliest stages of herpes simplex virus-1 infection suppress subsequent antiviral immunity, implicating impaired dendritic cell function*, 184 *J. Immunol.* 1867-75 (2010).

¹³ See, e.g., Ben-Nathan and Feuerstein, *supra*.

22. Crucially, a variety of stressors cause inflammation in the lungs. Stress-induced lung inflammation has been demonstrated in a number of ways through a variety of outcomes. This includes increased presence in the lungs of inflammatory cells and increased release of their chemical messengers, and increased presence of markers of inflammation in exhaled breath (e.g., increased levels of nitric oxide).
23. Moreover, such stress-induced pulmonary inflammation impairs lung function, including increased bronchoconstriction, increased airway impedance (the former term is a measure of how much lung airways are tightened by smooth muscle; the latter describes the extent of resulting disruption of air flow), decreased lung capacity, tidal volume instability and ribcage abdominal asynchrony (the former is a measure of breathing irregularity; the latter is the likely physiological cause of such irregularities). These pro-inflammatory effects of stress occur in the absence of disease. The result is that the lung tissue will be more vulnerable, people will already have more trouble breathing, and the lungs will have more difficulty clearing fluids. In that sense, the effect of stress on the lungs is similar to that of asthma.
24. Stress also worsens the outcome of pulmonary diseases. The textbook example of this is the ability of stress to cause or worsen asthma. A variety of stressors worsen the pathological features of asthma including worsening of asthma-induced bronchoconstriction, airway impedance, airway inflammation, accumulation of inflammatory lung fluid and decreased lung volume.
25. Finally, stress specifically exacerbates features of viral pulmonary diseases. This includes specifically impairing antiviral immune defenses in lung tissue, and increasing the incidence of respiratory viral infections.
26. Collectively, these studies show that even in the absence of disease, stress causes lung inflammation and impairs lung function. This effect will be particularly pronounced as stress rises from moderate to severe levels. Furthermore, stress worsens the outcome of a number of pulmonary disorders (including pneumonia, as with asthma, and for similar physiological reasons), including respiratory viral diseases. This means that people who have experienced chronic stress who contract a respiratory viral disease are significantly more likely to become seriously ill or even die.
27. Because of the novelty of SARS-CoV-2 and the chaos of an ongoing pandemic, there has been little study to date about the effects of stress on anti-viral defenses against SARS-CoV-2 or against coronaviruses in general. This is not unique, as even with certain preexisting physical conditions, our knowledge of the ways in which they increase vulnerability to SARS-CoV-2 is continually evolving. And nonetheless, as to stress, extrapolations can be made with considerable confidence.
28. First, there are considerable genomic, functional and structural similarities across the entire coronavirus family, allowing one to tentatively generalize to SARS-CoV-2.¹⁴

¹⁴ See, e.g., Y. Wang et al., *Nsp1 proteins of group I and SARS coronaviruses share structural and functional similarities*, 10 *Infection, Genetics and Evolution* 919-924 (2010); H. Jayaram et al., *X-Ray structures of the N- and*

29. Second, there are some key similarities between the coronavirus family and the viruses discussed above. For example, the essential viral proteins that are used to fuse to and invade host cells are heavily conserved between coronaviruses and influenza virus.¹⁵ As another example, Epstein Barr Virus and coronaviruses have their pro-inflammatory effects through heavily overlapping mechanisms.¹⁶ As a final example, bats can be primary zoonotic reservoirs for both papilloma viruses and coronaviruses.¹⁷
30. Third, a study has found that “low levels of CD3⁺CD8⁺ T cells” are predictors of high mortality rates from COVID-19.¹⁸ These cells help mediate an immune cascade that kills virally-infected cells. Chronic stress can decrease circulating levels of these cells.
31. Fourth, the CDC lists as one of the groups at high risk from COVID-19 people who have had “prolonged use of corticosteroids and other immune weakening medications.”¹⁹ Corticosteroids are synthetic versions of the glucocorticoid stress hormones discussed in several of the studies I have cited. These hormones play an important role as a pathway through which chronic stress weakens the immune system.
32. Finally, the CDC includes among its high risk categories for COVID-19 “[p]eople who are immunocompromised” and notes that “[m]any conditions can cause a person to be immunocompromised.”²⁰ It is my professional judgment that high levels of chronic stress would place people in this category.

PLAINTIFFS’ HEIGHTENED RISK FROM COVID-19

33. In reaching my assessment in this section, I have reviewed the declaration of Karla Ostolaza, describing the medical histories and current situations of [REDACTED], [REDACTED], [REDACTED], [REDACTED], and [REDACTED].
34. Based on a number of factors, including their serious mental health conditions and the highly stressful nature of their current situations, it is my professional judgment based on my decades of work on stress that each of these individuals has experienced and will continue to experience exceptionally high levels of stress.

C-terminal domains of a coronavirus nucleocapsid protein: Implications for nucleocapsid formation, 80 J. Virology 6612-6620 (2006).

¹⁵ See P. Chambers et al., *Heptad repeat sequences are located adjacent to hydrophobic regions in several types of virus fusion glycoproteins*, 71 J. General Virology 3075-3080 (1990).

¹⁶ See, e.g., E. Tirotta, *Epstein–Barr virus-induced gene 3 negatively regulates neuroinflammation and T cell activation following coronavirus-induced encephalomyelitis*, 254 J. Neuroimmunology 110-116 (2013).

¹⁷ See, e.g., Tse H. et al., *Identification of a novel bat papillomavirus by metagenomics*, 7(8)PLoS ONE e43986 (2012).

¹⁸ R.-H. Du et al., *Predictors of mortality for patients with COVID-19 pneumonia caused by SARS-CoV-2: A Prospective Cohort Study*, 55(4) European Respiratory Journal (2020).
<https://erj.ersjournals.com/content/early/2020/04/01/13993003.00524-2020>.

¹⁹ Centers for Disease Control and Prevention, *Groups at higher risk for severe illness* (last accessed Apr. 11, 2020).
<https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/groups-at-higher-risk.html>

²⁰ *Id.*

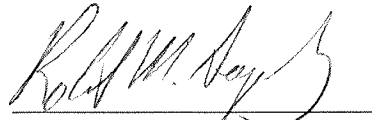
35. Because of the effects of such high levels of stress on the immune system and the body described above, these individuals are therefore likely at considerably heightened risk of contracting COVID-19 and becoming severely ill and even dying if they do.

CONCLUSIONS

36. An extensive peer-reviewed literature demonstrates that stress compromises antiviral immune defenses and increases viral virulence. These findings are derived from studies of humans and other species, with large sample sizes, examining a variety of stressors and viral insults. Moreover, in the absence of disease, stress causes inflammation in the lungs and impairs respiration. Furthermore, stress specifically exacerbates the consequences of the respiratory viral diseases that are most similar to SARS-CoV-2. This is partly because stress can exacerbate the pulmonary inflammation and buildup of fluid that is central to the pathology of pneumonia. Finally, these effects are of sufficient magnitude that they significantly worsen critical disease outcomes, which include mortality rates. In other words, people who have experienced chronic stress are more likely to die of viral respiratory illnesses.
37. As noted above, there has been insufficient time for the study of stress effects on immune defenses against SARS-CoV-2 and the pathogenic course of COVID-19 specifically. However, given the array of viruses and viral diseases examined in the stress literature, including respiratory diseases that are quite close to COVID-19, and the array of pathophysiological outcomes, I can state with considerable confidence that stress will significantly increase the risk of SARS-CoV-2 infection and markedly worsen the consequences of this infection. This includes a heightened risk of severe illness or even death.
38. It is therefore my professional judgment that people who have suffered from chronic stress are at heightened risk from COVID-19 and fall into the CDC high risk category of “[p]eople who are immunocompromised.”²¹ Further, given the gravity of this heightened risk, it would be dangerous for both those who have suffered chronic stress and those around them to not treat them as if they were at particularly elevated risk.
39. For these reasons, it is also my professional judgment that [REDACTED], [REDACTED], [REDACTED], [REDACTED], and [REDACTED] are at significantly heightened risk from COVID-19.

I declare under penalty of perjury that the foregoing is true and correct.

April 13th, 2020
Palo Alto, California


Robert M. Sapolsky

²¹ *Id.*

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Educational History

1984 Ph.D. Neuroendocrinology, The Rockefeller University
1978 A.B. Biological Anthropology, Harvard University, summa cum laude, Phi Beta Kappa

Professional Experience

2002: John A. and Cynthia Fry Gunn Professor, Department of Biological Sciences, Stanford University; Departments of Neurology and Neurological Sciences, and of Neurosurgery, Stanford University School of Medicine.
1995: Professor
1991 - 1995: Associate Professor with tenure
1987 - 1991: Assistant Professor, Department of Biological Sciences, Stanford University
1985: Research Associate, Institute of Primate Research, National Museums of Kenya, Nairobi, Kenya
1985 - 1987: Postdoctoral Fellow, Peptide Biology Laboratory, Salk Institute

Professional Awards and Honors

1985: Lindsley Prize of the Society for Neuroscience (outstanding doctoral thesis in behavioral neuroscience).
1987: MacArthur Fellow of the John D. and Catherine T. MacArthur Foundation
Faculty Scholar, Alzheimers Association
Faculty Scholar, Sloan Foundation
Faculty Scholar, Klingenstein Foundation
Presidential Young Investigator, National Science Foundation
1990: A.B. Bennett Award, Society of Biological Psychiatry, young investigator of the year
C. Richter Award, International Society of Psychoneuroendocrinology, young of the year
Winner of Dean's Award for distinguished teaching
1992: Young Investigator of the Year Award, Society for Neuroscience
Research Career Development Award, NIH
Winner of Associated Students of Stanford University Teaching Award
1993: Bing Award for Teaching Excellence
1994: Finalist, Los Angeles Times Book Award for "Why Zebras Don't Get Ulcers"
1996: Elected as Fellow, American Association of the Advancement of Science
1997: Elected as Fellow, California Academy of Sciences
1998: Finalist, Los Angeles Times Book Award for "The Trouble With Testosterone."
Hoagland Prize for Teaching Excellence, Stanford University
2002: "A Primate's Memoir": winner of non-fiction book of the year, Bay Area Book Reviewers Association; finalist, Aventis Prize; finalist, The Natural World Book Prize. Winner of Book of the Year, BILD DER WISSENSCHAFT, German edition.
Winner of "The Emperor Has No Clothes" Award, Freedom From Religion Foundation
2004: Finalist, National Magazine Award, essay division for "The pleasures (and pain) of 'maybe'", Natural History magazine.
Paper # 315 included in Pinker S 2004 The Best American Science and Nature Writing. Houghton Mifflin Co, NY.
2006: Earl Usdin Award of the West Coast College of Biological Psychiatry
2007: John P. McGovern Award, American Association for the Advancement of Science.
2009: Gores Award for Teaching Excellence, Stanford University.
Lewis Thomas Award, Rockefeller University.

2
Fellow, Association of Psychological Science
2010: Isaac Asimov Award, American Humanist Association
2013: Distinguished Scientific Contributions Award, American Psychological Association
Paper #440 included in Mukherje S 2013 Best American Science and Nature Writing. Mariner Books, NY.
2018: Winner, LA Times Book Prize, Science & Technology
Winner, Phi Beta Kappa Science Book Award
The Against Stupidity award of the Philosophy Now Society, London.
Finalist, PEN Literary Award.

Current and Past Professional Activities

Editorial board: Neurobiology of Aging
 Journal of Neuroscience
 Psychoneuroendocrinology
 Journal of Neurochemistry
 Stress
 Frontiers in Cultural Psychology

Professional Writing Associations

2012 - Contributing writer to the Opinion column, Los Angeles Times.
2013 - 2017 Bi-weekly columnist, "Mind & Matter" column, Wall Street Journal.

Patents

8,101,158: Methods for treating cerebrovascular disease comprising administering an agent that inhibits prokineticin receptor activity.

College Courses on Video/Audio: The Teaching Company/The Great Courses

Sapolsky R. Biology and Human Behavior: The Neurological Origins of Individuality. 1994; 2005 2nd edition.
Sapolsky R. Stress and Your Body. 2010.
Sapolsky R. Being Human: Life Lessons from the Frontiers of Science. 2012.

Publications: Books

1. Sapolsky R. 1992 *Stress, the Aging Brain, and the Mechanisms of Neuron Death*. MIT Press.
2. Sapolsky R. 1994 *Why Zebras Don't Get Ulcers: A Guide to Stress, Stress-Related Disease and Coping*. Scientific American/Freeman Press/Henry Holt. Thirteen foreign language editions; Los Angeles Times Book Award finalist. 1998: Second edition. 2004: third edition.
3. Sapolsky R 1997 *'The Trouble With Testosterone' and Other Essays on the Biology of the Human Predicament*. Simon and Schuster/Scribner, 1997; Two foreign language editions; Los Angeles Times Book Award finalist.
4. Sapolsky R 2001 *A Primate's Memoir*. Simon and Schuster/Scribner, nine foreign language editions. Non-fiction book of the year, Bay Area Book Reviewer's Association, 2002; finalist, Aventis Prize; finalist, The Natural World Book Prize.
5. Sapolsky R. 2005 *'Monkeyluv' and Other Essays on our Lives as Animals*. Simon and Schuster/Scribner. Four foreign language editions.
6. Sapolsky R. 2017 *Behave: The Biology of Humans at our Best and Worst*. Penguin/Random House. Nineteen foreign language editions. Winner, LA Times Book Prize; Winner, Phi Beta Kappa Science Book Award; Finalist, 2018 PEN Literary Science Writing Prize.

Publications: Articles and Book Chapters

1. Sapolsky R, H Eichenbaum 1980 Thalamo-cortical mechanisms in odor guided behavior. *Brain Behavior and Evolution* 17, 276.
2. Sapolsky R, H Eichenbaum 1980 Still-life photographs: The power of human ethology in the explanation of human behavior. *Behavioral and Brain Sciences* 3, 628.
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10. Sapolsky R, Krey L, McEwen B, Rainbow T 1984 Do vasopressin-related peptides induce hippocampal corticosterone receptors? Implications for aging. *Journal of Neuroscience* 4, 1479.
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