CHILDHOOD TRAUMA: A BASIS FOR THE INSANITY DEFENSE AND OTHER MITIGATING THEORIES IN LIGHT OF TRAUMA-LINKED BIOCOGNITIVE DEFICIENCIES

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A growing body of research shows a link between early life stress and neuropsychiatric irregularities in adulthood. The consequences of such childhood trauma include increased sympathetic (fight-or-flight) arousal as well as quantifiably lower levels of behavior control. In criminal cases where an accused has a background of trauma in early life, the documented sequelae of childhood trauma may form the basis for a number of defense strategies. These theories include insanity, reduced capacity for specific intent crimes, and mitigation at sentencing.

INTRODUCTION

As scientific understanding of childhood trauma and its aftereffects grows, so too does the potential to apply this understanding to the criminal justice system. There is a traceable correlation between childhood trauma and criminal activity. In August of 2019, two experts stated that the majority of mass shooters in the US since the 1960s have undergone some form of childhood trauma.1 A study published in 2013 found that a group of offenders, who were convicted of crimes including assault, child abuse, and sexual offenses, had four times the amount of adverse childhood trauma in comparison to a normative group.2

The connection between trauma and criminal activity raises the question of how trauma and its long-term and biopsychiatric consequences affect criminal

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liability for actions committed by survivors of early life stress. Recently, Gohara (herself an experienced defense attorney and clinical faculty at Yale Law School) has argued passionately and eloquently that the effects of trauma should be invoked by defense attorneys in non-capital cases (where sentencing hearings are less extensive than death penalty cases). Specifically, she has argued that childhood trauma should be investigated by defense counsel and presented as scientific mitigating evidence at the sentencing phase of a trial, after an accused offender has been convicted. 

Here I take a somewhat similar tack, asking how childhood trauma might be invoked by a savvy criminal defense attorney in non-capital cases. But I approach this matter with a different emphasis. First, I offer a more mechanistic approach to the biocognitive sequelae of childhood trauma, offering examples of how scientists go about the painstaking work of elucidating the connections between trauma and biological derangements. This information may be useful to practitioners of criminal defense, who are often resource-stretched, in collating information and organizing a defense based on childhood trauma. Second, I offer a bolder theory of how these neuropsychiatric changes might form the basis for criminal defenses, including the insanity defense—which would remove criminal culpability from an accused.

**Effects of Trauma on the Brain and Body**

The long-term implications of childhood trauma were explored extensively in a landmark study of over 9,000 adults in 1998 that looked at adverse childhood experiences (“ACE”) and their effects later in life. Examples of such adverse experiences included psychological, physical, and sexual abuse as well as severe poverty and incarceration of a household member. Notably, the ACE study found that those with four or more examples of childhood trauma were at higher risk for a wide range of problems, including heart disease, diabetes, stroke, suicide, mental health issues, tobacco and other substance use. In other words, childhood trauma is associated with negative consequences (mental and physical) that follow survivors into adulthood.

Since then, science has engaged in more focused study of how childhood trauma affects the brain’s functioning. While much has been written on the biological effects of childhood trauma and summarized by De Bellis and Zisk as 

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CHILDHOOD TRAUMA IS ASSOCIATED WITH HORMONAL DISRUPTION

First, trauma can disrupt the brain’s secretion of stress hormones, including cortisol and cortisol-releasing factor (“CRF”), which is also known as cortisol-releasing hormone (“CRH”). When the body encounters stress, be it psychological or physiological, the brain releases CRF/CRH, which then causes the release of cortisol. These hormones can interact with the sympathetic nervous system, colloquially referred to as our “fight-or-flight” mechanism. Activation of the sympathetic nervous system can create an agitated state with increased vigilance, a fast heart rate, increased blood pressure, and dilated (enlarged) pupils.

While scientists are still working to precisely map out how childhood trauma affects the release of stress hormones and the sympathetic nervous system, a number of articles and studies point to a disruption in CRF/CRH and cortisol levels, which could then promote increased sympathetic, fight-or-flight, activation. For example, one study of about fifty adults (depressed and non-depressed) examined the cerebrospinal fluid, which runs from the brain through the spine, and found increased levels of CRF/CRH among subjects who perceived early life adversity. This hormonal increase was independent of the participants’ depression status. Another study of twenty men with personality disorders found a similar result: increased CRF/CRH that correlated with reported childhood trauma.

What is the significance of the elevated CRF/CRH in those reporting childhood trauma? While much is to be clarified, it is known that CRF/CRH activates a part of the brain called the locus coeruleus. The locus coeruleus releases the hormone norepinephrine which causes sympathetic (fight-or-flight) activation across the whole body. Taken together, it stands to reason that childhood trauma is associated with elevated levels of CRF/CRH in the brain which can in turn activate the fight-or-flight sympathetic nervous system. Indeed, this idea is supported by the finding that a group of Vietnam combat veterans diagnosed with

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7. De Bellis & Zisk, supra note 5.
8. Id.
9. Id.
11. Royce Lee et al., Childhood Trauma and Personality Disorder: Positive Correlation With Adult CSF Corticotropin-Releasing Factor Concentrations, 162 AM. J. PSYCHIATRY 995, 995–97 (2005).
post-traumatic stress disorder ("PTSD") had a higher amount of CRF/CRH in their cerebrospinal fluid.\textsuperscript{13}

A brief note on PTSD. PTSD is a psychiatric condition recognized in the Diagnostic and Statistical Manual of Mental Disorders ("DSM-5") which follows after witnessing trauma. PTSD is accompanied by symptoms that can include intrusive recollection of the trauma, dissociative reactions (flashbacks), nightmares, and social withdrawal.\textsuperscript{14} Note that trauma is not synonymous or one-to-one with PTSD. While all people with PTSD have experienced trauma, not everyone who experiences trauma (in early life or otherwise) ends up with PTSD. This article aims to address the broader changes evoked by early life stress, rather than the very specific condition of PTSD, which burdens some but not all trauma survivors.

In addition to increased CRF/CRH in the spinal fluid, childhood trauma may also be associated with a change in cortisol levels that would affect sympathetic activity. Cortisol is a hormone released when the body faces stress.\textsuperscript{15} A longitudinal study of sexually abused young women found that they showed decreased levels of cortisol in adulthood.\textsuperscript{16}

It has been suggested that cortisol may act as a restraint on sympathetic activity.\textsuperscript{17} The inverse relationship between decreasing cortisol levels and increasing sympathetic activation is alluded to in a study which documented levels of cortisol and catecholamines (which are increased during sympathetic activation) in children with PTSD following motor vehicle accidents. This study found that cortisol levels initially increased in the month after the trauma (a predictable response to stress) and later decreased to normal by month six.\textsuperscript{18} On the other hand, catecholamine levels increased between months one and six.\textsuperscript{19} There is thus a hypothesis that cortisol may act as a restraint on the sympathetic system.\textsuperscript{20} If trauma, then, were to cause a later-life decrease in cortisol levels, that decrease could take the brakes off the amping-up effects of the sympathetic system.


\textsuperscript{14} AM. PSYCHIATRIC ASSN, \textit{DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS} 645, 649 (5th ed. 2013).

\textsuperscript{15} Penelope K. Trickett et al., \textit{Attenuation of Cortisol Across Development For Victims of Sexual Abuse}, 22 DEV. & PSYCHOPATHOLOGY 165 (2010).

\textsuperscript{16} Id.

\textsuperscript{17} Eva Fries et al., \textit{A New View on Hypocortisolism}, 30 PSYCHONEUROENDOCRINOLOGY 1010, 1012 (2005); See also Panagiota Pervanidou et al., \textit{The Natural History of Neuroendocrine Changes in Pediatric Posttraumatic Stress Disorder (PTSD) After Motor Vehicle Accidents: Progressive Divergence of Noradrenaline and Cortisol Concentrations Over Time}, 62 BIOLOGICAL PSYCHIATRY 1095 (2007).

\textsuperscript{18} Pervanidou, supra note 17, at 1095.

\textsuperscript{19} Id.

\textsuperscript{20} Fries, supra note 17, at 1012 (“Interestingly, increased catecholamine concentrations have been reported in patients with stress-related disorders characterized by hypocortisolemic stress responses”); See also Pervanidou, supra note 17, at 1095.
CHILDHOOD TRAUMA IS ALSO ASSOCIATED WITH QUANTIFIABLE CHANGES IN BRAIN FUNCTION

In addition to hormonal effects that would predispose survivors of childhood trauma to an agitated state of higher sympathetic activation, scientists have documented changes to the brain activity of those who have survived childhood trauma. One study used electroencephalography (where electrodes are attached to the head to measure brain activity) to study participants’ abilities to regulate their actions. Participants’ childhood trauma experiences were assessed, and they were put in front of a monitor with instructions to click when an even number was shown (referred to as “Go” stimulus) and not click when an odd number is shown (“NoGo” stimulus, as in “no go”). As the scientists who conducted this study predicted, the sub-group reporting higher childhood trauma showed decreased activity in parts of the brain associated with inhibitor processes, i.e., regulators of impulsivity.21

Another study using a similar Go/NoGo test (this one required clicking target letters x, y, or z that were interspersed with other letters) found a similar correlation between early childhood trauma and poorer performance. Notably, this result held true between those with bipolar disorder and healthy controls. As in the previously referenced study, those with childhood trauma had a harder time not clicking erroneously—which some might view as a marker for impulsivity.22

How might poorer impulse control play itself out? A study published in late 2017 aimed to answer this question by looking at the how childhood trauma correlated with adult decision-making.23 Fifty-four adult participants (average age of 20.5 years) were chosen from a group who had been previously surveyed for early life stress during their childhood.24 Twenty-nine of the participants had high stress in childhood, twenty-five of whom had relatively low levels of stress.25 These participants underwent testing first in a gambling exercise and second in a separate exercise where participants had to quickly press a button while a cue was shown on a computer screen after being shown the potential to either win or lose money or break even. These exercises were correlated with brain imaging.

The results of this study showed demonstrable differences between the high childhood stress group and the low-stress group. During the gambling exercise, participants from the high childhood stress group placed more losing bets even after gaining experience with the game, while members of the low childhood stress group learned from their errors and made lower bets when the odds were

22. David F. Marshall et al., Deficient Inhibitory Control As An Outcome of Childhood Trauma, 235 PSYCHIATRY RES. 7, 7–12 (2016).
24. Id.
25. Id.
worse.\textsuperscript{26} During the second exercise, participants from the high-stress group showed less brain activity (as measured by a kind of MRI) when contemplating potential wins and losses.\textsuperscript{27} The imaging information from the second exercise was then compared with the results from the gambling tests. It turned out that those with higher activation considering loss (correlated with lower childhood stress) did a better job adapting to the gambling game.\textsuperscript{28}

Lastly, the scientists looked at adult risky behavior like substance use, carrying a weapon, criminal activity, and sex. The group with high early childhood stress had an overall higher rate of risky behavior. Moreover, this higher rate correlated with lower brain activation during consideration of future loss in the second exercise, specifically in a part of the brain called the putamen.\textsuperscript{29}

As a whole, the results of Birn, Roeber, & Pollak’s study suggest a link between higher childhood stress and impaired decision making as an adult.\textsuperscript{30}

LEGAL IMPLICATIONS OF DOCUMENTED EFFECTS OF CHILDHOOD TRAUMA ON MENTAL FUNCTION

In summation, there is a growing body of evidence that childhood trauma can affect brain function into adulthood. Of particular note is the activation of the fight-or-flight sympathetic system, triggered by hormone imbalances as well as measurable changes to the brain’s function, particularly as it concerns impulsivity and self-control. If childhood trauma can negatively impact self-control by predisposing survivors to hypervigilant sympathetic activation and by limiting self-regulation, what does this mean for the criminal justice system?

CHILDHOOD TRAUMA AND THE INSANITY DEFENSE

One possibility would be limiting criminal liability for childhood trauma survivors based on fairness. It does seem fundamentally unfair to punish someone for actions over which they possess impaired self-control. The most far-reaching of these possibilities would be to completely exculpate such trauma survivors under the defense theory commonly referred to as the insanity defense.

Different states follow different rules for the insanity defense, and some do not offer the accused this defense at all. While there are many nuances that are best summarized elsewhere,\textsuperscript{31} the two most widespread standards, which differ by jurisdiction, are the \textit{M’Naghten} rule and the Model Penal Code. The \textit{M’Naghten} rule, a version of which is incorporated into the American federal penal code, states that a defendant may be acquitted if he “as a result of a severe mental

\begin{itemize}
\item\textsuperscript{26} Id.
\item\textsuperscript{27} Id.
\item\textsuperscript{28} Id.
\item\textsuperscript{29} Id.
\item\textsuperscript{30} Id.
\item\textsuperscript{31} AAPL Practice Guideline for Forensic Psychiatric Evaluation of Defendants Raising the Insanity Defense, 42 J. AM. ACAD. PSYCHIATRY & L. S3 (2014) [hereinafter AAPL Practice Guide].
\end{itemize}
disease or defect, was unable to appreciate the nature and quality or the wrong-
fullness of his acts." The Model Penal Code, generally less restrictive, allows
for acquittal where mental disease or defect either prevents the defendant from
appreciating the wrongfulness of their conduct (which is similar to the M’Naught-
ten rule) or prevents the defendant from conforming their conduct to the require-
ments of the law. Thus, one could meet the Model Penal Code’s insanity re-
quirement by either not being able to appreciate that their conduct is wrong or
by having some unspecified impairment in controlling their actions.

At the outset, the Model Penal Code offers a framework more conducive to
using childhood trauma as a basis for the insanity defense because (a) it offers
two different ways to show insanity and (b) one of those two tests is a vaguer
standard that gives defense counsel flexibility.

In regard to the second Model Penal Code standard of inability to conform
one’s actions to the law (the first standard of appreciating wrongdoing will be
addressed as well), an accused person with a background of early life stress may,
as described above, have atypical levels of hormones that predispose them to
agitation and hypervigilance, as well an impaired ability to control impulsivity.
Of particular relevance are studies on the “NoGo” brain function, which relate to
the brain’s ability to exercise self-restraint. Additionally, Birn et. al’s study of
decreased brain activity when contemplating losses could be invoked to show
a decreased ability to consider potential repercussions and adjust course accord-
ingly. Someone with limited self-control would naturally have difficulty con-
forming their conduct to the law’s requirements.

Even under the narrower M’Naughten standard—which is similar to the
Model Penal Code’s first test—a defendant might claim that their sympathetic
activation put them into a primal, fight-or-flight state that made it difficult to
appreciate right from wrong. This, however, might be a more difficult case to
make to a judge or jury.

It is noteworthy that PTSD has been successfully invoked under the insan-
ity defense. As noted earlier, however, this article aims to take a broader view
of childhood trauma to address more than those who have PTSD.

The kinds of offenses for which one might cite childhood trauma as a basis
for an insanity defense would likely be those involving a shorter timeline, i.e.,
crimes in the heat of the moment. This is because the effects of childhood trauma
seem to implicate decision-making in the short-term, when a person might be
amped-up, so to speak, with sympathetic stimulation and burdened with an im-
paired ability to self-regulate. One could imagine a situation where childhood
trauma is used as a defense in an assault case that started as an escalating verbal
argument where the passion of the moment took hold of the defendant. In this

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34. Kim, supra note 21; Marshall, supra note 22; Birn, supra note 23.
35. Birn, supra note 23.
case, a defendant might argue temporary insanity due to the sequelae of childhood trauma. On the other hand, it could strain credulity to argue that childhood trauma drove a terrorist to methodically plan an attack over the course of months.

**OTHER POSSIBLE CRIMINAL DEFENSE STRATEGIES INVOLVING CHILDHOOD TRAUMA**

A successful insanity defense would lead to a finding of not guilty with transfer to a psychiatric institution for care—with the possibility of partial or full release upon medical determination. In that case, the insanity defense removes all criminal culpability, making it potentially less appealing due to the shades of grey inherent in criminal cases. Maybe the defendant’s self-control was impaired but not to the extent that they deserve to be found not guilty. Two other possibilities that split the Solomonic baby are finding the defendant guilty of a lesser crime under a theory of diminished responsibility or finding the defendant guilty by using the history of childhood trauma (and the attending differences in brain function) to justify a lower sentence.

The defense of diminished capacity would emphasize that a defendant’s mental state prevented him from having the required state of mind (mens rea in Latin) required for an offense. One example would be reducing first-degree murder, which requires premeditation, to manslaughter, which is an intentional wrongful killing.

This defense does not have an illustrious history, as it was most famously cited as the “Twinkie” defense in the murder trial of San Francisco politician Dan White. White was charged with first-degree (premediated) murder in the killing of Supervisor Harvey Milk and Mayor George Moscone. While White admitted to bringing a gun to City Hall to kill both former colleagues, even reloading between the first and second killings, the jury did not find him guilty of first-degree murder. Instead, White was convicted of manslaughter and spent about five years in confinement. White succeeded by invoking major depressive disorder as a basis for diminished capacity. In other words, his legal team persuaded the jury that White’s depression prevented him from forming the cold, calculating state of mind that first-degree murder seeks to punish.

To clarify the historical origin of the term “Twinkie defense,” a testifying psychiatrist did point out that switching to a diet of junk food like soda and sweets could be evidence of the depression itself—not that Twinkies or any other food product caused White to kill. A satirical cartoon artist, however, later coined the term “Twinkie defense,” which has somehow persisted in the popular lexicon. In response, California eliminated the defense of diminished capacity.

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38. Id.
39. Id.
40. CAL. PENAL CODE 25(a).
Nonetheless, the defense of diminished capacity is still available for specific intent crimes in some jurisdictions such as the Ninth Circuit Court of Appeals.41 “Specific intent” refers to a goal, such as in first-degree murder, where the killing was done with purpose of killing someone. Manslaughter, by contrast, requires only a voluntary killing without the pre-mediated, coolly-formed intent to kill. One might imagine the difference between a hunter who shoots a hiker by accident (pulling the trigger intentionally but without intent to kill a person) versus a hitman who identifies and kills their target (intentionally pulling the trigger with a well-formed plan to end a person’s life). This gradation allows the penal system to distinguish between so-called crimes of passion and the most nefarious offenses, those that are coldly-planned.

The second possibility for accounting for childhood trauma while not letting a defendant evade complete responsibility is allowing evidence of childhood trauma during sentencing. This is the course advocated by Gohara, who writes in detail about the social justice aspects of this approach.42 Criminal trials consist of a guilt phase, where a judge or jury determines if the accused committed a crime without justification or defense, and a sentencing phase, where the penalty is imposed, most often by a judge.43 A convicted offender, having already been found guilty, might choose to describe their childhood trauma in asking for a more lenient sentence. The offender employing this strategy would point out the documented neurocognitive effects of such trauma, and appeal to a sense of fairness on the sentencing authority’s part.

In federal courts in the United States, judges impose penalties aside from death (which requires a jury decision). Without delving into the intricacies of the federal sentencing system, federal judges follow non-mandatory guidelines issued by the US Sentencing Commission. These guidelines provide that:

A downward departure may be warranted if (1) the defendant committed the offense while suffering from a significantly reduced mental capacity; and (2) the significantly reduced mental capacity contributed substantially to the commission of the offense. Similarly, if a departure is warranted under this policy statement, the extent of the departure should reflect the extent to which the reduced mental capacity contributed to the commission of the offense.44

Thus, a survivor of childhood trauma might argue that their mental capacity was diminished due to the lingering aftereffects on their body’s chemistry and brain function. Note that diminished capacity here could lead to a reduced sentence without changing the quality of the crime charged. This is unlike the case of Dan

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41 Megan Testa & Susan Hatters Friedman, Diminished Capacity, 40 J. AM. ACAD. PSYCHIATRY & L. Online 567, 568 (2012).
42 Gohara, supra note 3, at 25–32.
43 Definition of Key Terms, CAPITAL PUNISHMENT IN CONTEXT, https://capitalpunishmentincontext.org/resources/definitions (last visited Feb. 20, 2020).
44 U.S. SENTENCING GUIDELINES MANUAL § 5K2.13 (U.S. SENTENCING COM’N 2018).
White, noted above, where diminished capacity led to a conviction for a lesser offense. Raising the issue of childhood trauma, and its effects on the defendant’s cognitive processes, provides something of a compromise whereby a defendant is held accountable with a conviction, but their limited capacity for self-regulation might be recognized with a downward departure in sentencing.

**CHALLENGES AND TACTICAL CONSIDERATIONS**

Several tactical considerations would be relevant to attorneys seeking to invoke childhood trauma as the basis for an insanity defense, a diminished capacity defense, or mitigation in sentencing.

First and foremost, there is no ready DSM-5 diagnosis for “survivor of childhood trauma with concomitant neuropsychological changes.” Thus, the attributes most contributing to the inability to recognize wrongdoing or adequately control conduct will have to be shoehorned into other diagnoses that are well recognized such as PTSD, depression, and anxiety. Experts like a forensic psychiatrist would play a critical role in teaching the judge or jury about how childhood trauma underlies the accused’s behavior.

This role of the expert raises a second challenge, the question of whether a court would admit evidence of the link between childhood trauma and behavioral impairment. Solid expert testimony would be critical to meeting the test of admissibility. The most common standard set forth in the *Daubert* case and codified in Federal Rule of Evidence 702 set forth criteria for the admission of expert testimony. These criteria include relevance, whether the testimony is based on sufficient data, whether the testimony is the product of reliable principles, and whether these principles have been reliably applied to a case. Additionally, *Daubert* also included an element of general scientific acceptance.

Given the relative novelty of applying childhood trauma to criminal defense, one might expect government prosecutors to work to prevent expert testimony on childhood trauma.

Third, the experiments referenced here deal with specialized labs doing ad hoc testing. In all but the highest-stakes trials (e.g., capital cases), it would be cumbersome and resource-intensive to perform tests like the EEG, fMRI, and lumbar puncture to replicate these experiments. And doing those tests may not yield a result helpful for the accused. It would fall to expert testimony (versus imaging and other “objective” tests) to carry the day.

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46. FED. R. EVID. 702.
47. Id.
Part of a workaround in the absence of the ability to perform “objective” testing would be to use the ACE questionnaire\textsuperscript{49} or the Childhood Trauma Questionnaire\textsuperscript{50} to illustrate the experiences of the accused. In some ways, this is similar to the idea of a clinical diagnosis in medicine where a child who is symptomatic for strep throat may be treated with penicillin. The idea is that expensive testing is unnecessary if there is sufficient childhood trauma and there is evidence of behavior linked to childhood trauma. For example, a defendant may demonstrate a personal history of impulsiveness and difficult behavior going back to childhood.

Lastly, even if these technical challenges are overcome, there remains the popular idea of blind justice. That no one but the defendant did what they did, and that it would be unfair to let some defendants win leniency by invoking stories of difficult childhoods. Not everyone, after all, with a hard story ends up committing crimes. Solving this issue definitively is beyond the scope of this article.

That said, vigorous and intrepid advocates would point out that the weight of the current scientific body of knowledge has revealed lasting biological changes linked to childhood trauma. In the same way that not all schizophrenics or people with PTSD commit crimes but, when they do, they might be found not guilty where their faculties were lacking. The same logic would hold true for childhood trauma survivors. In other words, mental illness may contribute to acts chargeable as crimes, even if that link is not always one-to-one. And when it does, it should be accounted for in the interest of fairness to a brain and body damaged by the effects of early childhood stress.

CONCLUSION: THE WAY FORWARD IN RECOGNIZING CHILDHOOD TRAUMA IN THE CRIMINAL JUSTICE SYSTEM

Moving forward, the burden of advancing the incorporation of childhood trauma into criminal defense will likely rest with defense counsel. Elected prosecutors and legislators may not be eager to seem “soft” on crime. Some may even be sympathetic to the idea that childhood trauma may create effects that should limit a defendant’s criminal liability. Nonetheless, the jeopardy of a defendant’s freedom will provide a powerful motivator for adopting a defense based on a theory of childhood trauma.

Defense attorneys should ask their clients questions about trauma background the same way they would issue-spot affirmative defenses like self-defense or probe for evidentiary holes in the government’s case. Many artful attorneys already aim to point out a client’s hardship in both trial and sentencing. Addressing childhood trauma scientifically would build on this strategy which plays out day after day in trial courts.

\textsuperscript{49} Felitti, supra note 4.
\textsuperscript{50} Marshall, supra note 22.
Scientists and clinicians can support this effort by continuing to study the biological and psychological effects of childhood trauma. Each study gives more insight into how early life stress can cause pernicious aftereffects. Additionally, there will need to be a body of experts ready to testify and teach judges and juries about the long-lasting effects of childhood trauma. Forensic psychiatrists in particular, with their medical backgrounds and advanced legal training, are well-positioned to discuss the biological and psychological effects of childhood trauma.

Lastly, this article is not intended to provide legal advice or serve as a comprehensive manual in the defense of criminal cases involving childhood trauma. Rather, the goal is to encourage consideration of how known sequelae of early life adversity might affect the survivors when they encounter the criminal justice system as defendants. At the very least, one might call into question the fairness of punishing those whose capacities for self-regulation are limited in light of hormonal and functional changes to their brains.