IN THE SUPREME COURT OF TENNESSEE
AT KNOXVILLE

STATE OF TENNESSEE,	)	
	)	
Appellee,	)	Knox County Criminal 108568
	)	
v.	)	C.C.A. No. E2018-01439-CCA-R3-CD
	)	
TYSHON BOOKER,	)	S. Ct. No. E2018-01439-SC-R11-CD
	)	
Appellant.	)	

BRIEF OF *AMICI CURIAE* NACDL, TACDL, AMOS BROWN, AND CHARLES LOWE-KELLEY

David R. Esquivel (#021459)
Jeff H. Gibson (#026321)
Sarah Miller (#033441)
Angela L. Bergman (#031981)
BASS, BERRY & SIMS PLC
150 Third Avenue South, Suite 2800
Nashville, TN 37201
Phone: 615-742-6200
Email: desquivel@bassberry.com
jgibson@bassberry.com
smiller@bassberry.com
abergman@bassberry.com

Counsel for Charles E. Lowe-Kelley

Bradley A. MacLean (#009562)
Law Office of Bradley A. MacLean
1702 Villa Place
Nashville, TN 37212

Counsel for Charles Lowe-Kelley and Amos Brown

Lucille A. Jewel (#032666)
1505 W. Cumberland Avenue
Knoxville, TN 37996-1810

-and-

Stephen Ross Johnson (#022140)
Sixth Circuit Vice-Chair
Amicus Committee of the
National Association of Criminal
Defense Lawyers

Ritchie, Dillard, Davies, & Johnson
606 W. Main Street, Suite 300
Knoxville, TN 37902

Counsel for National Association of Criminal Defense Attorneys

Jonathan D. Cooper (#016041)
President,
Tennessee Association of Criminal
Defense Lawyers
530 Church Street, Suite 300
Nashville, TN 37219
(615) 329-1338

Counsel for Tennessee Association of Criminal Defense Attorneys

TABLE OF CONTENTS

TABLE OF AUTHORITIES

Cases **Page(s)**

State Statutes

Rules

Constitutional Provisions

Other Authorities

Amici Curiae the National Association of Criminal Defense Attorneys ("NACDL"), the Tennessee Association of Criminal Defense Attorneys ("TACDL"), Amos Brown, and Charles Lowe-Kelley submit this brief in support of Appellant Tyshon Booker's application for permission to appeal under Tenn. R. App. P. 11.

I. STATEMENT OF THE ISSUE

Amici urge the Court to address the following issue:

> Whether a minimum 51-year term of prison confinement mandatorily imposed on a juvenile, without consideration of the juvenile's youth, immaturity, or other mitigating circumstances, violates the Cruel and Unusual Punishments Clauses and other provisions of the federal and state constitutions, in that it deprives the juvenile of a "meaningful opportunity to obtain release based on demonstrated maturity and rehabilitation."

II. INTERESTS OF *AMICI*

The interests of *Amici* are more fully described in their *Motion for Leave to File Amici Curiae Brief*, filed contemporaneously herewith. Their interests can be briefly summarized as follows:

NACDL is the leading national bar association for criminal defense attorneys. NACDL's mission includes working for improvement in the criminal justice system. To fulfill this mission, NACDL submits amicus briefs on important criminal justice issues of national significance.

TACDL is the leading bar association for Tennessee criminal defense attorneys whose mission includes working for improvement in the criminal justice system. To fulfill this mission, TACDL submits amicus briefs on important issues that affect the administration of criminal justice in Tennessee.

Amos Brown is serving a life sentence for felony murder for a crime that occurred when he was 16 years old and will not be eligible for release until he is at least 69 years old. He is challenging the constitutionality of his 51-year mandatory minimum life sentence in a post-conviction proceeding that is pending in McMinn County. *Amos Brown v. State*, No. 4-CR-64 (McMinn Cnty. Cir. Ct.). In support of his petition, Mr. Brown filed the *Declaration of Dr. Julie A. Gallagher*, a forensic psychologist who summarized the current scientific research (as of May 2018) on adolescent psychology and brain development that the Supreme Court has deemed relevant in addressing issues concerning juvenile sentencing under the Eighth Amendment. Dr. Gallagher's Declaration is attached hereto as Appendix A. Mr. Brown also filed the *Declaration of Dr. Michael Freeman*, an epidemiologist who reviewed demographic data from the Tennessee Department of Correction to ascertain that Tennessee inmates confined in the Tennessee prison system have an average life expectancy of 52 years old. Dr. Freeman's Declaration is attached hereto as Appendix B.

Charles Lowe-Kelley is currently serving two consecutive life sentences for crimes that occurred when he was 16 years old. Under current Tennessee law, he will be ineligible for release until he is well over 100 years old, which means that he will certainly die in prison. The sentencing judge expressly stated that Mr. Lowe-Kelley's youth would not be considered as a mitigating factor in his sentencing. Mr. Lowe-Kelley is challenging the constitutionality of his sentence in a pending federal habeas corpus proceeding in the Middle District of Tennessee. *Lowe-Kelley v. Washburn*, No. 1:16-cv-00082 (M.D. Tenn.).

III. REASONS TO GRANT THE APPEAL

In *Miller v. Alabama*, 567 U.S. 460 (2012), and *Montgomery v. Louisiana*, 136 S. Ct. 718 (2016), the United States Supreme Court, declaring that juveniles are constitutionally different from adults, invalidated mandatory life without parole ("LWOP") sentences imposed on juveniles convicted of murder. The Supreme Court held that, in light of our contemporary understanding of adolescent psychology and brain development, it is unconstitutional to mandatorily deprive a juvenile offender of "a meaningful opportunity to obtain release based on demonstrated maturity and rehabilitation." Tennessee's mandatory life sentence for first-degree murder deprives juvenile defendants of such a "meaningful opportunity" and is therefore unconstitutional.

Tennessee's life sentence requires a minimum of 51 years in prison, making it among the most extreme in the country.[1] It is mandatorily imposed on juveniles—the minimum sentence any juvenile convicted of first-degree homicide can receive is life. This sentencing structure entirely forecloses consideration of the characteristics of youth the Supreme Court has dictated must be examined under the Constitution. This sentence also exceeds the average life expectancy of

[1] See *False Hope: How Parole Systems Fail Youth Serving Extreme Sentences*, Appendix A at 160-61 (ACLU, Nov. 2016) (listing Tennessee's mandatory life sentence as the most extreme among the states).

Tennessee juveniles serving life sentences.[2] A 51 year sentence is in fact more extreme for teens than adults, because the average teenager sentenced to life will spend more time and a greater percentage of his life in prison before he dies. This violates the constitutional principle that juveniles are less culpable and more amendable to rehabilitation, and therefore should be treated less harshly than adult offenders.[3]

Amici acknowledge that the Tennessee Court of Criminal Appeals has previously rejected this claim, and on prior occasions this Court has denied permission to appeal on this issue. But, *Amici* respectfully contend that the Court of Criminal Appeals has erroneously applied a narrow, formulaic rule that misconstrues the holdings in *Miller* and *Montgomery*. Finding that *Miller* and *Montgomery* only apply when the sentence is expressed as "life without parole," the Court of Criminal Appeals has concluded that because Tennessee's life sentence theoretically allows for some remote chance of release after 51 years, *Miller* and *Montgomery* do not apply in Tennessee. This is wholly out of step with jurisdictions across the country, which have held that even a

---

[2] According to the evidence submitted in Amos Brown's case, average life expectancy in prison is 52 years of age. *See Freeman Declaration* attached at Appendix B, at 4. Moreover, research shows that juveniles sentenced to life in prison have an even lower life expectancy. *See id.* at 5.

[3] *See Miller*, 567 U.S. at 475 ("And this lengthiest possible incarceration is an especially harsh punishment for a juvenile, because he will almost inevitably serve more years and a greater percentage of his life in prison than an adult offender." (quoting *Graham*, 560 U.S. at 70)).

term-of-years sentence (as opposed to the life sentence at issue here) operates as a *de facto* LWOP sentence, raising *Miller* constitutional issues.[4]

The essential holding of *Miller* and *Montgomery* is that a mandatory sentence violates the Constitution if it deprives a juvenile of a "meaningful opportunity to obtain release based on demonstrated maturity and rehabilitation." The critical determination is: What amounts to a "meaningful opportunity" in this context? The extremely remote and highly unlikely possibility of a geriatric release after a half-century of incarceration, if a person can survive that long in prison, is not "meaningful."

For at least four reasons, in order to secure settlement of questions of public interest and of important questions of law, this issue is ripe for review by this Court under Tenn. R. App. P. 11(a)(2) and (3).

First, this issue is of profound public interest because it concerns our conception and treatment of juveniles, who have always received protection under the law and who have been shown, by contemporary science, to be less culpable and more capable of rehabilitation than adults.

Second, this issue raises fundamental questions of federal and state constitutional interpretation including how states provide the "meaningful opportunity" guaranteed by the federal Constitution.

[4] See cases cited in Section V.D., *infra*.

Third, because Tennessee's 51 year mandatory minimum sentence for juveniles convicted of murder is among the most extreme in the nation,[5] this Court should reconsider Tennessee's outlier position. Courts across the country have held that excessively long mandatory sentences deprive juveniles of a "meaningful opportunity." This case offers the Court the opportunity to consider whether Tennessee should align itself with these other jurisdictions.

And fourth, even members of the Tennessee Court of Criminal Appeals have recently expressed concern about the severity of a 51-year mandatory minimum sentence for a juvenile, pointing out that, in reality, such a sentence deprives a juvenile of a "meaningful opportunity." As Judge Thomas explained:

> [A]lthough Tennessee's sentencing scheme allows for possible release of a defendant convicted of first degree murder after the service of fifty-one years, it is only in the rare instance, if ever, that a ***juvenile*** so sentenced would be released back into society. Even if the judge or jury decides that the features of the juvenile or the circumstances of the homicide require a sentence other than life without parole, the effect of the sentence is still the same. ***The juvenile has no meaningful opportunity for release whether you name the sentence imprisonment for life or imprisonment for life without the possibility of parole, and the juvenile will likely die in prison. "While the logical next step may be to extend protection to these types of sentences, that is not the precedent which now exists" in this State***.

[5] *See* section V.E., *infra.*

State v. Zachary Everett Davis, No M2016-01579-CCA-R3-CD (Tenn. Crim. App. Dec. 11, 2017) (Thomas, J. and McMullen, J., concurring) (quoting *Floyd Lee Perry, Jr., v. State*, No. W2013–00901–CCA–R3–PC, 2014 WL 1377579, at *4 (Tenn. Crim. App. Apr. 7, 2014), *perm. app. denied* (Tenn. Sept. 18, 2014)) (emphasis added).[6] *See, also, Jacob Brown v. State*, No. W2015-00887-CCA-R3-PC, 2016 WL 1562981, at *7 (Tenn. Crim. App. Apr. 15, 2016), *perm. app. denied* (Aug. 19, 2016), *cert. denied,* 137 S. Ct. 1331 (2017) (expressing "misgivings" about consecutive life sentences for a juvenile).

Despite a pattern of doubt over whether a juvenile can ever have a meaningful opportunity for release under Tennessee's scheme, the Court of Criminal Appeals has adhered to its narrow view. This Court should now consider modern developments in brain science and adolescent psychology confirming that children are less culpable and more amenable to rehabilitation, as well as evolving community standards of punishment, to determine whether Tennessee's mandatory life sentence is inconsistent with the holdings of *Miller* and *Montgomery*.

IV. STATEMENT OF THE CASE

Tyshon Booker's case provides this Court with an ideal opportunity to review the issue presented, because it is a textbook case of how

[6] *See, also, State v. Henderson*, No. W2016-00911-CCA-R3-CD, 2018 WL 1100972, at *6-7 (Tenn. Crim. App. Feb. 26, 2018); *State v. Collins*, No. W201601819CCAR3CD, 2018 WL 1876333, at *20–21 (Tenn. Crim. App. Apr. 18, 2018), *appeal denied* (Aug. 8, 2018), *cert. denied*, 139 S. Ct. 649 (2018) (both opinions quoting Judge Thomas's concurring opinion at length).

Tennessee's mandatory sentencing scheme in Tennessee fails juveniles. First, juveniles are less culpable than adults. Because of their young age, juveniles have a developmentally limited ability to self-regulate and resist outside peer influences. Second, for similar developmental reasons, juveniles are more likely to rehabilitate than adults. The record below includes expert testimony on the modern science of adolescent psychology and brain development, mitigating circumstances concerning Tyshon's traumatic childhood, and reasons why Tyshon is amenable to rehabilitation - the exact kinds of factors that the Supreme Court has found relevant in considering the constitutionality of mandatory sentencing for juveniles.

V. ARGUMENT

A. Juveniles are constitutionally different for sentencing purposes.

Beginning in 2005, the United States Supreme Court recognized that the Constitution requires states to distinguish juveniles from adults for sentencing purposes, "tak[ing] into account how children are different, and how those differences counsel against irrevocably sentencing them to a lifetime in prison." *Miller*, 567 U.S. at 480. Absent a finding that a child is "irreparab[ly] corrupt[]" and incapable of rehabilitation, a child cannot be denied "hope for some years of life outside prison walls." *Montgomery*, 136 S. Ct. at 736-37. Children must be given "a meaningful opportunity to obtain release based on demonstrated maturity and rehabilitation." *Miller*, 567 U.S. at 479 (quoting *Graham*, 560 U.S. at 75.

The Supreme Court first ruled in *Roper v. Simmons*, 543 U.S. 551 (2005), that the Eighth Amendment prohibits the death penalty for juveniles, based on advancing scientific understanding of developmental psychology and neuroscience. The *Roper* Court recognized three general differences between juveniles and adults, relevant to criminal sentencing.

First, "[a] lack of maturity and an underdeveloped sense of responsibility are found in youth more often than in adults and are more understandable among the young. These qualities often result in impetuous and ill-considered actions and decisions. ... In recognition of the comparative immaturity and irresponsibility of juveniles, almost every State prohibits those under 18 years of age from voting, serving on juries, or marrying without parental consent." *Id.* at 569 (citations and internal quotations omitted).

Second, "juveniles are more vulnerable or susceptible to negative influences and outside pressures, including peer pressure….This is explained in part by the prevailing circumstance that juveniles have less control, or less experience with control, over their own environment….'[A]s legal minors, [juveniles] lack the freedom that adults have to extricate themselves from a criminogenic setting.'" *Id.* (internal citations omitted).

Third, "the character of a juvenile is not as well formed as that of an adult. The personality traits of juveniles are more transitory, less fixed." *Id.* at 570. Accordingly, "[f]rom a moral standpoint it would be misguided to equate the failings of a minor with those of an adult, for a greater possibility exists that a minor's character deficiencies will be

reformed." *Id.* Indeed, "[t]he relevance of youth as a mitigating factor derives from the fact that the signature qualities of youth are transient; as individuals mature, the impetuousness and recklessness that may dominate in younger years can subside." *Id.* (citation and internal quotations omitted).

Then, in *Graham v. Florida*, 560 U.S. 48 (2010), the Court extended *Roper's* reasoning to invalidate mandatory LWOP sentences for juveniles convicted of non-homicide offenses. After *Graham*, while a "[s]tate is not required to guarantee eventual freedom to a offender," it "must impose a sentence that provides some meaningful opportunity to obtain release based on demonstrated maturity and rehabilitation." *Id.* at 75 (emphasis added). In *Graham*, the Court compared LWOP terms to "death sentences," because imprisoning an offender until he dies 'alters the offender's life by a forfeiture that is irrevocable," and such a sentence "is an especially harsh punishment for a juvenile, because he will almost inevitably serve more years and a greater percentage of his life in prison than an adult offender." *Id.* at 69-70. The *Graham* Court reiterated *Roper's* three "salient" characteristics that distinguish juveniles from adults and also noted that juveniles have a reduced capacity to assist in their own defense, which puts them "at a significant disadvantage in criminal proceedings." *Id.* at 68, 78.

In *Miller*, the Supreme Court invalidated mandatory LWOP sentences for juvenile *homicide* offenders. The Court reiterated that under the Eighth Amendment "children are constitutionally different from adults for purposes of sentencing, and a system that fails to recognize those differences, "[b]y removing youth from the balance—by

subjecting a juvenile to the same life-without-parole sentence applicable to an adult…prohibit[s] a sentencing authority from assessing whether the law's harshest term of imprisonment proportionately punishes a juvenile offender." *Miller*, 567 U.S. at 471, 474. The Court went on to explain:

> Mandatory life without parole for a juvenile precludes consideration of his chronological age and its hallmark features—among them, immaturity, impetuosity, and failure to appreciate risks and consequences. It prevents taking into account the family and home environment that surrounds him—and from which he cannot usually extricate himself—no matter how brutal or dysfunctional. It neglects the circumstances of the homicide offense, including the extent of his participation in the conduct and the way familial and peer pressures may have affected him. Indeed, it ignores that he might have been charged and convicted of a lesser offense if not for incompetencies associated with youth—for example, his inability to deal with police officers or prosecutors (including on a plea agreement) or his incapacity to assist his own attorneys…. And finally, this mandatory punishment disregards the possibility of rehabilitation even when the circumstances most suggest it.

Id. at 477-78.

Finally, in *Montgomery*, the Supreme Court held that *Miller* had announced a new "substantive rule" of constitutional law, meaning that a conviction or sentence that violates the rule "is, by definition, unlawful," and that the rule must be retroactively applied in state collateral proceedings. *Montgomery v. Louisiana*, 136 S. Ct. 718, 723 (2016), *as revised* (Jan. 27, 2016). In establishing this jurisprudence, the Supreme Court explained that its decisions were based on common sense, "what any parent knows," but also on the science and social science

indicating that juveniles exhibit a "transient rashness, proclivity for risk, and inability to assess consequences," both of which lessen a child's "moral culpability" and enhance the prospect that, as the years go by and neurological development occurs, his/her "'deficiencies will be reformed.'" *Miller*, 567 U.S. at 472 (internal citations omitted). The science of adolescent brain development and psychology continues to progress, as was explained by the testimony of forensic psychologist Dr. Keith Cruise in the instant case. Transcript of Evidence Vol. 38 at 19-45. *See, also*, Declaration of forensic psychologist Dr. Gallagher, attached hereto as Appendix A (noting that the amicus briefs filed in *Miller* by the American Psychological Association and the American Medical Association "offer good descriptions of the state of research as of that point in time. Research in this area continues, and the most recent scientific findings add further support to the Court's holdings in *Roper*, *Graham*, *Miller* and *Montgomery*.").

B. Tennessee's sentencing scheme for first-degree murder offers no flexibility to account for the circumstances of youth as required by *Miller* and its progeny.

Tennessee's sentencing scheme for first-degree murder violates the Constitution because it mandates a minimum sentence of life imprisonment, even for juvenile offenders, and forecloses the sentencing court from considering the characteristics of youth, which, according to the Supreme Court, must be analyzed. For a first-degree homicide conviction, the minimum sentence for any defendant, including a juvenile, is life, with no possibility of release until after he has served 51 years in prison. Tenn. Code Ann. § 39–13–204; *Brown v. Jordan*, 563

S.W.3d 196, 202 (Tenn. 2018). This is among the most severe sentences imposed in the country for homicide.[7]

Given juveniles' distinctive capacity for change, such lengthy mandatory sentences are incompatible with the penological goal of rehabilitation. As the Supreme Court explained in *Roper*, "[f]or most teens, [risky or antisocial] behaviors are fleeting; they cease with maturity as individual identity becomes settled. Only a relatively small proportion of adolescents who experiment in risky or illegal activities developed entrenched patterns of problem behavior that persist into adulthood." 543 U.S. at 570.

In insisting that youth be treated differently than adults in sentencing, the Supreme Court has cautioned against imposing sentences that reflect a premature decision about a juvenile's incorrigibility. *See Graham*, 560 U.S. at 72. Instead, the Eighth Amendment requires that any sentence imposed on a juvenile reflect the youth's ability to change. *See id.* at 73. Juveniles "must be given the opportunity to show their crime did not reflect irreparable corruption" before being stripped of "hope for some years of life outside prison walls." *Montgomery*, 136 S. Ct. at 736-37.

The conclusion that a child must be irretrievably depraved or permanently incorrigible based on the crime alone, is untenable under the reasoning of *Roper*, *Graham*, *Miller*, and *Montgomery*. A constitutional sentence must provide some opportunity for the offender

[7] *See False Hope: How Parole Systems Fail Youth Serving Extreme Sentences*, note 1, *supra*.

to show the potential for growth and rehabilitation with time and maturity despite the severity of his youthful misconduct. Tennessee's first-degree murder sentencing scheme flies in the face of these constitutional requirements, allowing for no consideration of youth at all.

C. Imposition of a mandatory minimum fifty-one-year sentence on a juvenile is unconstitutional because it deprives him of "a meaningful opportunity to obtain release based on demonstrated maturity and rehabilitation."

The central holding of *Graham, Miller,* and *Montgomery* is that, for the reasons outlined above, the state may not deny a juvenile offender a "meaningful opportunity to obtain release based on demonstrated maturity and rehabilitation." The prospect of release after 51 years of continuous prison confinement is not meaningful to any juvenile, so Tennessee's mandatory minimum life sentence violates the constitutional prohibition against cruel and unusual punishment.

(1) A 51-year mandatory minimum life sentence is a *de facto* LWOP sentence.

Tyshon will not be eligible for release until he is at least 67 years old. This is well past average life expectancy in prison, and there is little chance that he will live that long. Effectively, he has been condemned to die in prison.

The average life expectancy for a Tennessee resident at birth is 76 years,[8] and the "healthy life expectancy at birth"[9] is 65 years. But the average juvenile who is serving a life sentence in Tennessee will not live nearly that long. *See* Dr. Michael Freeman's Declaration attached hereto as Appendix B. Dr. Freeman, an epidemiologist, analyzed prison demographic statistics furnished by TDOC and concluded that individuals serving life sentences in Tennessee have a probable life expectancy of 52 years old. An incarcerated juvenile is likely to have an even shorter life because of the adverse effects of lengthy imprisonment beginning at such a young age. The chance that a juvenile serving a life sentence in Tennessee could survive 51 years of continuous incarceration is less than 10%.

Dr. Freeman's conclusions are consistent with published studies and other authorities. For example, a study conducted by Campaign for the Fair Sentencing of Youth found that Michigan juveniles with life sentences have average life expectancy of 50.6 years, much lower than the general population.[10] One reason for this life expectancy disparity

---

[8] See U.S. Burden of Disease Collaborators, *The State of US Health, 1990-2016*, J. Am. Med. Ass'n (*JAMA*) 2018:319(14):1444, Table 3 at 1452.

[9] "Healthy life expectancy" is defined as "the number years that a person at a given age can expect to live in good health, taking into account mortality and disability." *Id.* at 1446.

[10] Deborah LaBelle, *Michigan Life Expectancy Data for Youth Serving Natural Life Sentences* 2 (2012-2015), available at http://www.lb7.uscourts.gov/documents/1712441.pdf.

may be that a large number of incarcerated defendants come from impoverished and traumatic backgrounds that diminish longevity—circumstances that are common among juvenile defendants. But the harsh conditions of prison life also contribute to this discrepancy. One study of inmate life expectancy in New York, for example, found that a "person suffers a 2 year decline of life expectancy for every year served in prison."[11]

Additionally, the United States Sentencing Commission has defined a life sentence as 470 months (or just over 39 years).[12] "This figure [of 470 months] reflects the average life expectancy of federal defendants at the time of sentencing as determined by the United States Census Bureau." *United States v. Nelson*, 491 F.3d 344, 349-50 (7th Cir. 2007). Courts too have acknowledged the reduced life expectancy of the incarcerated. *See, e.g., United States v. Taveras*, 436 F. Supp.2d 493, 500 (E.D.N.Y. 2006) (acknowledging that life expectancy within federal prison is "considerably shortened"), *vacated in part on other grounds sub nom, United States v. Pepin*, 514 F.3d 193 (2d Cir. 2008); *People v. Buffer*, 137 N.E.3d 763, 778 (Ill. 2019) (Burke, J., specially concurring)

---

[11] Evelyn J. Patterson, *The Dose-Response of Time Served in Prison on Mortality: New York State, 1989-2003*, 103 Am. J. Pub. Health 523-28 (2013). *See also* Christopher J. Mumola, Bureau of Justice Statistics, No. NCJ 216340, *Medical Causes of Death in State Prisons*, 2001-2004 (Jan. 2007) (concluding that state prisoners age 55 to 64 had death rates 56% higher than the general population).

[12] United States Sentencing Commission, *Life Sentences in the Federal System*, at 10 & n. 52 (Feb. 2015).

(noting that "the life expectancy of a minor sentenced to a lengthy prison term is … diminished"); *State v. Null*, 836 N.W.2d 41, 71 (Iowa 2013) (acknowledging that "long-term incarceration [may present] health and safety risks that tend to decrease life expectancy as compared to the general population").

Indeed, after an intensive review of the available data, undersigned counsel are not aware of any Tennessee prisoner who has survived 51 years of continuous incarceration. Given the average life expectancy of Tennessee prisoners, a life sentence with a 51 year mandatory minimum is the functional equivalent of life without parole, meaning that juveniles sentenced to life in Tennessee are effectively and almost certainly condemned to die in prison.

(2) Release after 51 years offers virtually no opportunity to meaningfully engage in free society.

The *Miller* and *Graham* "meaningful opportunity" standard invokes not only an opportunity for release, but also an opportunity for a meaningful life outside of prison. The Supreme Court intended

> more than to simply allow juveniles-turned-nonagenarians the opportunity to breath their last breaths as free people. The intent was not to eventually allow juvenile offenders the opportunity to leave prison in order to die but to live part of their lives in society.

State v. Moore, 76 N.E.3d 1127, 1137 (Ohio 2016). Assuming that a juvenile defendant could defeat the staggering odds and survive 51 years of continuous incarceration in Tennessee's prison system, and assuming that he then could obtain a release from prison in his late 60's, he

nevertheless would be deprived of any opportunity to meaningfully engage in free society for several reasons.

First, if he survives that long, his remaining life expectancy would be quite short. He would have little time to adjust to the outside world in order to pursue any kind of meaningful life.

Second, in all likelihood he would be suffering from the burdens of old age and ill health, severely limiting his physical capacity to "get on with his life."

Third, anyone reentering society after a long incarceration finds himself in a strange new world and faces enormous practical and legal obstacles, and those obstacles are greater for an elderly person. It takes time for a newly freed individual to negotiate these obstacles. In addition to dealing with a myriad of "collateral consequences" of a conviction, those reentering society from prison face challenges related to many of the basic necessities of life, such as finding employment and housing and obtaining access to healthcare and other public benefits.[13] These obstacles to meaningful reentry are compounded in the case of an elderly person released from prison after spending 51 years, his entire adult life, in confinement.

---

[13] *See,* e.g., Anthony C. Thompson, *Navigating the Hidden Obstacles to Ex-Offender Reentry*, 45 Boston L. Rev. 255, 272-73 (2004). For an inventory of legally imposed collateral consequences of conviction, see The Counsel of State Governments Justice Center, *National Inventory of the Collateral Consequences of Conviction*, available at https://niccc.csgjusticecenter.org.

Fourth, spending one's entire adult life subject to the institutionalizing effects of the highly structured and authoritarian prison environment makes it psychologically difficult to adjust to the pressures and demands of living free in society, especially at such an old age. *See* Craig Haney, *The Psychological Impact of Incarceration: Implications for Post-Prison Adjustment*, available at https://aspe.hhs.gov/basic-report/psychological-impact-incarceration-implications-post-prison-adjustment (U.S. Dep't of Health & Human Servs., 2001). Professor Haney explains how inmates psychologically adapt to the harsh conditions of prison life, in ways that enable them to survive in prison but impair their capacity to adjust to the free world upon release. It stands to reason that the adverse psychological impact of incarceration is more pronounced (i) when the incarceration begins at a young age, especially if it begins while the prisoner is a juvenile, and (ii) when the incarceration is for a longer period of time. *Id.* at 5.

Finally, it is well known that "persons who return to the free world lacking a network of close, personal contacts with people who know them well" have an especially difficult time adjusting. As Professor Haney points out, "Eventually…when severely institutionalized persons confront complicated problems or conflicts, especially in the form of unexpected events that cannot be planned for in advance, the myriad of challenges that the non-institutionalized confront in their everyday lives outside the institution may become overwhelming." *Id.* at 8-9. A person who has been continuously incarcerated for 51 years is not likely to have any remaining connections to family or community upon release. After

spending half a century in prison, he will be "lost" in a foreign, complex, and stressful environment without a stable social network for support.

By withholding release eligibility until the twilight of a juvenile offender's life, requiring him to spend his entire adult life undergoing the institutionalizing and stigmatizing effects of incarceration, a 51-year mandatory minimum sentence "gives no chance for fulfillment outside prison walls, no chance for reconciliation with society, no hope." *Graham*, 560 U.S. at 79.

(3) Imposing a 51-year mandatory minimum forswears altogether the rehabilitative ideal.

A fundamental scientific principle underlying the constitutional premise that "juveniles are different" is that—because a juvenile's mental traits and vulnerabilities are merely "transitory"—juveniles have great potential to rehabilitate as their minds and bodies mature. *Miller*, 567 U.S. at 473. Fifty-one years of detention, however, extends far beyond the period within which a juvenile will mature and rehabilitate. Forcing a juvenile offender to wait beyond his life expectancy, until he is a geriatric with virtually no prospect for a meaningful and productive life in the free world, defeats the entire purpose of the requirement that juvenile offenders be given "a meaningful opportunity for release based upon demonstrated maturity and rehabilitation." Such a sentence "means a denial of hope" and "share[s]. . . characteristics with death sentences" because it denies any chance for a maturing youthful offender to work toward a brighter future; despite "good behavior and character improvment," he will remain in prison for the rest of his days." *Graham*,

516 U.S. at 69-70 (citation and internal quotations omitted). In a word, this kind of sentence "forswears altogether the rehabilitative ideal." *Miller*, 567 U.S. at 473 (citing *Graham*, 560 U.S. at 74).

D. Many other jurisdictions hold that similarly lengthy mandatory minimum sentences for juveniles violate the Eighth Amendment under *Miller* and *Montgomery.*

A majority of state courts have employed the principles embodied in *Roper*, *Miller, Graham,* and *Montgomery* to invalidate minimum mandatory life sentences, constituting a lengthy minimum term of years, because they deprive juvenile offenders of a "meaningful opportunity" for release.[14] In 2013, Iowa became one of the first jurisdictions to hold that a juvenile sentenced to a *de facto* LWOP sentence is constitutionally entitled to *Miller*-type protections affording a "meaningful opportunity for release based on demonstrated maturity and rehabilitation." *Null,* 836 N.W.2d at 63 (quoting *Graham*, 560 U.S. at 75). The defendant in *Null* received a mandatory minimum aggregate sentence of 52.5 years for second-degree murder and first-degree robbery for an offense that occurred when he was sixteen years old. *Id.* at 45. Under the Iowa

[14] In addition, several federal courts have applied these principles to mandatory sentences expressed as a term of years. The Seventh Circuit has opined that courts should apply a "children are different" approach to sentencing to both traditionally defined life sentences as well as *de facto* life sentences. *McKinley v. Butler*, 809 F.3d 908, 914 (7th Cir. 2016) (Posner, J.). The Ninth Circuit has adopted similar reasoning, holding that a lengthy term of years sentence violates *Miller* and *Graham's* requirement that juveniles be given a meaningful opportunity to re-enter society. *Moore v. Biter*, 725 F.3d 1184, 1191–92 (9th Cir. 2013).

sentencing scheme, he would not be eligible for parole until was sixty-nine. *Id.* The court, in a thorough and well-reasoned discussion, applied *Graham* and *Miller* to hold that this kind of punishment for a juvenile offender violates the Eighth Amendment and the Iowa constitution. *Id.* at 60-77.

The *Null* court based its decision in great part on the scientific evidence discussed in *Roper*, reasoning that juveniles have not fully developed cognitive structures for risk evaluation, self-management, and impulse control. *Id.* at 55. The court noted that juveniles are also much more prone to peer influence, and their development runs part and parcel with experimentation with "risky, illegal, or dangerous activities." *Id.* And while the adolescent brain can tend toward criminal behavior, it is also highly transformable. As the young person develops into an adult, science confirms that the impulse control and risk assessment issues fade away. *See id.* There are no strong penological justifications for lengthy juvenile sentences, because juveniles have the ability, and indeed proclivity, for change in a positive direction.

The *Null* court, and many others, have construed *Graham* and *Miller* to require a juvenile sentence to provide more time outside of prison than a few years of freedom at the end of one's life. The *Null* court declared that "[t]he prospect of geriatric release, if one is to be afforded the opportunity for release at all, does not provide a 'meaningful opportunity' to demonstrate the 'maturity and rehabilitation' required to obtain release and reenter society as required by *Graham*." *Id.* at 71. Similarly, the Connecticut Supreme Court held that under *Miller*, a mandatory minimum 50-year sentence for a juvenile offender was

unconstitutional, because it did not allow for a "meaningful opportunity" for release, which requires a chance to engage with civic society, to be employed, and to have a family. *Casiano v. Comm'r of Correction*, 115 A.3d 1037, 1046-47 (Conn. 2015). Analysis of what constitutes a "meaningful opportunity" must also take into consideration that a juvenile offender, released from prison at the end of his/her life will also have a diminished quality of life, having an increased risk for age-related health disorders, such as heart disease, hypertension, stroke, asthma, cancer, and arthritis. *Id.* According to the Supreme Courts of Iowa and Connecticut, such a degraded experience is not meaningful, under the mandates of *Graham* and *Miller*.

A groundswell of recent decisions confirms the principle that mandatory long-term sentences for juveniles convicted of homicide do not pass constitutional muster. *See State v. Davilla*, 462 P. 3d 748, 752 (Or. Ct. App. 2020) (50 year sentence required modification er); *Buffer*, 137 N.E.3d at 774 (50 year sentence); *Davis v. State*, 415 P.3d 666, 676 (Wyo. 2018) (homicide sentence of approximately 45 years before parole eligibility); *Carter v. State*, 192 A.3d 695, 702 (Md. 2018), *reconsideration denied* (Oct. 4, 2018) (100 year sentence with eligibility for parole in 50 years); *State ex rel. Carr v. Wallace*, 527 S.W.3d 55, 60–62 (Mo. 2017) (50 years until eligibility for parole); *State v. Zuber*, 152 A.3d 197, 216 (N.J. 2017) (55 year sentence); *California v. Ramirez*, 2017 WL 5824286 (Cal. Ct. App. Nov. 29, 2017) (40 year sentence); *California v. Fernandez*, 2015 WL 1283486 (Cal. Ct. Ap. Mar. 18, 2015) (50 year sentence); *Washington v. Ronquillo*, 361 P.3d 779, 789 (Wash. Ct. App. 2015) (51.3 year sentence); *Bear Cloud v. State*, 334 P.3d 132 (Wyo. 2014) (homicide

sentence of 45 years prior to parole); *Adams v. Florida*, 188 So.3d 849 (Fla. St. App. 2012) (50 year sentence).

These cases reveal a pattern. For juveniles, mandatory sentences with no eligibility for parole until after 50 years offend the teachings of both *Graham* and *Miller*. Recently, the Maryland Supreme Court noted that "[m]any courts have concluded that a sentence of a term of years that precludes parole consideration for a half century or more is equivalent to a sentence of life without parole." *Carter*, 92 A.3d at 729; *see also*, *White v. Premo*, 443 P.3d 597, 605 (Or. 2019), *cert. dismissed sub nom. Kelly v. White*, 140 S. Ct. 993 (2020) ("We know of no state high court that has held that a sentence in excess of 50 years for a single homicide provides a juvenile with a meaningful opportunity for release."). The Maryland Supreme Court noted that the fifty-year benchmark likely originated from *Graham's* description, as constitutionally problematic, of a defendant not being eligible for release "even if he spends the *next half century* attempting to atone for his crimes and learn from his mistakes." *Carter,* 192 A.3d at 728-29 (quoting *Graham*, 560 U.S. at 79 (emphasis added)). The "meaningful opportunity" standard "means a sentence with parole eligibility significantly short of the 50-year mark." *Id.* at 735. The undeniable trend in the case law confirms that a term-of-years sentence longer than 50 years does not comply with the strictures of *Graham* and *Miller*. Based on the reasoning of these decisions, Tennessee's mandatory scheme does not give vulnerable and cognitively underdeveloped juvenile offenders any hope for a rehabilitated and productive life in civic society beyond the prison walls.

E. Evidencing our nation's evolving standard of decency, a large number of states have enacted new sentencing schemes in response to *Graham*, *Miller* and *Montgomery*, leaving Tennessee as an outlier.

In addition to the many state court decisions voiding lengthy prison terms for children, in the wake of *Graham* and *Miller*, twenty-five states have adopted legislation limiting juvenile homicide sentences and providing within the regulatory scheme a meaningful opportunity for the inmate to demonstrate rehabilitation and maturity. Some states have capped sentences for juvenile homicide while other approaches redefine parole eligibility for juveniles previously sentenced to LWOP. *See* Ariz. Rev. Stat. Ann. §§ 13-751, 13-752 (juvenile sentences for homicide limited to 25 to 35 years); Ark. Code Ann. § 16-93-621(a)(2)(a) (juvenile homicide offenders eligible for parole after 25 years); Cal. Penal Code § 3051 (juveniles sentenced to LWOP entitled to a parole hearing no later than twenty-five years of incarceration); Colo. Rev. Stat. Ann. § 18-1.3-401(4)(c)(I)(A) & (B) (juvenile offenders sentenced to LWOP for first degree murder entitled to a re-sentencing hearing and a sentence between 30 to 50 years); Conn. Gen. Stat. Ann. § 54-125a(f)(1) (juvenile offenders sentenced to over 50 years eligible for parole after 30 years, and juvenile offenders sentenced to between 10 and 50 years eligible for parole after the greater of 12 years or 60% of the sentence); Del. Code Ann. tit. 11, § 4204A(d)(2) (juvenile offenders convicted of first-degree murder eligible for resentencing after 30 years); D.C. Code Ann. § 24-403.03(a) (juvenile offenders eligible for sentence reduction after 20 years]); Fla. Stat. Ann. § 921.1402(2)(b) (juvenile offenders sentenced to over 25 years entitled to review of sentence after 25 years); Haw. Rev.

Stat. § 706-656(1) (all juvenile offenders entitled to life with the possibility of parole on a date to be established through a rehabilitation plan); Ky. Rev. Stat. Ann. § 640.040 (statute pre-dating *Graham* and *Miller* provides that youthful offenders convicted of a capital crime are eligible for parole after 25 years); La. Code Crim. Proc. Ann. Art 878.1 (generally, juveniles convicted of homicide eligible for parole after serving 25 years unless a special hearing is conducted determining that LWOP is appropriate); Mass. Gen. Laws Ann. ch. 279, § 24 (juveniles convicted of first-degree murder are eligible for parole in 20 or 30 years, as determined by the court); Mich. Comp. Laws Ann. §§ 769.25 (juvenile homicide offenders limited to a sentence of 25 to 40 years); Mo. Ann. Stat. § 558.047(1) (juvenile offenders sentenced to LWOP eligible for review of sentence after 25 years); Neb. Rev. Stat. Ann. § 28-105.02 (juvenile LWOP sentences become eligible for parole after 40 years); Nev. Rev. Stat. Ann. § 213.12135) (juvenile offenders for a homicide of [only one victim] eligible for parole after 20 years); N.C. Gen. Stat. Ann. §15A-1340.19A (juvenile LWOP sentences allow parole eligibility after 25 years); N.J. Stat. Ann. §2C:11-3 (juveniles convicted of first degree murder eligible for parole within 30 years); N.D. Cent. Code Ann. § 12.1-32-13.1 (juvenile offenders eligible for sentence reduction after 20 years); Or. Rev. Stat. Ann. §163.115 (juveniles sentenced to a life sentence for homicide eligible for parole in twenty-five years); Tex. Govt. Code Ann. § 508.145 (all juvenile offenders serving a life sentence are eligible for parole in 40 years); Utah Code Ann. § 76-3-206 (juvenile homicide sentence limited to 25 years); W.Va. Code § 61-11-23(b) (juvenile offenders eligible for parole after 15 years); Wyo. Stat. Ann. § 6-10-301(c)

(juvenile offenders sentenced to life eligible for parole after 25 years); Wash. Rev. Code § 9.94A.730(1) (juvenile offenders eligible for release after 20 years, except for those serving sentences for aggravated first degree murder or certain sex offenses).

The legislative history rests on the consensus that children are different and that a lengthy sentence should not be imposed on a child in the same way as upon adults. *See, e.g.*, Ark. Code Revision Comm'n, Notes on Ark. Code Ann. § 16-93-621(a)(2)(a) ("The General Assembly acknowledges and recognizes that minors are constitutionally different from adults and that these differences must be taken into account when minors are sentenced for adult crimes."); Statutory Notes for Haw. Rev. Stat. § 706-656(1) ("The legislature acknowledges and recognizes that children are constitutionally different from adults and that these differences must be taken into account when children are sentenced for adult crimes."). *See also*, Conf. Comm. Rpt. La. Code Crim. Proc. Ann. Art 878.1 (June 6, 2017) (noting that a change in Louisiana's sentencing law was necessary in response to *Miller*, *Graham*, and *Montgomery*).

These recent sentencing reforms confirm a changed community standard recognizing that juvenile sentences of more than 50 years without parole eligibility are not consistent with the Eighth Amendment. *See Carter*, 192 A.3d at 729 n.43 (noting an emerging legislative consensus that a fifty plus year sentence for juveniles offends the constitution in relation to *Graham* and/or *Miller*). Tennessee's mandatory sentencing scheme, which treats juvenile defendants exactly the same as adults and forecloses parole until after 51 years, is plainly an outlier among the states. Tennessee's mandatory sentencing approach is out-of-

step with prevailing community standards for what is appropriate punishment for juvenile offenders, who do not carry the same attributes of culpability as adult offenders.

F. Tennessee's Constitution, Art. I, §§ 13, 16 and 32, provides greater protection than the federal constitution against the unnecessary rigor and inhumanity of imposing a 51-year mandatory minimum prison term on a juvenile.

The Tennessee Constitution creates additional protections against excessive punishment and, viewed independently from the federal Constitution, should also be construed to invalidate a mandatory minimum 51-year sentence for a juvenile.

This Court has long recognized that, "as the final arbiter of the Tennessee Constitution, [it] is always free to expand the minimum level of protection mandated by the federal constitution." *State v. Ferguson*, 2 S.W.3d 912, 916 (Tenn. 1999) (citation and internal quotations omitted). *See also, Miller v. State*, 584 S.W.2d 758, 760 (Tenn. 1979), *overruled by State v. Pruitt,* 510 S.W. 3d 398, 416 (Tenn. 2016) ("[A]s to Tennessee's Constitution, we sit as a court of last resort, subject solely to the qualification that we may not impinge upon the minimum level of protection established by the Supreme Court interpretation of the federal constitutional guarantees. But state supreme courts, interpreting state constitutional provisions, may impose higher standards and stronger protections than those set by the federal constitution.") (emphasis added).

Following these principles, Art. I, § 16, Tennessee's Cruel and Unusual Punishments Clause, should be independently construed to

protect juveniles against mandatory minimum 51-year sentences. The Tennessee Constitution's special concern about excessive punishment is further set forth in Art. I, § 13, which provides "[t]hat no person arrested and confined in jail shall be treated with unnecessary rigor"; and in Art. I, 32, which provides "[t]hat the erection of safe prisons, the inspection of prisons, and the humane treatment of prisoners, shall be provided for." Given these additional provisions against excessive punishments, strong grounds exist for independently applying the Tennessee Constitution to protect against the kind of mandatory life sentence that was imposed on Appellant Tyshon Booker.

VI. CONCLUSION

For the foregoing reasons, *amici curiae* Amos Brown, Charles Lowe-Kelley, the Tennessee Association of Criminal Defense Lawyers, and the National Association of Criminal Defense Lawyers urge the Court to grant Tyshon's application for permission to appeal.

Respectfully submitted,

s/ Sarah B. Miller
David R. Esquivel (#021459)
Jeff H. Gibson (#026321)
Sarah B. Miller (#033441)
Angela L. Bergman (#031981)
BASS, BERRY & SIMS PLC
150 Third Avenue South, Suite 2800
Nashville, TN 37201
Phone: 615-742-6200
Email: desquivel@bassberry.com
jgibson@bassberry.com

smiller@bassberry.com
abergman@bassberry.com

Counsel for Charles E. Lowe-Kelley

s/ Bradley A. MacLean
Bradley A. MacLean (#009562)
Law Office of Bradley A. MacLean
1702 Villa Place
Nashville, TN 37212

Counsel for Charles Lowe-Kelley and Amos Brown

s/ Lucille A. Jewel
Lucille A. Jewel (#032666)
1505 W. Cumberland Avenue
Knoxville, TN 37996-1810

-and-

Stephen Ross Johnson (#022140)
Sixth Circuit Vice-Chair
Amicus Committee of the
National Association of Criminal
Defense Lawyers

Ritchie, Dillard, Davies, & Johnson
606 W. Main Street, Suite 300
Knoxville, TN 37902

Counsel for National Association of Criminal Defense Attorneys

s/ Jonathan D. Cooper
Jonathan D. Cooper
President
Tennessee Association of Criminal
Defense Lawyers
530 Church Street, Suite 300
Nashville, TN 37219
(615) 329-1338

Counsel for Tennessee Association of Criminal Defense Attorneys

CERTIFICATE OF ELECTRONIC FILING COMPLIANCE

Under Tennessee Supreme Court Rule 46, § 3.02, I hereby certify that this brief contains 7,496 words as calculated by Microsoft Word, and it was prepared using 14-point Century font with 1.5x line spacing.

s/ Sarah B. Miller
Sarah B. Miller

CERTIFICATE OF SERVICE

I certify that a true and exact copy of the foregoing motion was served electronically or mailed via U.S. first class mail, postage prepaid, this 9th day of June, 2020, to:

Nicholas Spangler
Attorney General's Office
Criminal Justice Division
PO Box 20207
Nashville, TN 37202

Jonathan Harwell
Assistant Public Defender
1101 Liberty Street
Knoxville, TN 37919

s/ Sarah B. Miller

APPENDIX A

IN THE CIRCUIT COURT FOR MCMINN COUNTY, TENNESSEE
AT ATHENS

AMOS BROWN (TDOC #287845),	)	
	)	
Petitioner,	)	
	)	
vs.	)	**No. 4-CR-64**
	)	
STATE OF TENNESSEE	)	
	)	
Respondent.	)	
	)	

PETITIONER'S NOTICE OF FILING OF
DECLARATION OF DR. JULIE A. GALLAGHER

Plaintiff Amos Brown gives notice that he is filing the attached Declaration of Dr. Julie A. Gallagher, a forensic psychologist, in support of his claims in this case. Dr. Gallagher's Declaration regards her expert opinion concerning adolescent brain development that justifies treating juvenile offenders differently from adult offenders for sentencing purposes under the Cruel and Unusual Punishments Clauses and other Clauses of the United States and Tennessee Constitutions.

Respectfully submitted,

BRADLEY A. MACLEAN, BPR #009562
Counsel for Plaintiff
1702 Villa Place
Nashville, TN 37212
Phone: 615-943-8716
Email: brad.maclean9@gmail.com

CERTIFICATE OF SERVICE

I hereby certify that on this 1st day of May, 2018, a true copy of the foregoing was served by first class mail and by email on:

Stephen D. Crump, Esq.
District Attorney General
93 Ocoee Street, Suite 2
Cleveland, TN 37311

Courtney N. Orr, Esq.
Assistant Attorney General
Criminal Appeals Division
P.O. Box 20207
Nashville, TN 37202-3491

Stephen Ross Johnson, Esq.
Ritchie, Dillard, Davies & Johnson, PC
606 W. Main Street, Suite 300
Knoxville, TN 37902

Bradley A. MacLean

IN THE CIRCUIT COURT FOR MCMINN COUNTY, TENNESSEE
AT ATHENS

AMOS BROWN (TDOC #287845),	)	
Petitioner,	)	
	)	**No.: 4-CR-64**
v.	)	
STATE OF TENNESSEE,	)	
Respondent.	)	

DECLARATION OF DR. JULIE A. GALLAGHER

Pursuant to Tenn. R. Evid. 703 and Tenn. R. Civ. Pro. 72, Dr. Julie A. Gallagher declares as follows:

1. I have been retained by Bradley A. MacLean, counsel for Petitioner Amos Brown, as an independent expert in the above-captioned matter. I make this Declaration based on my own personal knowledge and, if called as a witness, I could and would testify competently to the truth of the matters set forth herein.

2. A true and correct copy of my expert report, dated February 1, 2018, along with my current CV, is attached hereto. The information in my report and CV is true and correct to the best of my knowledge, information and belief.

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

Executed on 5 March, 2018.

Julie A. Gallagher, Psy.D. ABPP
Board Certified in Forensic Psychology

JULIE A. GALLAGHER, PSY.D. ABPP
CLINICAL AND FORENSIC PSYCHOLOGIST
BOARD CERTIFIED IN FORENSIC PSYCHOLOGY

2200 21st Avenue South, Suite 401
Nashville, Tennessee 37212
www.DrJulieGallagher.com

Phone (615) 491-3229
Fax (615) 750-5796
Dr.Julie.Gallagher@gmail.com

February 1, 2018

Brad MacLean
1702 Villa Place
Nashville, TN 37212

Re: Amos Brown case

Dear Mr. MacLean,

You asked me to summarize recent research on adolescent brain development. In a series of decisions beginning with *Roper v. Simmons*, 543 U. S. 551 (2005), the United States Supreme Court has considered research on adolescent brain development in making determinations regarding the proportionality of punishments as applied to juvenile offenders (*Graham v. Florida*, 560 U. S. 48 (2010); *Miller v. Alabama*, 567 U. S. 460 (2012); *Montgomery v. Louisiana*, 136 S. Ct. 718 (2016)). This document is intended to summarize the basic research and expand upon the state of the research since the writing of the last in that series of opinions, which were heard in 2016.

For your reference, I have attached relevant amicus briefs filed in *Miller,* describing the science of adolescent brain development upon which the Court relied in reaching its decision. These briefs offer good descriptions of the state of research as of that point in time. Research in this area continues, and the most recent scientific findings add further support to the Court's holdings in *Roper*, *Graham*, *Miller* and *Montgomery*.

Qualifications

I am a clinical and forensic psychologist, board certified in forensic psychology by the American Board of Professional Psychology. My expertise is primarily in criminal forensic psychology, with specialization in juvenile justice work. Please refer to my attached CV for more information on my training and experience.

The Phases of Adolescent Brain Development

There are three overlapping phases of brain development that occur during adolescence. During all three phases, the brain changes through a process of synaptic pruning and myelination of particular brain regions. Pruning is the removal of unneeded connections, which strengthens and makes other connections more efficient, just as pruning a tree causes its main branches to grow stronger. Myelination is the insulation of those connections, resulting in greater white matter in the brain. This allows brain cells to transmit information more rapidly along those connections.

The primary brain regions affected during adolescence are the limbic system, which regulates emotional arousal, and the prefrontal cortex, which regulates self-control and rational decision-making. Notably, the changes in these regions occur at different times, with the limbic system maturing well before the prefrontal cortex, resulting in a significant maturational imbalance. This imbalance has a profound effect on thinking and behavior. In fact, research has found that these changes follow an identifiable pattern that is consistent with the behavioral changes that occur during adolescence. Only when these brain regions complete development and become fully interconnected is

development complete. Research has consistently shown that these changes are not complete until the early 20s.

The first phase of adolescent brain development is triggered by the hormonal changes accompanying puberty (Steinberg, 2017). Puberty, which typically starts around age 12, remodels the brain and makes it more plastic, or moldable (Selemon, 2013). The hormones released during puberty have a profound effect on the limbic system, which is deep in the center of the brain. This leads to changes that alter the way the brain, and especially the limbic system, responds to the neurotransmitters dopamine and serotonin. Neurotransmitters are chemicals that transmit nerve impulses from one brain cell to the next. Dopamine affects how the brain responds to rewards and serotonin plays an important role in mood regulation. As a result, the adolescent brain becomes much more easily emotionally aroused and more sensitive to rewards, including the social rewards of approval by peers. It also becomes less sensitive to negative outcomes, as rewards become more salient. This is why adolescents typically seek out intense and exciting experiences and are greatly influenced by the presence of peers, while discounting possible negative consequences. So, during this phase, the limbic system is responsible for the dramatic ups and downs of emotion experienced by adolescents, their greater sensitivity to the influence of their peers and their greater sensation-seeking (Braams et al 2015; Bjork et al 2012; Galvan 2013; Luciana & Collins 2012). Recent research has revealed that these findings hold true for adolescents around the world and across various species of mammals, including mice, rats and other primates (Steinberg, 2014; Steinberg et al., 2017).

The second phase begins during preadolescence and occurs gradually, ending around age 16. During this phase, the prefrontal cortex, which is responsible for self-regulation, becomes better organized. This occurs through a process of pruning of unneeded connections between neurons in the prefrontal cortex, and myelination, or increasing insulation around connections, which strengthens those connections (Spear, 2013). In other words, the pathways in the prefrontal cortex that are most needed for decision-making, problem-solving and planning ahead ("executive functions") become clearer and stronger (Dwyer et al 2014; Ladouceur et al 2012; Smith et al 2014).

These so-called "executive functions," have also been described as a "braking system," in the brain (Steinberg, 2014). During this stage, despite improvements in the organization of the prefrontal cortex, this "braking system" is not yet completely online. This is because the connections between the limbic system and the prefrontal cortex are not yet fully in place. As a result, the prefrontal cortex is still very vulnerable to interference. It can be easily derailed by emotional arousal and fatigue. Thus, adolescents in this age group have more difficulty than older adolescents demonstrating self-control when they are upset, excited or tired (Figner & Weber, 2011; van Duijvenvoorde et al., 2010). Under such circumstances they are more likely to engage in risky behavior and make decisions without considering the consequences of their actions.

The third phase of brain development, which takes place in late adolescence, helps that "braking system" to become more stable and more reliable. This is the result of the development of increased interconnections between the prefrontal cortex and the limbic system (Hwang et al., 2013; Sherman et al., 2014; Dosenbach et al., 2013). This increase in structural and functional connectivity allows multiple brain systems to work together

much more efficiently, with different brain systems activating together during particular tasks (Dosenbach et al., 2010; Ernst et al., 2015; Sherman et al., 2014). As a result, the executive functions of the prefrontal cortex are no longer as vulnerable to the emotional arousal of the limbic system. Instead, these areas work together to modulate emotion, assess risks and engage in decision-making. This allows adolescents to gain better control of their impulses, think about the long-term consequences of their decisions and better resist peer pressure.

Diffusion tensor imaging has allowed us to visualize these weak connections that become stronger as adolescence progresses. These structural imaging studies have revealed immature connections within the front-parietal-striatal brain system that affect executive functioning (Olesen, Nagy, Westerberg & Klingberg, 2003; Schmithorst & Yuan, 2010; Vincent et al., 2008). As these connections strengthen over the course of adolescence, greater impulse control is seen (Liston et al., 2006). This results in significant improvements in self-regulation. These have been demonstrated in various studies using functional MRI. Using functional MRI, a number of studies have also shown greater neural activity during adolescence in parts of the brain that play an important role in the processing of emotional and social information and in the prediction of reward and punishment, the ventral striatum and the ventromedial prefrontal cortex (Galvan et al., 2006; Hare et al, 2008; Luciana & Collins, 2012). In addition, functional MRI has revealed changes in patterns of activation during tasks that require working memory, planning and response inhibition (Casey, 2015; Luna, Padmanabhan & O'hearn, 2010; Stevens, Kiehl, Pearlson & Calhoun, 2007). These processes are important for impulse control and planning ahead. Research has consistently revealed that this process

is not complete until the early 20s (Casey et al 2005; Hooper et al 2004; Paus, 2009; Dosenbach et al 2010).

Notably, research has revealed that this first phase, triggered by puberty, is occurring earlier due to a range of environmental influences (Steinberg, 2016). Because the second phase still occurs at the same time, adolescents are left with a longer period of time during which they seek out risks but do not yet have the capacity for self-control necessary to manage those risks (Steinberg, 2014). Thus, they are pressing on the accelerator longer despite the fact that their braking system is not yet online.

The Behavioral Science

In 2013, a panel organized by the National Academy of Sciences concluded a review of the research on adolescent development and its impact on juvenile justice (Bonnie et al., 2013). They came to three primary conclusions. The first was that, in emotionally charged situations, adolescents do not have a mature capacity for self-regulation compared to adults (Somerville, Fani, and McClure-Tone, 2011). The second was that, relative to adults, adolescents are much more vulnerable to peer influence and immediate incentives (Gardner and Steinberg, 2005; Figner et al., 2009). The third was that adolescents lack time perspective, impairing their ability to make judgments and decisions that require future orientation (Steinberg, 2009).

A substantial body of research has demonstrated that from mid to late adolescence there is a peak in risk-taking behavior. A similar peak is seen in involvement in violent and nonviolent crime. This peak occurs because the development of the limbic system outpaces the development of the prefrontal cortex (Steinberg, 2017). In other words,

adolescents have a strong desire to seek out risks but lack the judgment and decision-making abilities necessary to keep them safe. "In essence, the brain changes in ways that may provoke individuals to seek novelty, reward and stimulation several years before the complete maturation of the brain systems that regulate judgment, decision making and impulse control (Galvan 2010: Padmanabhan et al 2011; Van Leijenhorst et al 2010)." (Steinberg, 2017).

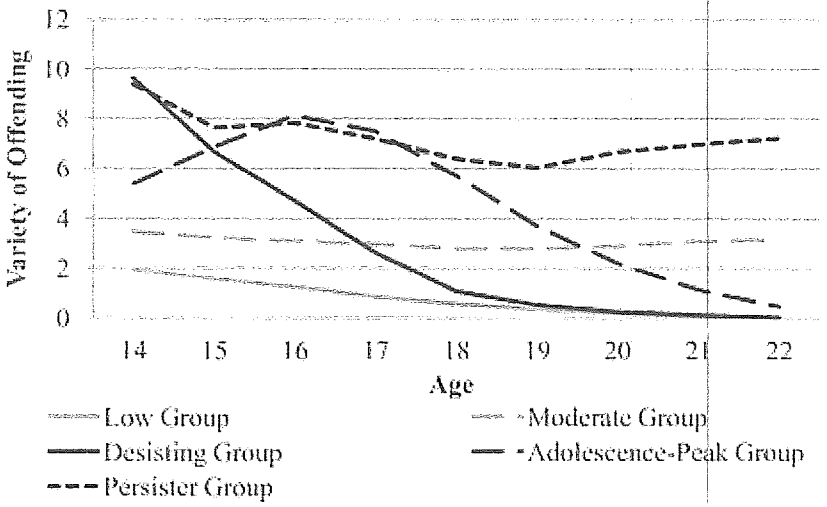
As Steinberg (2017) explains in his recent review of the literature, "The heightened responsiveness of this socioemotional incentive processing system is thought to overwhelm, or at the very least, tax, the capacities of the self-regulatory system, compromising adolescents' abilities to temper strong positive and negative emotions and inclining them toward sensation seeking, risk-taking, and impulsive antisocial acts (Casey et al., 2010; Shulman et al., 2016)." This is also reviewed in Scott and Steinberg's book Rethinking Juvenile Justice (2008), where they state that most juvenile crimes are impulsive acts that are committed without full consideration of their long-term consequences.

In the decades since the *Roper* decision, research in this area has expanded extensively. In his recent review of the literature, Laurence Steinberg explained that numerous studies have shown that,

> Compared to adults, adolescents are more impulsive (Steinberg et al., 2008), less likely to consider the future consequences of their actions (Steinberg et al., 2009), more likely to engage in sensation seeking (Steinberg et al., 2008), and more likely to attend to the potential rewards of a risky decision than to the potential costs (Cauffman et al., 2010). Other studies have provided support for the contention that

adolescents are indeed more vulnerable to coercive pressure than adults (Steinberg & Monahan, 2007); that the presence of peers makes adolescents more sensitive to rewards (Chein et al., 2011; Silva et al., 2016; Weigard et al., 2014); are especially attentive to immediate rewards; and that the presence of peers increases risky decision-making among adolescents but not older individuals (Chein et al., 2011; Gardner & Steinberg, 2005; Smith, Chein & Steinberg, 2014).

It has been well-established in the research literature that most juveniles engage in antisocial behavior to some degree during adolescence. It has also been well-established that most do not continue that behavior into adulthood (Moffitt, 2015). This was confirmed by the Pathways to Desistance study, which followed 1,354 serious juvenile offenders ages 14–18 for seven years, making it the largest study of recidivism in juvenile offenders to date. As can be seen below, they found that only approximately 10% of serious offenders (the "persister" group), continued to report high levels of antisocial acts (Monahan et al., 2009).



That study also revealed that longer stays in correctional institutions did not reduce recidivism in juvenile offenders (Mulvey, 2011). Confirming the transient nature of juvenile offending, they found that "The most important conclusion of the study is that even adolescents who have committed serious offenses are not necessarily on track for adult criminal careers" (Mulvey, 2011). In a recent presentation to the Joint Ad-hoc Tennessee Blue Ribbon Task Force on Juvenile Justice in Tennessee, Professor Edward P. Mulvey, Ph.D., the lead researcher on that study presented it and other recent research and concluded that there is "No convincing evidence that confinement of juvenile offenders beyond a minimum amount required to provide intense services reduces [the] likelihood of subsequent offending."

Conclusion

In the years since the Court decided *Graham, Miller* and *Montgomery*, the scientific evidence has grown stronger. That research has continued to confirm what parents have always known, namely that adolescents are different from adults in important ways. They are more emotional, more impulsive, more vulnerable to peer influence, take greater risks without considering the consequences, and are not as capable of planning and making decisions.

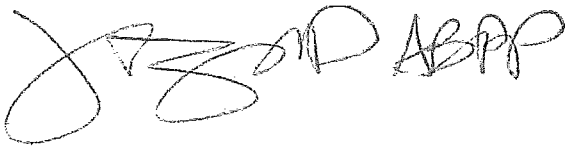
The maturation of the adolescent brain follows a specific and predictable pattern that is consistent with the patterns of change observed in adolescent behavior. In recent years we have gained the ability to visualize the changes that occur between childhood and adulthood in the developing adolescent brain as structures change and different parts of the brain respond differently to different hormones and neurotransmitters. We can

now watch these structures interconnect and begin to work together as the brain matures. Recent studies have even demonstrated that these changes are true across cultures and across species.

One of those patterns of behavior change that has been consistently observed is that the risky, impulsive and sometimes antisocial behavior demonstrated by adolescents does not continue into adulthood in the vast majority of adolescent offenders. It should thus come as no surprise that research has also found that incarceration beyond the time necessary to provide intensive services does not reduce offending. It seems that they grow out of it regardless.

As a group, adolescents' immaturity is a transient state and not an enduring part of their character. As Justice Kennedy wrote in *Roper*, "The reality that juveniles still struggle to define their identity means it is less supportable to conclude that even a heinous crime committed by a juvenile is evidence of irretrievably depraved character." Since *Roper*, the research on adolescent brain development, adolescent behavior patterns and, specifically, desistance both confirms and strengthens this assertion.

Sincerely,



Julie A. Gallagher, Psy.D. ABPP
Board Certified in Forensic Psychology

REFERENCES

Cases

Graham v. Florida, 560 U.S. 48 (2010)

Miller v. Alabama, 567 U.S. 460 (2012)

Montgomery v. Louisiana, 136 S. Ct. 718 (2016)

Roper v. Simmons, 543 U.S. 551 (2005)

Briefs

Brief for the American Medical Association and the American Academy of Child and Adolescent Psychiatry as *Amici Curiae* in Support of Neither Party, *Miller v. Alabama*, 567 US 460 (2012), (Nos. 10-9646 & 10-9647).

Brief for the American Psychological Association et al. as *Amici Curiae* in Support of Petitioners, *Miller v. Alabama*, 567 US 460 (2012), (Nos. 10-9646 & 10-9647).

Brief of *Amici Curiae* J. Lawrence Aber et al. in Support of Petitioners, *Miller v. Alabama*, 567 US 460 (2012), (Nos. 10-9646 & 10-9647).

Research Articles and Books

Aarthi Padmanabhan et al., *Developmental changes in brain function underlying the influence of reward processing on inhibitory control*, 1 DEVELOPMENTAL COGNITIVE NEUROSCIENCE 517–529 (2011).

Adriana Galván, *Adolescent development of the reward system*, FRONTIERS IN HUMAN NEUROSCIENCE (2010).

Adriana Galván, *The Teenage Brain*, 22 CURRENT DIRECTIONS IN PSYCHOLOGICAL SCIENCE 88–93 (2013).

Adriana Galván et al., *Earlier Development of the Accumbens Relative to Orbitofrontal Cortex Might Underlie Risk-Taking Behavior in Adolescents*, 26 JOURNAL OF NEUROSCIENCE 6885–6892 (2006).

Alexander Weigard et al., *Effects of anonymous peer observation on adolescents preference for immediate rewards*, 17 DEVELOPMENTAL SCIENCE 71–78 (2013).

Anna C. K. Van Duijvenvoorde et al., *Affective and Cognitive Decision-Making in Adolescents*, 35 DEVELOPMENTAL NEUROPSYCHOLOGY 539–554 (2010).

Ashley R. Smith, Jason Chein & Laurence Steinberg, *Peers increase adolescent risk taking even when the probabilities of negative outcomes are known.*, 50 DEVELOPMENTAL PSYCHOLOGY 1564–1568 (2014).

B. J. Casey, *Beyond Simple Models of Self-Control to Circuit-Based Accounts of Adolescent Behavior*, 66 ANNUAL REVIEW OF PSYCHOLOGY 295–319 (2015).

B. J. Casey et al., *Imaging the developing brain: what have we learned about cognitive development?*, 9 TRENDS IN COGNITIVE SCIENCES 104–110 (2005).

B. J. Casey et al., *The storm and stress of adolescence: Insights from human imaging and mouse genetics*, DEVELOPMENTAL PSYCHOBIOLOGY (2010).

B. R. Braams et al., *Longitudinal Changes in Adolescent Risk-Taking: A Comprehensive Study of Neural Responses to Rewards, Pubertal Development, and Risk-Taking Behavior*, 35 JOURNAL OF NEUROSCIENCE 7226–7238 (2015).

Beatriz Luna, Aarthi Padmanabhan & Kirsten O'Hearn, *What has fMRI told us about the Development of Cognitive Control through Adolescence?*, 72 BRAIN AND COGNITION 101–113 (2010).

Bernd Figner et al., *Risky choice in children, adolescents, and adults: Affective versus deliberative processes and the role of executive functions*. 35 JOURNAL OF EXPERIMENTAL PSYCHOLOGY: LEARNING, MEMORY, AND COGNITION 709-730 (2009).

Bernd Figner & Elke U. Weber, *Who Takes Risks When and Why?*, 20 CURRENT DIRECTIONS IN PSYCHOLOGICAL SCIENCE 211–216 (2011).

Catalina J. Hooper et al., *Adolescents Performance on the Iowa Gambling Task: Implications for the Development of Decision Making and Ventromedial Prefrontal Cortex.*, 40 DEVELOPMENTAL PSYCHOLOGY 1148–1158 (2004).

Cecile D. Ladouceur et al., *White matter development in adolescence: The influence of puberty and implications for affective disorders*, 2 DEVELOPMENTAL COGNITIVE NEUROSCIENCE 36–54 (2012).

Conor Liston et al., *Frontostriatal Microstructure Modulates Efficient Recruitment of Cognitive Control*, 16 CEREBRAL CORTEX 553–560 (2005).

D. B. Dwyer et al., *Large-Scale Brain Network Dynamics Supporting Adolescent Cognitive Control*, 34 JOURNAL OF NEUROSCIENCE 14096–14107 (2014).

Edward P. Mulvey, *Highlights From Pathways to Desistance: A Longitudinal Study of Serious Adolescent Offenders*, Office of Juvenile Justice and Delinquency Prevention, *available at* https://www.ncjrs.gov/pdffiles1/ojjdp/230971.pdf. (2011)

Edward P. Mulvey, *Juvenile Justice Research: Implications for Intervention and Policy*, (2017).

Elizabeth Cauffman et al., *Age differences in affective decision making as indexed by performance on the Iowa Gambling Task.*, 46 DEVELOPMENTAL PSYCHOLOGY193–207 (2010).

Elizabeth P. Shulman et al., *The dual systems model: Review, reappraisal, and reaffirmation*, 17 DEVELOPMENTAL COGNITIVE NEUROSCIENCE 103–117 (2016).

ELIZABETH S. SCOTT & LAURENCE D. STEINBERG, RETHINKING JUVENILE JUSTICE (2010).

James M. Bjork et al., *Brain Maturation and Risky Behavior: The Promise and the Challenges of Neuroimaging-Based Accounts*, CHILD DEVELOPMENT PERSPECTIVES (2012).

Jason Chein et al., *Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry*, 14 DEVELOPMENTAL SCIENCE (2010).

Justin L. Vincent et al., *Evidence for a Frontoparietal Control System Revealed by Intrinsic Functional Connectivity*, 100 JOURNAL OF NEUROPHYSIOLOGY 3328–3342 (2008).

Kai Hwang, Michael N. Hallquist & Beatriz Luna, *The Development of Hub Architecture in the Human Functional Brain Network*, 23 CEREBRAL CORTEX 2380–2393 (2012).

Karol Silva et al., *Peers Increase Late Adolescents Exploratory Behavior and Sensitivity to Positive and Negative Feedback*, 26 JOURNAL OF RESEARCH ON ADOLESCENCE 696–705 (2015).

Kathryn C. Monahan et al., *Trajectories of antisocial behavior and psychosocial maturity from adolescence to young adulthood.*, 45 DEVELOPMENTAL PSYCHOLOGY 1654–1668 (2009).

L. D. Selemon, *A role for synaptic plasticity in the adolescent development of executive function*, 3 TRANSLATIONAL PSYCHIATRY (2013).

Lauren E. Sherman et al., *Development of the Default Mode and Central Executive Networks across early adolescence: A longitudinal study*, 10 DEVELOPMENTAL COGNITIVE NEUROSCIENCE 148–159 (2014).

Laurence Steinberg, *Adolescent Development and Juvenile Justice*, 5 ANNUAL REVIEW OF CLINICAL PSYCHOLOGY 459–485 (2009).

LAURENCE D. STEINBERG, AGE OF OPPORTUNITY: LESSONS FROM THE NEW SCIENCE OF ADOLESCENCE (2015).

LAURENCE D. STEINBERG, ADOLESCENCE (2017).

Laurence Steinberg, *Adolescent brain science and juvenile justice policymaking*, 23 PSYCHOLOGY, PUBLIC POLICY, AND LAW 410–420 (2017).

Laurence Steinberg, *Commentary on Special Issue on the Adolescent Brain: Redefining Adolescence*, 70 NEUROSCIENCE & BIOBEHAVIORAL REVIEWS 343–346 (2016).

Laurence Steinberg et al., *Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model.*, 44 DEVELOPMENTAL PSYCHOLOGY 1764–1778 (2008).

Laurence Steinberg et al., *Age Differences in Future Orientation and Delay Discounting*, 80 CHILD DEVELOPMENT 28–44 (2009).

Laurence Steinberg et al., *Around the world, adolescence is a time of heightened sensation seeking and immature self-regulation*, DEVELOPMENTAL SCIENCE (2017).

Laurence Steinberg & Kathryn C. Monahan, *Age differences in resistance to peer influence.*, 43 DEVELOPMENTAL PSYCHOLOGY1531–1543 (2007).

Linda Van Leijenhorst et al., *What Motivates the Adolescent? Brain Regions Mediating Reward Sensitivity across Adolescence*, 20 CEREBRAL CORTEX 61–69 (2009).

Leah H. Somerville, Negar Fani & Erin B. McClure-Tone, *Behavioral and Neural Representation of Emotional Facial Expressions Across the Lifespan*, 36 DEVELOPMENTAL NEUROPSYCHOLOGY 408–428 (2011).

Nico U. F. Dosenbach et al., *Prediction of Individual Brain Maturity Using fMRI*, 329 SCIENCE 1358–1361 (2010).

Nico U. F. Dosenbach, Steven E. Petersen & Bradley L. Schlaggar, *The Teenage Brain*, 22 CURRENT DIRECTIONS IN PSYCHOLOGICAL SCIENCE 101–107 (2013).

Margo Gardner & Laurence Steinberg, *Peer Influence on Risk Taking, Risk Preference, and Risky Decision Making in Adolescence and Adulthood: An Experimental Study.*, 41 DEVELOPMENTAL PSYCHOLOGY 625–635 (2005).

Michael C. Stevens et al., *Functional neural networks underlying response inhibition in adolescents and adults*, 181 BEHAVIOURAL BRAIN RESEARCH 12–22 (2007).

Monica Luciana & Paul F. Collins, *Incentive Motivation, Cognitive Control, and the Adolescent Brain: Is It Time for a Paradigm Shift?*, CHILD DEVELOPMENT PERSPECTIVES (2012).

Monique Ernst et al., *fMRI Functional Connectivity Applied to Adolescent Neurodevelopment*, 11 ANNUAL REVIEW OF CLINICAL PSYCHOLOGY 361–377 (2015).

Pernille J. Olesen et al., *Combined analysis of DTI and fMRI data reveals a joint maturation of white and grey matter in a fronto-parietal network*, 18 COGNITIVE BRAIN RESEARCH 48–57 (2003).

RICHARD J. BONNIE, REFORMING JUVENILE JUSTICE: A DEVELOPMENTAL APPROACH (2013).

Terrie E. Moffitt, *Life-Course-Persistent versus Adolescence-Limited Antisocial Behavior, in* DEVELOPMENTAL PSYCHOPATHOLOGY, SECOND EDITION (Dante Cicchetti and Donald J. Cohen eds. 2015).

Todd A. Hare et al., *Biological Substrates of Emotional Reactivity and Regulation in Adolescence During an Emotional Go-Nogo Task*, 63 BIOLOGICAL PSYCHIATRY 927–934 (2008).

Tomas Paus, *Brain Development, in* HANDBOOK OF ADOLESCENT PSYCHOLOGY (R. Lerner and L. Steinberg eds. 2009).

Vincent J. Schmithorst & Weihong Yuan, *White matter development during adolescence as shown by diffusion MRI*, 72 BRAIN AND COGNITION 16–25 (2010).

JULIE A. GALLAGHER, PSY.D. ABPP

CLINICAL AND FORENSIC PSYCHOLOGIST
BOARD CERTIFIED IN FORENSIC PSYCHOLOGY

2200 21st Ave. S., Suite 401
Nashville, Tennessee 37212
www.DrJulieGallagher.com

Phone (615) 491-3229
Fax (615) 750-5796
Dr.Julie.Gallagher@gmail.com

Board Certification:
Forensic Psychology, American Board of Professional Psychology (ABPP), Diplomate #7177

Licensure:
Tennessee, Psychologist and Health Service Provider #3160
Virginia, Clinical Psychologist #0810003319
Washington State, Clinical Psychologist #PY00003261

Professional Service Positions:
President-Elect, American Academy of Forensic Psychology, 2017-present
Secretary, American Academy of Forensic Psychology, 2015-2016
Examination Faculty, American Board of Forensic Psychology, 2016-present
Consultant, Tennessee Board of Examiners of Psychology, 2017-present

Education:

Psy.D. in Clinical Psychology	Baylor University, Waco, TX, August 2002
M.S. in Clinical Psychology	Baylor University, Waco, TX, August 2000
B.A. in Psychology	Cornell University, Ithaca, NY, May 1997

Internship and Residency:

Residency in Forensic Psychology	St. Elizabeths Hospital, Washington, DC September 2002 - 2003
APA-accredited Clinical Internship	St. Elizabeths Hospital, Washington, DC September 2001 - 2002

Academic Affiliation:

Clinical Assistant Professor	Department of Psychiatry and Behavioral Sciences University of Washington School of Medicine Seattle, Washington (2006-2011)

Professional Memberships:
Fellow, American Academy of Forensic Psychology
Member, American Psychological Association
Member, American Psychology-Law Society
Member, Society for Personality Assessment

Specialized Training:

Sex Offender Treatment Provider Training	Tennessee Sex Offender Treatment Board, Nashville, TN November 2015
Forensic Evaluator Certification Training	Tennessee Department of Mental Health and Substance Abuse Services, Nashville, TN October 2013
Basic & Advanced Forensic Evaluation	Institute of Law, Psychiatry and Public Policy University of Virginia, Charlottesville, VA September 2003, May 2004 & January 2005
Juvenile Forensic Evaluation	Institute of Law, Psychiatry and Public Policy University of Virginia, Charlottesville, VA April 2005
Sex Offender Evaluation	Institute of Law, Psychiatry and Public Policy University of Virginia, Charlottesville, VA November 2003

Clinical Experience:

Forensic Psychologist January 2012 – present

Clinical and Forensic Private Practice, Nashville, Tennessee

- Perform forensic assessments of adult and juveniles' competency to stand trial; competency to waive Miranda; mental state at the time of the offense (insanity and diminished capacity); risk for dangerousness; factors relevant to sentencing; psychological injury; and juvenile transfer to adult court
- Testify in state and federal court as an expert witness
- Perform pre-employment assessments for the Federal Aviation Administration (FAA)
- Provide expert consultation and continuing education in forensic mental health to mental health institutions, universities and court systems
- Provide consultation and report-writing assistance to other forensic psychologists across the country

Director, Forensic Services Program January 2009 – February 2011

Child Study and Treatment Center, Tacoma, Washington

- Ran a juvenile forensic evaluation program serving the entire state of Washington
- Performed Court-ordered assessments addressing juveniles' competency to stand trial, mental state at the time of the offense (diminished capacity), dangerousness and risk for re-offense
- Testified as an expert witness in juvenile courts throughout the state
- Participated in re-writing the relevant state laws that govern forensic evaluations
- Supervised a forensic evaluator, an administrative assistant, postdoctoral fellows, practicum students, and interns who were part of an APA-approved internship program

Director, Forensic Psychology Postdoctoral Fellowship Program March 2007 – February 2011
Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Seattle, Washington (Juvenile Program: January 2009 – February 2011; Adult Program: March 2007 – January 2009)

- Coordinated numerous administrative activities including supervision, program evaluation, didactic activities, and various committees
- Developed and implemented a curriculum to prepare fellows for board certification in forensic psychology and psychiatry
- Supervised postdoctoral fellows performing forensic evaluations
- Provided continuing education in forensic psychology to the Pacific Northwest legal and mental health communities

Clinical Psychologist September 2005 – December 2008
Inpatient Forensic Evaluation Program, Center for Forensic Services, Western State Hospital, Tacoma, Washington

- Performed Court-ordered assessments of male and female adult defendants, addressing such questions as competency to stand trial, mental state at the time of the offense (insanity and diminished capacity), dangerousness and risk for re-offense
- Evaluated and petitioned the court for the civil commitment of seriously mentally ill individuals
- Testified as an expert witness in both criminal and civil courts
- Quickly achieved senior evaluator status and was assigned the most challenging cases, including high-profile murder cases
- Served in a stand-in administrative role for the program as needed, liaising with local courts, attorneys, and hospital administrators
- Developed best practice guidelines for forensic assessment within the institution as an invited member of the Forensic Practice Standards Committee
- Supervised psychology interns as part of an APA-accredited internship program and regularly provided peer supervision to more junior forensic psychologists and the hospital neuropsychologist

Clinical Psychologist September 2003 – 2005
Forensic Services Program, Fairfax County Adult Detention Center, Fairfax, Virginia

- Performed Court-ordered assessments of male and female adult defendants, addressing such questions as competency to stand trial, mental state at the time of the offense, risk assessment for sex offenders, and treatment needs
- Testified as an expert witness in Superior Court
- Provided crisis intervention, suicide assessment, and group psychotherapy to inmates in a maximum security detention facility
- Created effective behavior management plans for difficult to manage personality disordered inmates and trained correctional staff in their implementation
- Supervised psychology interns and residents performing forensic evaluations and group psychotherapy
- Consulted with law enforcement and legal personnel, as well as family members and other service providers

Clinical Psychologist December 2002 – September 2005

Clinical and Forensic Private Practice, Arlington & Alexandria, Virginia

- Performed pre-employment assessments of applicants for careers in law enforcement, public safety and security
- Performed risk assessments and treatment-related assessments of adolescent sex offenders and their families
- Provided group psychotherapy to adolescent sex offenders and their parents

Acting Clinical Administrator July 2003 – September 2003

Pre-Trial Services Branch, John Howard Pavilion, St. Elizabeths Hospital, Department of Mental Health, Washington, DC

- Performed half of the Court-ordered assessments of male pre-trial defendants in the District of Columbia
- Assessed issues including competency to stand trial, criminal responsibility and mental health as it relates to sentencing
- Provided crisis intervention, multidisciplinary treatment planning and administrative management of an inpatient ward of male pre-trial defendants
- Consulted with law enforcement and legal personnel, as well as family members and other service providers
- Provided forensic training to medical students and psychiatry residents

Forensic Psychology Intern and Resident September 2001 – 2003

Department of Psychology, John Howard Pavilion, St. Elizabeths Hospital, Department of Mental Health, Washington, DC

- Performed Court-ordered assessments of pre- and post-trial inpatients and outpatients, addressing such questions as competency to stand trial, criminal responsibility, neuropsychological functioning, risk for violence, and treatment needs
- Provided long-term individual and group psychotherapy to insanity acquittees with serious mental illness
- Provided crisis intervention, competency restoration, and anger management group therapy to pre-trial defendants
- Supervised students providing individual psychotherapy to insanity acquittees
- Prepared and presented cases to the multidisciplinary Forensic Review Board
- Participated in multidisciplinary treatment team meetings and developed treatment plans for pre-and post-trial inpatients
- Consulted with law enforcement and legal personnel, as well as family members and other service providers
- Participated in weekly didactic training in forensics, assessment, and group psychotherapy

Practicum Trainee July 2000 – June 2001

Adult and Juvenile Probation Departments, Groesbeck, Texas

- Coordinated sex offender treatment program serving three rural counties
- Led adult sex offender and co-led anger management therapy groups
- Performed Court-ordered and treatment-related assessments of adult probationers and juvenile offenders
- Performed crisis intervention as well as short- and long-term individual psychotherapy with adult probationers
- Consulted with law enforcement, probation, and legal personnel, as well as family members, victims, and other psychological service providers

Teaching Assistant January – May, 2000 & 2001

Interpersonal, Existential and Humanistic Psychotherapy course. Baylor University Psy.D. Program, Waco, Texas

- Supervised first year doctoral students doing crisis intervention and individual psychotherapy with undergraduates
- Taught interviewing and assessment techniques

Graduate Assistant Therapist July 1999 – June 2000

Baylor University Counseling Center, Waco, Texas

- Provided short- and long-term individual psychotherapy to college students with a range of diagnoses
- Performed crisis intervention on an on-call basis addressing problems ranging from panic attacks to suicidality
- Consulted with faculty, staff, parents, medical and law enforcement personnel

Psy.D. Trainee July 1998 – June 1999

Special Treatment Center for Developmentally Disabled Youth, Mexia State School, Mexia, Texas

- Evaluated juvenile offenders with developmental disabilities, assessing intellectual functioning, adaptive behavior, competency to stand trial, and psychopathology
- Developed and implemented individual behavior management plans based on functional analyses
- Performed individual and group therapy focusing on issues including sexual offending, sexual abuse, bereavement, and the management of aggressive behavior
- Participated in multidisciplinary treatment team meetings

Clinical Psychoendocrinology Trainee June 1997 - June 1998

Children's Hospital of Buffalo, New York

- Evaluated gender identity/role and sexual orientation of child and adolescent intersex and adult transsexual patients
- Participated in the multidisciplinary treatment planning and management of newborn intersex children
- Organized weekly Psychoendocrine Journal Club

Volunteer Counselor June 1997 – June 1998

Sexually Inappropriate Behavior Program, Buffalo, New York

- Interviewed parents of adolescent sex offenders
- Wrote Court-ordered psychological evaluations addressing appropriateness for sex offender treatment
- Administered, scored, and interpreted assessment inventories
- Created materials advertising the program

Research Experience:

Research Coordinator Summer 1996 & June 1997 – June 1998

Psychoendocrine Outcomes of Individuals with Congenital Variations of the Genitals. Children's Hospital of Buffalo, New York

- Completed psychosexual interviews of female subjects
- Interviewed subjects' parents by phone
- Developed questionnaires for the intersexed population
- Reviewed charts and selected subjects for inclusion
- Located adult subjects for follow-up
- Coordinated schedules of doctors, nurses and psychologist
- Performed data entry and analysis using SPSS

Research Coordinator October 1997 – June 1998

Effect of Growth Hormone Replacement Therapy in Childhood-Onset Growth Hormone Deficient Patients Previously Treated to Final Height. Children's Hospital of Buffalo, New York

- Recruited subjects
- Located necessary technical equipment
- Coordinated patient visits and drug administration

Research Assistant October 1997 – June 1998

Safety and Efficacy of ProLease hGH Administered Monthly in Children with Growth Failure Due to Growth Hormone Deficiency. Children's Hospital of Buffalo, New York

- Coordinated supervisory visits between drug company representatives and nursing staff
- Coordinated drug availability and patient visits
- Prepared samples for lab work

Research Assistant Summer 1996

Post-treatment Outcomes of Childhood Onset Growth Hormone Deficiency: Medical and Psychosocial Status in Adulthood. Children's Hospital of Buffalo, New York

- Performed data input and analysis using SPSS

Publications:

Haun, J.J., **Gallagher, J.A.**, & Milz, A.A. (2010). The influence of time and treatment on recall of mental state at the time of offense: Incompetent defendants and evaluation of insanity. Journal of Forensic Psychology Practice, 10(5), 464-475.

Mazur, T., Sandberg, D.E., Perrin, M.A., **Gallagher, J.A.**, & MacGillivray, M.H. (2004). Male pseudohermaphroditism: Long-term quality of life outcome in five 46,XY individuals reared female. Journal of Pediatric Endocrinology and Metabolism, 17(6), 809-823.

Conference Presentations:

Gallagher, J.A. (2016, August). Minimizing bias in forensic evaluations. In **J.A. Gallagher** (Chair), Threats to reliability in forensic evaluations. Symposium conducted at the meeting of the American Psychological Association, Denver, CO.

Boss, A. & **Gallagher, J.A.** (2016, March). Hands-on report writing workshop. Half-day workshop presented at the meeting of the Society for Personality Assessment, Chicago, IL.

Gallagher, J.A. (2015, August). Juvenile disposition and treatment evaluations. In **J.A. Gallagher** (Chair), Juvenile disposition and treatment evaluations. Symposium conducted at the meeting of the American Psychological Association, Toronto, CA.

Walker, J.S., **Gallagher, J.A.,** Auble, P.A., Gale, S., Milliner, K., Phillips, M., Engle, M. & Walker, S. (2014, October). Psychology in the courtroom: Critical issues and controversies. Symposium presented at the meeting of the Tennessee Psychological Association, Nashville, TN.

Gallagher, J.A. & Boss, A. (2014, March). How to write a better forensic report. Half-day workshop presented at the meeting of the Society for Personality Assessment, Arlington, VA.

Gallagher, J.A. (2010, October). Evaluation of juveniles' competency to stand trial. Full-day workshop presented at the meeting of the Montana Psychological Association, Missoula, MT.

Gallagher, J.A. (2004, May). Evaluation of sanity: A complex case. Advanced Forensic Evaluation Training, Institute of Law, Psychiatry and Public Policy, University of Virginia, Charlottesville, VA.

Mazur, T., **Gallagher, J.A.**, Sandberg, D.E., Nadgir, U.M., Buchlis, J.G., & MacGillivray, M.H. (1998, June). Micropenis and adult sexual functioning: A report on three couples. Poster presentation at the meeting of the Endocrine Society, New Orleans, LA.

Gallagher, J.A., Mazur, T., Sandberg, D.E., Nadgir, U.M., Buchlis, J.G. & MacGillivray, M.H. (1998, May). Psychosexual outcomes of children with a micropenis. Poster presentation at the Great Lakes Conference on Child Health Psychology, Louisville, KY.

Other Presentations and Workshops:

Gallagher, J.A. (2017 & 2015). Forensic psychology. Presented to the Davidson County Juvenile Court, Nashville, TN.

Jacobs, M., Auble, P., & **Gallagher, J.A.** (2016). A damaged brain: A case of seizures, neurosurgery & criminal behavior. Presented to the Forensic Psychology Interest Group, Nashville, TN.

Gallagher, J.A. (2016). Racial and ethnic fairness and the law. Presented to the Forensic Interest Group, Nashville, TN.

Gallagher, J.A. (2015). Assessment of malingering in competency evaluations. Invited workshop at the State of Utah Annual Forensic Evaluator Conference, Provo, UT.

Gallagher, J.A. (2015). Forensic assessment instruments in competency to stand trial evaluations. Invited workshop presented at the State of Utah Annual Forensic Evaluator Conference, Provo, UT.

Gallagher, J.A. (2015). Juvenile transfer to adult court. Presented to the Forensic Psychology Interest Group, Nashville, TN.

Gallagher, J.A. (2015). Overcoming common errors in forensic report writing. Presented to the Forensic Interest Group, Nashville, TN.

Gallagher, J.A. (2014). Overcoming common errors in forensic report writing. Presented to the Forensic Psychology Interest Group, Nashville, TN.

Gallagher, J.A. (2013). Mental health and competency to stand trial. Presented to the Tennessee Bar Association, Nashville, TN.

Gallagher, J.A. (2013). Juvenile transfer to adult court. Invited Grand Rounds presentation at Meharry Medical College, Nashville, TN.

Gallagher, J.A. (2010). Evaluation of juveniles' competency to stand trial. Half-day workshop provided for the Spokane Juvenile Court and local evaluators.

Gallagher, J.A. (2009). An introduction to forensic mental health assessment. Invited lecture provided for law students at Seattle University Law School, Seattle, WA.

Gallagher, J.A. & Hendrickson, R.H. (2008). Advanced issues in competency assessment. Professional seminar provided to students and staff at Western State Hospital, Tacoma, WA.

Gallagher, J.A. (2008). An introduction to mens rea and diminished capacity. Annual Forensic Mental Health Symposium, Western State Hospital, Tacoma, WA.

Gallagher, J.A. (2007). Mental illness and criminal law: An overview. Invited lecture provided for second and third year law students at Seattle University Law School, Seattle, WA.

Gallagher, J.A. (2006). Mental illness and criminal law: An overview. Invited lecture provided at the Seattle City Attorney's Office, Seattle, WA.

Gallagher, J.A. (2005). Forensic psychology in correctional settings. Invited lecture provided for Administration of Justice undergraduates at George Mason University, Manassas, VA.

Gallagher, J.A. & Daniels, I.N. (2004). Forensic psychology in correctional settings. Invited lecture provided for Administration of Justice undergraduates at George Mason University, Manassas, VA.

Gallagher, J.A. (2003). Assessment and treatment of intersex individuals. Professional seminar provided for students and staff at St. Elizabeths Hospital, Washington, DC.

Gallagher, J.A. & Eyler V.A. (2002, 2003). Understanding psychological testing. Professional seminar provided for psychiatry residents at St. Elizabeths Hospital, Washington, DC.

Gallagher, J.A. (2000). Stress management. Workshop provided for undergraduates at Baylor University, Waco, TX.

2012 WL 121237 (U.S.) (Appellate Brief)
Supreme Court of the United States.

Evan MILLER, Petitioner,

v.

STATE OF ALABAMA, Respondent.

Kuntrell JACKSON, Petitioner,

v.

Ray HOBBS, Director, Arkansas Department of Correction, Respondent.

Nos. 10-9646, 10-9647.
January 13, 2012.

On Writ of Certiorari to the Alabama Court of Criminal Appeals
On Writ of Certiorari to the Supreme Court of Arkansas

Brief for the American Medical Association and the American Academy of Child and Adolescent Psychiatry as Amici Curiae in Support of Neither Party

Khai LeQuang
Elliott S. Henry
Orrick, Herrington & Sutcliffe LLP
777 South Figueroa Street, Suite 3200
Los Angeles, CA 90017
213-629-2020

E. Joshua Rosenkranz
Counsel of Record
Orrick, Herrington & Sutcliffe LLP
51 West 52nd Street
New York, NY 10019
jrosenkranz@orrick.com
212-506-5000
Counsel for Amici Curiae

*i QUESTION PRESENTED

Whether the Eighth Amendment's ban on cruel and unusual punishment prohibits the imprisonment of a juvenile for life without the possibility of parole as punishment for the juvenile's commission of a homicide offense.

*ii TABLE OF CONTENTS

*iv TABLE OF CITED AUTHORITIES

Scientific Authorities

*1 INTERESTS OF AMICI CURIAE [*]

The American Medical Association. The American Medical Association (AMA) is the largest professional association of physicians, residents and medical students in the United States. Additionally, through state and specialty medical societies and other physician groups seated in its House of Delegates, substantially all U.S. physicians, residents and medical students are represented in the AMA's policy making process. Founded in 1847, the objects of the AMA are to promote the science and art of medicine and the betterment of public health.

The American Academy of Child and Adolescent Psychiatry. Founded in 1953, the American Academy of Child and Adolescent Psychiatry (AACAP) is comprised of over 7,500 child and adolescent psychiatrists and other interested physicians. Consistent with the focus of the juvenile court system on rehabilitation rather than retribution and multiple international treaties, including the UN Convention of Rights of the Child, the AACAP has adopted a policy statement strongly opposing the imposition of a sentence of life without *2 the possibility of parole for crimes committed as juveniles. AACAP Policy Statement, June 2009, *available at* http://www.aacap.org/cs/root/policy_statements/life_without_parole_for_juvenile_offenders.

Each of the above-referenced *amici* is committed to the advancement of science. While not taking a formal position on whether sentencing a juvenile to a term of imprisonment of life without the possibility of parole violates the protections provided by the Eighth Amendment of the U.S. Constitution, *amici* submit this brief to describe the scientific findings of medical, psychiatric, and psychological research relevant to this issue.

SUMMARY OF ARGUMENT

The adolescent's mind works differently from ours. Parents know it. This Court has said it. Legislatures all over the world have presumed it for decades or more. And scientific evidence has continued to shed more light on how and why adolescent behavior differs from adult behavior.

The differences in behavior have been documented by scientists along several dimensions. Scientists have found that adolescents as a group, even at later stages of adolescence, are more likely than adults to engage in risky, impulsive, and sensation-seeking behavior. This is, in part, because they overvalue short-term benefits and rewards, and are less

capable of controlling their impulses making them susceptible to acting in a reflexive rather than a *3 planned voluntary manner. Adolescents are also more emotionally volatile and susceptible to stress and peer influences. In short, the average adolescent cannot be expected to act with the same control or foresight as a mature adult.

Behavioral scientists have observed these differences for some time, but only recently have studies provided an understanding of the neurobiological underpinnings for why adolescents act the way they do. For example, brain imaging studies reveal that adolescents generally exhibit greater neural reactivity than adults or children in areas of the brain that promote risky and reward-based behavior. These studies also demonstrate that the brain continues to mature, both structurally and functionally, throughout adolescence in regions of the brain responsible for controlling thoughts, actions, and emotions. Together, these studies indicate that the adolescent period poses vulnerabilities to risk taking behavior but, importantly, that this is a temporary stage.

While science cannot gauge moral culpability, scientists can shed light on some of the measurable attributes that the law has long treated as highly relevant to culpability and the appropriateness of punishment. This brief focuses on what science can tell us about the neurological, physiological, psychological, emotional, and behavioral development of adolescents from the perspective of researchers and medical professionals.

*4 ARGUMENT

THE STRUCTURAL AND FUNCTIONAL IMMATURITIES OF THE ADOLESCENT BRAIN PROVIDE A BIOLOGICAL BASIS FOR THE BEHAVIORAL IMMATURITIES EXHIBITED BY ADOLESCENTS.

Although adolescents [1] can, and on occasion do, exhibit adult levels of judgment and control, their ability to do so is limited and unreliable compared to that of adults. Adolescents, as a group, value risks and rewards differently from adults, which, coupled with limitations in controlling their impulses and recognizing and regulating emotional responses, makes them vulnerable to impulsive acts. *See* Point A, *infra.*

Moreover, recent advances in brain-imaging technology confirm that the very regions of the brain that are associated with voluntary behavior control and regulation of emotional response and impulsivity are structurally immature during adolescence. Studies have also revealed that these structural immaturities are consistent with age-related differences in both brain function and behavior. *See* Point B, *infra.*

These findings have led to an "explosion of scientific papers and popular articles" about the *5 immaturities of the adolescent brain and how these immaturities explain the risky and impulsive behavior exhibited by teens. [2]

A. Adolescents Are Less Able Than Adults to Voluntarily Control Their Behavior.

Numerous studies of adolescent behavior over the last two decades confirm the stereotype that adolescents, as a group, are prone to making impulsive or reactive judgments. "Relative to individuals at other ages, ... adolescents ... exhibit a disproportionate amount of reckless behavior, sensation seeking and risk taking." [3] Sensation-seeking peaks during adolescence across cultures and species, and is believed to be an adaptive and normal part of development that promotes learning and independence. [4] *6 Nevertheless, sensation-seeking behavior can result in actions that compromise survival (referred to as "risk-taking" behaviors) and involve sub-optimal decision-making. Risk-taking of all sorts - whether drunk driving, unprotected sex, experimentation with drugs, or even criminal activity - is so pervasive that "it is statistically aberrant to refrain from such [risk-taking] behavior during adolescence." [5] The difference between adolescent and adult behavior, however, is not a function of adolescents' inability to distinguish right from wrong or

in their intellectual abilities *per se*, but rather from psychosocial limitations in their ability to consistently and reliably control their behavior. [6]

Specifically, adolescents are less able, on average, than adults to self-regulate, or "cognitively" control, their behavior. [7] Cognitive control refers to the ***7** ability to voluntarily exert goal-directed behavior while controlling compelling but goal-inappropriate responses. [8] Scientists have identified various interrelated immaturities in adolescents' self-regulatory abilities that contribute to their limitation in controlling their impulses and their greater tendency to engage in risky or reckless behavior. To name just a few, adolescents (1) tend to be more strongly motivated by the possibility of reward than adults; (2) have greater difficulty controlling their impulses; and (3) have greater difficulty recognizing and regulating emotional responses. We take a closer look at each of these factors below.

Reward Sensitivity. One of the main reasons adolescents are more likely to engage in risky behavior than adults is that adolescents tend to experience heightened levels of sensitivity to rewards, especially to immediate rewards. [9] ***8** Placing a higher value on the potential reward leads to lower risk-reward ratios for adolescents, relative to adults, and thus a higher likelihood of engaging in the risky behavior. [10] In other words, adolescent behavioral research suggests that adolescents take more risks because they overvalue the potential reward, not because they are less able to appreciate the risks, as was once believed. [11] "[A]dolescents' greater involvement in risk taking, compared to adults', does not appear to stem from youthful ignorance, irrationality, delusions of invulnerability, or misperceptions of risk." [12] Rather, it appears that adolescents and adults perceive *risks* similarly [13], but they evaluate potential *rewards* differently, especially when the risky behavior is weighed against the cost. [14]

***9** Furthermore, studies have shown that adolescents are more likely to take risks when they are in the presence of peers. "[O]ne of the hallmarks of adolescent risk taking is that it is much more likely than that of adults to occur in the presence of peers, as evidenced in studies of reckless driving, substance abuse, and crime." [15] More recent studies have also shown that this increased risk taking in the presence of peers is associated with greater neural activity in the areas of the brain associated with reward processing. [16] In fact, adolescents appear to place unique reward value on the presence of peers. With adolescents, "awareness of peers selectively amplifies activity in the [] brain's incentive processing system, which in turn influences subsequent decisions about risk." [17] Adults, on the other hand, "showed no differences in the ***10** activation of these regions as a function of social context." [18]

Impulse Control. "A cornerstone of cognitive development is the ability to suppress inappropriate thoughts and actions in favor of goal-directed ones, especially in the presence of compelling incentives." [19] Impulse control means allowing a goal-directed response to override a more compelling/reflexive, yet goal-inappropriate response. [20] The ability to control one's impulsive reactions to an event or problem is necessary to achieve adult levels of problem solving ability, logical reasoning, and the consistent exercise of good judgment. [21]

Adolescents have observable limitations in their ability to control their impulses. The relative inability of adolescents to control impulsive behavior is well-documented by studies on developmental changes in impulsivity and self-management over the course of adolescence. [22] "A number of classic developmental studies have shown that this ability develops throughout ***11** childhood and adolescence." [23] Capacity for self-direction has been shown to increase gradually throughout adolescence and into young adulthood. [24] Likewise, impulsivity tends to decline linearly from childhood to adulthood. [25] These findings indicate that adolescents have not yet attained adult levels of impulse control. In other words, adolescents are less able than adults to consistently reflect before they act.

Emotional Regulation. All individuals regulate their emotional responses to events. They increase or decrease their emotional reactions to stimuli in accordance with their behavioral goals.[26] The ability to regulate one's emotions efficiently is crucial for mental and physical health as well as for appropriate social interactions, and impairment of this capability is associated with affective disorders and a variety of other maladaptive psychological conditions.[27] This ability, however, continues to develop through adolescence into adulthood.[28] As a result, *12 similar to their ability to control impulses, adolescents have less ability to regulate their emotional responses to stimuli than adults.[29]

This relative limitation is important for understanding adolescents' ability to voluntarily control their behavior. Indeed, many situations, particularly those involving social interactions, arouse adolescents' emotional system and impact their ability to make informed decisions about their actions. Peer pressure, for example, can arouse emotions of fear, rejection, or desire to impress friends that can undermine the reliability of adolescent behavioral control systems and result in actions taken without full consideration or appreciation of the consequences.[30]

Each of these attributes continues to develop throughout adolescence and early adulthood, and is critical to the ability to effectively and consistently control one's behavior.[31] The developmental immaturities that adolescents *13 exhibit with respect to each of these attributes compound to make them particularly prone to engage in risky and sensation-seeking behavior.

Researchers have also found that these limitations are especially pronounced when other factors - such as stress, emotions, and peer pressure - enter the equation. These factors affect everyone's cognitive functioning, but they operate on the adolescent mind differently and with special force.

The interplay among stress, emotion, cognition, and voluntary behavior control in teenagers is particularly complex - and different from adults. Stress affects the ability to effectively regulate behavior as well as the ability to weigh costs and benefits and override impulses with rational thought.[32] Adolescents are more susceptible to stress from daily events than adults, which translates into a further distortion of their already skewed cost-benefit analysis.[33]

Emotion, like stress, also plays an important role in the ability to voluntarily control behavior, influencing decision-making and risk-taking behavior.[34] Because of their greater stress, *14 greater influx of gonadal hormones, and their relative inability to consistently regulate their emotional responses, adolescents are more emotionally volatile than adults - and children, for that matter.[35] As a result, adolescents tend to experience emotional states that are more extreme and more variable than those experienced by adults.[36]

In sum, the conclusion of the scientific research is that, for a variety of interrelated reasons, adolescents, as a group, cannot be expected to behave or make decisions in the same way as adults.

B. Recent Studies of the Brain Have Established a Biological Basis for the Observed Immaturities in Adolescent Behavior.

Modern brain research technologies have developed a body of data from the late 1990s to the present that provides a compelling picture of the inner workings of the adolescent brain.[37] *15 Indeed, brain imaging data provides convergent evidence for the ways in which adolescents are still immature.[38] Developmental neuroscience has now gathered extensive evidence that both the structure of the adolescent brain, and the way it functions, are immature compared to the adult brain.

This insight emerges from sophisticated and non-invasive brain imaging techniques performed by high-resolution structural and functional magnetic resonance imaging ("MRI") methods. [39] These imaging techniques are a quantum leap beyond previous methods for assessing brain development. Before the rise of neuroimaging, the understanding of brain development was gleaned ***16** largely from post-mortem examinations. [40] Modern imaging techniques, however, have begun to shed light on how a live brain operates, and how a particular brain develops over time. [41]

Technological breakthroughs have not only enabled scientists to confirm some of what was previously known or believed, but have also provided new evidence that has changed the way scientists understand the development of the human brain as it progresses from childhood through adolescence and into adulthood. [42] "[B]rain imaging studies in normal children and adolescents have been helpful in relating the dramatic maturation of cognitive, emotional, and social functions with the brain structures that ultimately underlie them." [43]

***17** In this regard, two complementary observations have been especially revealing. First, the parts of the brain that work together to support the control of behavior, including the prefrontal cortex (which comprises roughly the front third of the human brain), continue to mature even through late adolescence. [44] Second, in making behavioral choices, adolescents rely more heavily than adults on systems and areas of the brain that promote risk-taking and sensation-seeking behavior.

1. Adolescent Brains Are Structurally Immature in Areas of the Brain Associated with Enhanced Abilities of Executive Behavior Control.

When it comes to "response inhibition, emotional regulation, planning and organization," the so-called executive functions, a crucial part of the brain is the prefrontal cortex. [45] The prefrontal cortex is a core region that through its ability to integrate information across the brain supports ***18** planning of voluntary goal-directed responses and can exert control over more impulsive brain systems. As such, it is associated with a variety of cognitive abilities, [46] including those associated with voluntary behavior control and inhibition [47] such as risk assessment, [48] evaluation of reward and punishment, [49] and impulse control. [50] More generally, other functions associated with the prefrontal cortex include decision-making, [51] the ***19** ability to judge and evaluate future consequences, [52] recognizing deception, [53] responses to positive and negative feedback, [54] working memory, [55] and making moral judgments. [56]

The brain's frontal lobes are still structurally immature well into late adolescence, [57] and the prefrontal cortex is "one of the last brain regions to mature." [58] This, in turn, means that "response inhibition, emotional regulation, planning and ***20** organization ... continue to develop between adolescence and young adulthood." [59]

The adolescent brain, in particular the frontal lobes and specifically the prefrontal cortex, is immature in at least two distinct ways that directly affect an adolescent's ability to cognitively control behavior. First, the gray matter where neuronal brain cells reside continues to mature, supporting complex neural processing needed for generating cognitive plans. Second, the integrity of white matter neuronal connections, which supports the fast connectivity needed to executively control impulsive responses, is still improving. Maturation of processes in the gray and white matter of the brain support the complex information processing that underlies executive voluntary control of behavior, which underlies decreased risk taking in adulthood. When fully mature, the ability to effectively process complex information and quickly affect behavior supports the adult's ability to make better-informed executive decisions. [60]

***21** Pruning. The gray matter of the brain is where brain cell "neurons" reside and includes the top layer of the brain and also the nuclei within the brain. [61] As the brain matures, gray matter *thins* [62] through processes called synaptic

pruning, which is the programmed elimination of unused and cumbersome neuronal connections believed to support the ability for the brain to adapt to its environment. Just as the pruning of a rose bush strengthens the remaining branches, the pruning of excess connections leads to greater efficiency and strengthening of the ability for complex information processing that support consistent exercise of good judgment. [63] Maturational improvements in the gray matter continue to take ***22** place through adolescence and into adulthood. [64] Thus, changes in gray matter, including pruning, enhance the ability to process complex information quickly allowing the brain to make executive plans supporting voluntary control of behavior.

Scientists have known about pruning for decades, [65] but modern brain imaging technology has provided important insights into the process. [66] Until MRI technology emerged, the common wisdom was that the volume of gray matter spurted only once, shortly after birth, and then declined gradually over time. Brain scans have revealed a more complicated reality: In particular regions of the brain, gray matter blossoms once again later in childhood. [67] Gray matter volumes ***23** peak during the ages from 10-20 years, [68] and the prefrontal cortex is one of the places where gray matter increases - before adolescence - and then gets pruned over time, beyond adolescence. [69] The prefrontal cortex is also one of the last regions where pruning is complete and this region continues to thin past adolescence. [70] This means that one of the last areas of the brain to reach full maturity, as measured by pruning, is the region most closely associated with risk assessment, impulse control, emotional regulation, decision-making, and planning - in other words, the ability to reliably and voluntarily control behavior. [71]

Myelination. Another important measure of brain maturity is myelination. [72] Myelination is ***24** the process by which the brain's axonal connections become progressively insulated with a fatty white matter called myelin. Myelin surrounds the axons, which are neural fibers that use electrical impulses to carry information across long distances, and insulates the pathway, speeding the neural signal along the pathway. [73] "The presence of myelin makes communication between different parts of the brain faster and more reliable." [74] Myelination of "white matter" [75] continues through adolescence and into adulthood. [76]

***25** The integrity of the white matter, including myelination, matures at different rates across the brain. [77] Brain imaging data, supported by data gathered through the original histological (autopsy) techniques, [78] provides credible evidence that the connections from the prefrontal cortex are still developing well into adolescence and beyond, and are among the last pathways of the brain to mature. [79] In other words, maturation of prefrontal connectivity associated with voluntary behavior control (*i.e.*, risk assessment, impulse control, and emotional regulation) is not complete until late adolescence or beyond. Myelination also increases the efficiency of information processing and supports the integration of the widely ***26** distributed circuitry needed for complex behavior. [80] These structural changes are believed to underlie the functional integration (discussed below) of frontal regions with the rest of the brain. [81] The functional improvement of the connections between the various regions of the brain is believed to result from myelination that occurs during adolescence and is necessary for improved abilities of reliable self-control and better decision-making. [82] Efficient connectivity is needed for cognitive regions to interact with regions processing emotion, rewards, and social information in a timely and effective manner in order to control responses for optimal decision making. For example, recent research on the neural underpinnings of resistance to peer influence in adolescence indicates that improvements in this capacity may be linked to the development of greater connectivity between brain regions, and likely facilitates the better coordination of affect and cognition. [83] More generally, however, the development of improved self-regulatory abilities during and after adolescence is positively correlated with white matter maturation. [84]

*27 Top-Down Connectivity. Recent studies have shown that "development of top-down effective connectivity from cognitive control regions is critical in supporting active inhibitory control." [85] Top-down connectivity refers to the ability for executive regions, such as in the prefrontal cortex, to exert executive control on response regions. [86] fMRI has shown that the strength and number of top down functional connections continues to increase into adulthood. In addition, the organization of functional brain connections forming networks continues to optimize into adulthood. [87] These results are supported by studies measuring the integrity of structural white matter pathways, which show protracted development of the connections between the prefrontal cortex and subcortical regions of the brain areas that support cognitive control. [88] The protracted development of top-down connectivity therefore "may reflect a period of particular vulnerability to both the peak in risk-taking behavior during adolescence and the emergence and exacerbation of psychopathology, which is associated *28 with abnormalities in reward processing and cognitive control." [89]

2. Adolescent Brains Tend to Be More Active Than Adult Brains in Regions Associated With Risky, Impulsive, and Sensation-Seeking Behavior and Less Active in Regions Associated with the Ability to Voluntarily Control Behavior.

The brain is a complex network of interrelated parts. Each part is associated with different functions and works in conjunction with other parts to form systems. In general, the two neurobiological systems that inform our understanding of adolescent behavior, as discussed above in Point A, are (1) the motivational system, which includes the limbic and paralimbic regions of the brain; and (2) the cognitive control system, which is primarily comprised of the prefrontal cortex and its connections to the rest of the brain. [90] The differences between adolescent and adult behavior correlate with their respective and disparate reliance on each of these systems and their related brain structures. [91]

*29 The structural immaturities of the adolescent brain discussed above represent only one dimension of the immaturity of the adolescent brain. Developmental neuroimaging studies demonstrate that the regions of the brain associated with voluntary behavior control mature structurally at the same time as specific changes in how the brain functions. [92] These findings reveal that adolescents and adults exhibit different patterns of brain activity during decision-making tasks and provide insight into the neural underpinnings of the risky, impulsive, and sensation-seeking behavior of adolescents. [93]

Studies show that the motivational system, which underlies risky and reward-based behavior, develops earlier than the cognitive control system, which regulates such behavior. Furthermore, during adolescence, the motivational system *30 continues to develop more quickly than the cognitive control system. [94] The result is that adolescents experience increasing motivation for risky and reward-seeking behavior without a corresponding increase in the ability to self-regulate behavior.

The earlier development of the motivational system is evident in a number of areas of the brain. Among these are the amygdala and the nucleus accumbens which, in conjunction with specific neurochemical imbalances in the adolescent brain (see below), contribute to the relative dominance of the adolescent motivational system.

Amygdala. The amygdala is associated with aggressive and impulsive behavior. [95] The *31 amygdala is "a neural system that evolved to detect danger and produce rapid protective responses without conscious participation." [96] It dictates instinctive gut reactions, including fight or flight responses. [97] The amygdala is also a key component of circuitry involved in assessing salience, or the importance of environmental stimuli to survival, and is generally associated with processing emotional responses to a perceived danger. [98]

The prefrontal cortex - the primary region associated with self-regulation and the cognitive control system - modulates function in the amygdala [99] to which it is strongly connected. [100] A *32 still-maturing prefrontal cortex exerts less control over the amygdala and has less influence over behavior and emotions than a fully mature prefrontal cortex. [101]

Nucleus Accumbens. The nucleus accumbens, a brain region rich in dopamine, is associated with reward processing. Its primary function is to process responses to a potential reward. [102] Studies show that when making decisions, "relative to children and adults, adolescents show exaggerated activation of the accumbens, in concert with less mature recruitment of top-down prefrontal control." [103] This exaggerated activity is consistent with the tendency of adolescents to overvalue rewards in risk-reward assessment and provides a *33 neurobiological basis for the "increased impulsive and risky behaviors observed during [adolescence]." [104]

The nucleus accumbens, which is found in the ventral striatum, is a "critical node" in the reward related neurocircuitry of the brain, contributing to directing behavior toward appropriate goals by consolidating contextual and goal directed information from other areas of the brain. [105] Developmental studies have shown hyperactivity in the ventral striatum during anticipation of rewards in adolescents, as compared to adults. [106] In parallel with increased reward reactivity in the ventral striatum, these studies have found increased engagement of the regions that support the behavior that leads to the reward. Increased reactivity to rewards paired with increased engagement of response regions can lead to an impulsive reaction in the presence of rewards in adolescence. [107] Such increased reactivity, coupled with other aspects of the developing brain, is *34 thought to potentially contribute to the high rate of risk taking in adolescence. [108]

Dopamine and Serotonin. Dopamine is a neurotransmitter that underlies pleasure and motivation. [109] Around the time of puberty, adolescents experience "a rapid and dramatic increase in dopaminergic activity within the motivational system." [110] Because dopamine plays a critical role in the brain's reward circuitry this increase in activity is likely to promote reward-seeking behavior. [111] At the same time, adolescents have correspondingly lower levels of serotonin, a neurotransmitter known to support inhibitory control. [112] This imbalance between lower levels of serotonin and higher levels of dopamine *35 during adolescence is believed to underlie risky and impulsive decision making by adolescents.

In addition to motivation, dopamine also plays a crucial role in reinforcement learning. Thus, the adolescent period does not only include heightened motivation but also a greater capacity for learning [113] having implications for enhanced amenability for rehabilitation in the adolescent period compared to adulthood.

In sum, adolescent behavior is characterized by a hyperactive reward-driven system (involving the nucleus accumbens and increased dopamine), a limited harm-avoidant system (involving the amygdala), and an immature cognitive control system (involving the prefrontal cortex and decreased serotonin). [114] As a result, adolescent behavior is more likely to be impulsive and motivated by the possibility of reward, with less self-regulation and effective risk assessment. In other words, the adolescent brain is biologically biased to engage in exploring new environments and experiences which can involve taking risks.

Adolescence is a time of great physiological and psychological development. It is also a time marked by impulsive, risky, and sensation- *36 seeking behavior. Scientific research has shed light on the biological mechanisms that help to explain this behavior. And each time this Court has examined the constitutional limitations of imposing severe penalties on juvenile offenders, the scientific research on the development of the adolescent brain has grown. This research establishes that "the brain systems that are crucial for exerting cognitive control over behavior and processing rewards are still immature during adolescence." [115] "These immaturities result in a system that is able to exert cognitive control, but in an inconsistent manner with limited flexibility and motivational control." [116] In other words, "the basic elements

are established, but refinements are needed to support the necessary efficiency in circuit processing to establish reliable executive control." [117] As one researcher put it, the process of adolescent development is akin to "starting the engines without a skilled driver behind the wheel." [118]

*37 CONCLUSION

While not formally supporting either party in these cases, the *amici* hope that the Court will consider the scientific evidence presented here in its deliberations about whether, in the present case, the Eighth Amendment (1) requires that these defendants be held to a different standard of culpability from that which applies to adults and (2) prohibits the imposition of a sentence of life without the possibility of parole on an adolescent offender.

Footnotes

* The parties have consented to the filing of this brief. Pursuant to Rule 37.3(a), letters consenting to the filing of this brief are on file with the Clerk of the Court. No counsel for a party authored this brief in whole or in part, and no counsel or party made a monetary contribution intended to fund the preparation or submission of this brief. No person other than *amici curiae*, their members, or their counsel made a monetary contribution to its preparation or submission.

1 There is a continuum of differences in brain maturation and cognitive abilities between the youngest and oldest of adolescents. All of the scientific conclusions recounted in this brief, however, are applicable to adolescents as a class - ranging from ages 12 to 17.

2 David Dobbs, *Beautiful Brains*, 220:4 Nat'l Geographic 36, 48 (Oct. 2011).

3 Linda Patia Spear, *The Adolescent Brain and Age-Related Behavioral Manifestations*, 24 Neurosci. & Biobehav. Revs. 417, 421 n. 1 (2000); *see also* Lawrence Steinberg et al., *Age Differences in Sensation Seeking and Impulsivity as Indexed by Behavior and Self-Report: Evidence of a Dual Systems Model,* 44:6 Developmental Psychol. 1774 (2008); B.J. Casey et al., *The Adolescent Brain*, 28 Developmental Rev. 62, 62-77 (2008); *see generally* Sarah Durston & B.J. Casey, *What Have We Learned About Cognitive Development from Neuroimaging?,* 44 Neuropsychology 2149 (2006); Luciana Hooper et al., *Adolescents' Performance on the Iowa Gambling Task: Implications for the Development of Decision-Making and Ventromedial Prefrontal Cortex*, 40:6 Developmental Psychol. 1148 (2004).

4 Beatriz Luna, *The Maturation of Cognitive Control and the Adolescent Brain, in* From Attention to Goal-Directed Behavior 250 (Francisco Aboitiz and Diego Cosmelli eds., Springer Berlin Heidelberg 2009) (explaining that "these behaviors may be necessary to develop the social skills needed to gain independence in adulthood").

5 Spear (2000), *supra* note 3, at 421; *see also* Casey (2008), *supra* note 3, at 65 ("[R]isk-taking appears to increase during adolescence relative to childhood and adulthood....")

6 Elizabeth Cauffman & Lawrence Steinberg, *(Im)Maturity of Judgment in Adolescences: Why Adolescents May Be Less Culpable Than Adults*, 18 Behav. Sci. & L. 741, 742 (2000); see *also* William Gardner, *A Life-Span Rational-Choice Theory of Risk Taking, in* Adolescent and Adult Risk Taking: the Eighth Texas Tech Symposium on Interfaces in Psychology 66, 67 (N. Bell & R. Bell eds., 1993).

7 *See* Deborah Yurgelun-Todd, *Emotional and Cognitive Changes During Adolescence*, 17 Current Opinion in Neurobiology 251, 253 (2007); *see also* R. K. Lenroot & Jay N. Giedd, *Brain Development in Children and Adolescents: Insights from Anatomical Magnetic Resonance Imaging*, 30 Neurosci. & Behav. Revs. 718, 723 (2006); Luna (2009), *supra* note 4, at 249, 51; *see also* Lawrence Steinberg et al., *Age Differences in Future Orientation and Delay Discounting*, 80 Child Dev. 28, 40-41 (2009) [hereinafter Steinberg, *Future Orientation*] ("[C]hanges in impulse control and planning are mediated by a 'cognitive control' network ... which matures more gradually and over a longer period of time, into early adulthood.").

8 *See* Luna (2009), *supra* note 4, at 251.

9 *See* Laurence Steinberg, *Adolescent Development and Juvenile Justice*, 16:3 Ann. Rev. Clinical Psychol. 47, 57 (2009) [hereinafter Steinberg, *Adolescent Development]; see also* C.F. Geier, et al., *Immaturities in Reward Processing and Its Influence on Inhibitory Control in Adolescence*, 20:7 Cerebral Cortex 1613, 1624-26 (2010).

10 *See* Steinberg, *Adolescent Development, supra* note 9, at 57-58.

11 *Id.* at 58.

12 Elizabeth Cauffman & Elizabeth Shulman, *Age Differences in Affective Decision Making as Indexed by Performance on the Iowa Gambling Task*, 46:1 Developmental Psychol. 193, 194 (2010); *see also* Steinberg, *Adolescent Development, supra* note 9, at 57.

13 Valerie Reyna & Charles Brainerd, *Dual Processes in Decision Making and Developmental Neuroscience: A Fuzzy-Trace Model*, 31 Developmental Rev. 180, 193 (2011).

14 *See* Susan L. Andersen, *Trajectories of Brain Development: Point of Vulnerability or Window of Opportunity?* 27 Neurosci. and Blobehav. Revs 3, 3-18 (2003); Fulton Crews, Jun He & Clyde Hodge, *Adolescent Cortical Development: A Critical Period of Vulnerability for Addiction*, 86 Pharmacology Biochemistry and Behav. 189 (2007); Spear (2000), *supra* note 3; Cauffman & Shulman, *supra* note 12, at 206; Steinberg (2008), *supra* note 3, at 1776 (linking lack of impulse control to sensation seeking behaviors).

15 Albert Chein, et al., *Peers Increase Adolescent Risk Taking by Enhancing Activity in the Brain's Reward Circuitry*, 14:2 Developmental Sci. F1, F1 (2011) (internal citations omitted); Linda Patia Spear, *Rewards, Aversions and Affect in Adolescence: Emerging Convergences Across Laboratory Animal and Human Data*, 1 Developmental Cognitive Neurosci. 390, 400(2011).

16 *See* Chein, *supra* note 15, at F7. These areas include the ventral striatum and orbitofrontal cortex. *Id.* at F1, F7 ("Specifically, relative to adults, adolescents demonstrated significantly greater activation of VS and OFC as they rendered decisions about risk, but only when they were aware that friends were watching them.").

17 *Id.* at F8.

18 *Id.* at F7.

19 *See* Casey (2008), *supra* note 3, at 64.

20 *See* Luna, *supra* note 4, at 251.

21 *See id.*

22 *See* Steinberg, *Adolescent Development, supra* note 9, at 58; *see also* Laurence Steinberg & Kathryn C. Monahan, *Age Differences in Resistance to Peer Influence*, 43 Developmental Psychol. 1531, 1538 (2007); Steinberg (2008), *supra* note 3, at 1772-74.

23 *See* Casey (2008), *supra* note 3, at 64.

24 *See* Steinberg, *Future Orientation, supra* note 7, at 28-29, 38-40.

25 Steinberg (2008), *supra* note 3, at 1776; *see* Steinberg, *Adolescent Development, supra* note 9, at 57.

26 *See* Sang Hee Kim & Stephan Hamann, *Neural Correlates of Positive and Negative Emotion Regulation*, 19:5 J. Cognitive Neurosci. 776 (2007); Kelly Anne Barnes et al., *Developmental Differences in Cognitive Control of Socio-Affective Processing,* 32:3 Developmental Neuropsychol. 787 (2007).

27 *Id.* at 776.

28 *See* Casey (2008), *supra* note 3, at 65.

29 Isabelle M. Rosso et al., *Cognitive and Emotional Components of Frontal Lobe Functioning in Childhood and Adolescence*, 1021 Annals N.Y. Acad. Sci. 355, 360-61 (2004); *see also, e.g.*, Todd A. Hare et al., *Biological Substrates of Emotional Reactivity and Regulation in Adolescence During an Emotional Go-Nogo Task*, 63:10 Biological Psychiatry 927 (2008) (adolescents show exaggerated responses in subcortical brain regions involved in emotional behaviors, which is associated with risk taking and heightened emotional responses to empty threats).

30 *See* Steinberg (2007), *supra* note 22, at 1536-38 (explaining that "resistance to peer influence increases linearly over the course of adolescence, especially between ages 14 and 18").

31 *See* Casey (2008), *supra* note 3, at 68.

32 See Spear (2000), supra note 3, at 423; Lita Furby & Ruth Beyth-Maron, *Risk Taking in Adolescence: A Decision-Making Perspective*, 12 Developmental Rev. 1, 22 (1992).

33 *See* Spear (2000), *supra* note 3, at 423; Furby, *supra* note 32, at 22.

34 *See* Laurence Steinberg & Elizabeth S. Scott, *Less Guilty by Reason of Adolescence: Developmental Immaturity, Diminished Responsibility, and the Juvenile Death Penalty*, 58 Am. Psychol. 1009, 1011-13 (2003).

35 *See* Spear (2000), *supra* note 3, at 429.

36 *See id.;* Cauffman (2000), *supra* note 6, at 743-45, 756-57, 59.

37 *See* Sarah Durston et al., *Anatomical MRI of the Developing Human Brain: What Have We Learned?* 40 J. Am. Acad. Child & Adolescent Psychiatry 1012, 1012 (2001) (reviewing results of MRI studies of brain development in childhood and adolescence); Michael S. Gazzaniga et al., Cognitive Neuroscience: The Biology Of The Mind 20-21, 138 (2d ed. 2002).

38 *See* Nitin Gogtay et al., *Dynamic Mapping of Human Cortical Development During Childhood Through Early Adulthood*, 101 Proc. Nat'l Acad. Sci. 8174, 8177 (2004).

39 "MRI measures the response of atoms in different tissues when they are pulsed with radio waves that are under the influence of magnetic fields thousands of times the strength of the Earth's. Each type of tissue responds differently, emitting characteristic signals from the nuclei of its cells. The signals are fed into a computer, the position of those atoms is recorded, and a composite picture of the body area being examined is generated and studied in depth." Florence Antoine, *Cooperative Group Evaluating Diagnostic Imaging Techniques,* 81 J. Nat'l Cancer Inst. 1347, 1348 (1989); *see also* Yurgelun-Todd, *supra* note 7, at 251-52 (explaining that "structural MRI and functional MRI (fMRI), have become important modalities for research on brain development as they have been able to provide a more detailed picture of how the brain changes. The application of these methods to the study of children and adolescents provides an extraordinary opportunity to advance our understanding of neurobiological changes and functional abilities associated with the brain.")

40 *See* Gazzaniga, *supra* note 37, at 63.

41 *See generally* Elizabeth R. Sowell et al., *Development of Cortical and Subcortical Brain Structures in Childhood and Adolescence: A Structural MRI Study*, 44 Developmental Med. & Child Neurology 4 (2002); Elizabeth R. Sowell et al., *Mapping Continued Brain Growth and Gray Matter Density Reduction in Dorsal Frontal Cortex: Inverse Relationships During Postadolescent Brain Maturation*, 21 J. Neurosci. 8819 (2001).

42 *See* Elizabeth R. Sowell et al., *In Vivo Evidence for Post-Adolescent Brain Maturation in Frontal and Striatal Regions*, 2 Nature Neurosci. 859 (1999); *see also* Jay N. Giedd et al., *Brain Development During Childhood and Adolescence: A Longitudinal MRI Study*, 2 Nature Neurosci. 861 (1999).

43 Elizabeth R. Sowell et al., *Mapping Cortical Change Across the Human Life Span*, 6 Nature Neurosci. 309 (2003); see *also* Gogtay, *supra* note 38, at 8177.

44 *See* Casey (2008), *supra* note 3, at 68.

45 Sowell (1999), *supra* note 42, at 860; *see* Eveline A. Crone et al., *Neurocognitive Development of Relational Reasoning*, 12:1 Developmental Sci. 55, 56 (2009) (explaining that "[n]europsychological and neuroimaging studies have shown that prefrontal cortex is strongly implicated in relational reasoning."); see also Gazzaniga, supra note 37, at 75; Rosso, *supra* note 29, at 360-61 (finding a correlation between frontal lobe development in adolescents, response inhibition and social anxiety levels); *see generally*, Silvia A. Bunge et al., *Immature Frontal Lobe Contributions to Cognitive Control in Children: Evidence from fMRI*, 33 Neuron 301 (2002).

46 See B.J. Casey et al., *Structural and Functional Brain Development and Its Relation to Cognitive Development,* 54 Biological Psychol. 241, 244 (2000).

47 *See* R. Dias et al., *Dissociable Forms of Inhibitory Control Within Prefrontal Cortex with an Analog of the Wisconsin Card Sort Test: Restriction to Novel Situations and Independence from "On-Line" Processing*, 17 J. Neurosci. 9285 (1997); Durston, *supra* note 37, at 1016; *see also* Yurgelun-Todd, *supra* note 7, at 253.

48 *See* Facundo Manes et al., *Decision-Making Processes Following Damage to the Prefrontal Cortex*, 125 Brain 624 (2002).

49 *See* J. O'Doherty et al., *Abstract Reward and Punishment Representations in the Human Orbitofrontal Cortex*, 4 Nature Neurosci. 95 (2001); Robert D. Rogers et al., *Choosing Between Small, Likely Rewards and Large, Unlikely Rewards Activates Inferior and Orbital Prefrontal Cortex*, 20 J. Neurosci. 9029 (1999).

50 See Antoine Bechara et al., *Characterization of the Decision-Making Deficit of Patients with Ventromedial Prefrontal Cortex Lesions*, 123 Brain 2189, 2198-99 (2000).

51 *See* Samantha B. Wright et al., *Neural Correlates of Fluid Reasoning in Children and Adults*, 1:8 Frontiers Human Neurosci. 7 (2008) (finding that important changes in the prefrontal cortex during adolescence lead to the development of logical reasoning abilities); *see also* Antoine Bechara et al., *Dissociation of Working Memory from Decision Making Within the Human Prefrontal Cortex*, 18 J. Neurosci. 428 (1998).

52 *See* Bechara (2000), *supra* note 50.

53 *See* D. D. Langleben et al., *Brain Activity During Simulated Deception: An Event-Related Functional Magnetic Resonance Study*, 15 NeuroImage 727 (2002).

54 *See* R. Elliott et al., *Differential Neural Response to Positive and Negative Feedback in Planning and Guessing Tasks*, 35 Neuropsychology 1395 (1997).

55 *See* Luna, *supra* note 4, at 264.

56 *See* Jorge Moll et al., *Frontopolar and Anterior Temporal Cortex Activation in a Moral Judgment Task: Preliminary Functional MRI Results in Normal Subjects*, 59 Arq Neuropsiquiatr 657 (2001); Steve W. Anderson et al., *Impairment of Social and Moral Behavior Related to Early Damage in Human Prefrontal Cortex*, 2 Nature Neurosci. 1032 (1999).

57 *See* Gogtay, *supra* note 38, at 8174 (subjects of study aged 4 to 21 years); Giedd (1999), *supra* note 42, at 861 (subjects of study aged 4.2 to 21.6 years); Sowell (1999), *supra* note 42, at 860-61 (subjects of study aged 12 to 16 and 23 to 30 years); *see also* Sowell (2001), *supra* note 41, at 8826 (noting pronounced brain maturational processes continuing into post-adolescence; subjects of study aged 7 to 30 years); Sowell (2003), *supra* note 43, at 309 (subjects of study aged 7 to 87 years).

58 Casey (2000), *supra* note 46, at 243; *see also* Gogtay, *supra* note 38, at 8175.

59 Sowell (1999), *supra* note 42, at 860; *see also* Kenneth E. Towbin & John E. Schowalter, *Adolescent Development, in* Psychiatry 145, 151-52 (Allan Tasman ed., 2d ed. 2003).

This paper recognizes the link between "improvement during adolescence in specific cognitive skills such as organizing information, conceptualization, perspective taking, and social perception, to structural changes in frontal cortical and sub-cortical structures." *Id.* at 152.

60 *See* Steven Petersen et al., *Functional Brain Networks Develop from a "Local to Distributed" Organization*, 5:5 PLoS Computational Biology 1, 8 (2009) (increased connectivity "promote[s] interactions between brain regions ... allowing for a more effective 'solution' to any particular set of processing demands").

61 *See* Gazzaniga, *supra* note 37, at 64-65; *see* Eric R. Kandel et al., Principles Of Neural Science 9 (James H. Schwartz & Thomas M. Jessel, eds., McGraw-Hill 2000).

62 *See* Durston, *supra* note 37, at 1014; Jay N. Giedd et al., *Anatomical Brain Magnetic Resonance Imaging of Typically Developing Children and Adolescents*, 48:5 J. Am. Acad. Child Adolescent Psychiatry 465, 469 (2009); Gogtay, *supra* note 38, at 8174 (10 year study of gray matter loss showed continued gray matter loss until adulthood).

63 *See* Robert F. McGivern et al., *Cognitive Efficiency on a Match to Sample Task Decreases at the Onset of Puberty in Children*, 50 Brain & Cognition 73 (2002) (subjects of study aged 10 to 22 years); Casey, *supra* note 46, at 241 ("findings are consistent with the view that increasing cognitive capacity during childhood coincides with a gradual loss rather than formation of new synapses ..."); *see also* Daniel J. Siegel, The Developing Mind: Toward A Neurobiology Of Interpersonal Experience 13-14 (Guilford Press 1999).

64 *See* Gogtay, *supra* note 38, at 8175.

65 *See generally* Peter R. Huttenlocher, *Synaptic Density in Human Frontal Cortex: Developmental Changes and Effects of Aging*, 163 Brain Res. 195 (1979).

66 *See, e.g.*, Sowell (2002), supra note 41, at 4.

67 *See* McGivern, *supra* note 63, at 85; *see also* David N. Kennedy et al., *Basic Principles of MRI and Morphometry Studies of Human Brain Development*, 5 Developmental Sci. 268, 274 (2002).

Studies showed ... nonlinear changes in cortical gray matter, summarized as a preadolescent increase followed by a postadolescent decrease. Further localization of these changes indicated that the frontal and parietal lobe peaked at about age 12, the temporal lobe at about age 16, and the occipital lobe continued its increase through age 20, although the confidence intervals on these observations are large.

Giedd (1999), *supra* note 42, at 861.

68 *See* Giedd (1999), *supra* note 42, at 861; McGivern, *supra* note 63, at 85; Yurgelun-Todd, *supra* note 7, at 252, 55.

69 *See* Jay N. Giedd, *The Teen Brain: Insights from Neuroimaging*, 42 J. Adolescent Health 335, 339 (2008).

70 A study by the National Academy of Sciences measured gray matter density in individuals longitudinally from childhood to early adulthood and concluded that "the [gray matter] maturation ultimately involves the dorsolateral prefrontal cortex, which loses [gray matter] only at the end of adolescence." Gogtay, *supra* note 38, at 8175.

71 *See id.* at 8177 (explaining that "[l]ater to mature were areas involved in executive function"); *see also* Michael C. Stevens et al., *Functional Neural Networks Underlying Response Inhibition in Adolescents and Adults*, 181 Behav. Brain Research 12 (2007).

72 *See* Elkhonon Goldberg, The Executive Brain: Frontal Lobes & The Civilized Mind 144 (Oxford Univ. Press, 2001); *see also* Sowell (2001), *supra* note 41, at 8819; Sowell (2003), *supra* note 43, at 311; Yurgelun-Todd, *supra* note 7, at 253.

73 *See* Zoltan Nagy, Helena Westerberg & Torkel Klingberg, *Maturation of White Matter is Associated with the Development of Cognitive Functions During Childhood*, 16:7 J. Cognitive Neurosci. 1227, 1231-32 (2004) (explaining that "the physiological effects of increases in axon thickness and myelination are similar in that they both increase conduction speed."); Gazzaniga, *supra* note 36, at 31, 48-49.

74 Goldberg, *supra* note 72, at 144.

75 White matter is the tissue that composes the pathways between brain regions and that permits communication and interaction within the brain and between the brain and the body. *See* Gazzaniga, *supra* note 37, at 70, 72. For example, the corpus callosum, a critical white matter structure, bridges the two halves of the frontal lobes, permitting and regulating communication between the two halves of the brain. *See* Tomas Paus et al., *Structural Maturation of Neural Pathways in Children and Adolescents: In Vivo Study*, 283 Science 1908 (1999).

76 M. R. Asato et al., *White Matter Development in Adolescence: A DTI Study*, 20:9 Cerebral Cortex 2122, 2125 (2010) ("In agreement with other studies, we found evidence for continuing maturation of white matter throughout distributed brain regions from childhood into adulthood.") (internal citations omitted); *see* Nagy, Westerberg & Klingberg, *supra* note 73, *at* 1231-32; Durston, *supra* note 37, at 1014; Sowell (1999), *supra* note 42, at 860; Adolf Pfefferbaum et al., *A Quantitative Magnetic Resonance Imaging Study of Changes in Brain Morphology from Infancy to Late Adulthood*, 51 Archives Of Neurology 874, 885 (1994) (after age 20 white matter volume did not fluctuate until about age 70; subjects of study aged 3 months to 70 years).

77 *See* Sowell (2003), *supra* note 43, at 311; Sowell (2002), *supra* note 41, at 4; Towbin & Schowalter, *supra* note 59, at 151.

78 *See* Paus, *supra* note 75, at 1908.

79 *See* Gogtay, *supra* note 38 at 8177 (noting that different parts of the brain undergo myelination and pruning at different rates, and finding that the higher-order cortices mature later than lower-order cortices."); *see also* Sowell (1999), *supra* note 42, at 859; K. Rubia et al., *Functional Frontalisation with Age: Mapping Neurodevelopmental Trajectories with fMRI*, 24 Neurosci. & Blobehav. Revs. 13 (2000) (subjects of study aged 12 to 19 and 22 to 40 years).

80 *See* Luna (2009), *supra* note 4, at 257.

81 *See id.; see also* Giedd (2009), *supra* note 62, at 467.

82 *See* Steinberg, *Adolescent Development, supra* note 9, at 56; Beatriz Luna & John A. Sweeney, *The Emergence of Collaborative Brain Function: fMRI Studies of the Development of Response Inhibition*, 1021 Annals N.Y. Acad. Sci. 296, 296309 (2004); Damien A. Fair et al., *Development of Distinct Control Networks Through Segregation and Integration*, 104 Proc. Nat'l Acad. Sci. U.S. 13507 (2007).

83 *See* Steinberg, *Adolescent Development, supra* note 9, at 56.

84 *See* Nagy, Westerberg & Klingberg, *supra* note 73, at 1231-32.

85 Kai Hwang et al., *Strengthening of Top-Down Frontal Cognitive Control Networks Underlying the Development of Inhibitory Control: A Functional Magnetic Resonance Imaging Effective Connectivity Study*, 30:46 J. Neurosci. 15535, 15543 (2010).

86 *Id.* at 15542.

87 *Id.;* Nico Dosenbach et al., *Prediction of Individual Brain Maturity Using fMRI*, 329 Science 1358, 1360-61 (2010) (brain continues to mature until 22 years of age, with region of brain most highly correlated to brain maturity was pre-frontal cortex).

88 Asato, *supra* note 76 at 2128; Petersen, *supra* note 60, at 8.

89 Asato, *supra* note 76, at 2128; *see* Spear (2011), *supra* note 15, at 391 (top-down control gradually gains a "competitive edge" over "'bottom-up' systems that express exaggerated reactivity to motivational stimuli").

90 *See* Steinberg, *Adolescent Development, supra* note 9, at 54.

91 Stephanie Burnett et al., *Development During Adolescence of the Neural Processing of Social Emotion*, 21:9 J. Cognitive Neurosci. 173 (2009); S. J. Blakemore, *Adolescent Development of the Neural Circuitry for Thinking About Intentions*, 2:2 Soc. Cognitive & Affective Neurosci. 130 (2007).

92 Amy L. Krain et al., *An fMRI Examination of Developmental Differences in the Neural Correlates of Uncertainty and Decision Making*, 47:10 J. Child Psychol. & Psychiatry 1023, 1024 (2006); *see also* Liston C. Watts et al., *Frontostriatal Microstructure Predicts Individual Differences in Cognitive Control*, 16:4 Cerebral Cortex 553 (2006); B.J. Casey et al., *Contribution of Frontostriatal Fiber Tracts to Cognitive Control in Parent-Child Dyads with ADHD*, 164:11 Am. J. Psychiatry 1729 (2007).

93 Krain, *supra* note 92; *see also* Adriana Galvan et al., *Earlier Development of the Accumbens Relative to Orbitofrontal Cortex Might Underlie Risk-Taking Behavior in Adolescents*, 26:25 J. Neurosci. 6885 (2006); *see* Hare, *supra* note 29.

94 *See* Steinberg, Adolescent Development, *supra* note 9, at 54; *see also* Monique Ernst et al., *Neurobiology of the Development of Motivated Behaviors in Adolescence: A Window into a Neural Systems Model*, 93 Pharmacology, Biochemistry & Behav. 199 (2009).

95 *See generally* Jan Gläscher & Ralph Adolphs, *Processing of the Arousal of Subliminal and Supraliminal Emotional Stimuli by the Human Amygdala*, 23 J. Neurosci. 10274 (2003); Ralph Adolphs, *Neural Systems for Recognizing Emotion*, 12 Current Opinion In Neurobio. 169 (2002); Gazzaniga, *supra* note 37, at 553-72; K. Luan Phan et al., *Functional Neuroanatomy of Emotion: A Meta-Analysis of Emotion Activation Studies in PET and fMRI*, 16 Neuroimage 331, 336 (2002); Goldberg, *supra* note 72, at 31; Kevin S. LaBar et al., *Human Amygdala Activation During Conditioned Fear Acquisition and Extinction: A Mixed-Trial fMRI Study*, 20 Neuron 937 (1998); Richard D. Lane et al., *Neuroanatomical Correlates of Pleasant and Unpleasant Emotion*, 35 Neuropsychology 1437, 1441 (1997); Hans C. Breiter et al., *Response and Habituation of the Human Amygdala During Visual Processing of Facial Expression*, 17 Neuron 875 (1996); Steinberg, *Future Orientation, supra* note 7, at 40.

96 Abigail A. Baird et al., *Functional Magnetic Resonance Imaging of Facial Affect Recognition in Children and Adolescents*, 38 J. Am. Acad. Child & Adolescent Psychiatry 1, 1 (1999) (study found that adolescents 12-17 years old showed significant

amygdala activation in response to a task that required the judgment of fearful facial affect); *see also* William D.S. Killgore & Deborah Yurgelun-Todd, *Activation of the Amygdala and Anterior Cingulate During Nonconscious Processing of Sad Versus Happy Faces*, 21 Neuroimage 1215 (2004); Phan, *supra* note 95, at 336.

97 *See* Goldberg, *supra* note 76, at 31; Phan, *supra* note 95, at 336.

98 *See* Giedd (2008), *supra* note 69, at 338.

99 *See* Mario Beauregard et al., *Neural Correlates of Conscious Self-Regulation of Emotion*, 21 J. Neurosci. 165RC (2001); Ahmad Hariri et al., *Modulating Emotional Responses: Effects of a Neocortical Network on the Limbic System*, 11 NeuroReport 43 (2000).

100 Ralph Adolphs, *The Human Amygdala and Emotion*, 5 Neuroscientist 125, 125-26 (1999); *see also* Joseph LeDoux, The Emotional Brain: The Mysterious Underpinnings Of Emotional Life 303 (1996).

101 *See* Neir Eshel et al., *Neural Substrates of Choice in Adults and Adolescents: Development of the Ventrolateral Prefrontal and Anterior Cingulate Cortices*, 45 Neuropsychology 1270, 1270-71 (2007) (reporting prefrontal brain areas associated with higher-order cognition, emotional regulation, reward values, and behavior control are some of the last to mature and that this lag in maturation may explain why adolescents demonstrate poor decision-making); *see also* Gargi Talukder, *Decision-Making Is Still a Work in Progress for Teenagers,* Report dated July 2000 at http://www.brainconnection.com; *see also* Spear (2000), *supra* note 3, at 440 (reporting Dr. YurgelunTodd's research); *see also* Ralph Adolphs et al., *Fear and the Human Amygdala*, 15 J. Neurosci. 5879, 5889 (1995).

102 Galvan, *supra* note 93, at 6890.

103 *See* Casey, *supra* note 3, at 69.

104 *See id.* at 69-70.

105 Spear (2011), *supra* note 15, at 392; *see* Chein, *supra* note 15, at F1 (the ventral striatum is part of the "incentive processing system in the brain").

106 *See* Geier, *supra* note 9, at 1625; Aarthi Padmanabhan et al., *Developmental Changes in Brain Function Underlying the Influence of Reward Processing on Inhibitory Control*, 1 Developmental Cognitive Neuroscience 517, 526 (2011), Spear (2011); *supra* note 15, at 394 (adolescents have been reported by a number of groups to show heightened activation of the ventral striatum during *receipt* of rewards relative to younger and/or older individuals").

107 Geier, *supra* note 9, at 1626; Padmanabhan, *supra* note 106, at 523.

108 Padmanabhan, *supra* note 106, at 527; Chein, *supra* note 15, at F1 ("Many research groups ... have posited that adolescents' relatively greater propensity toward risky behavior" is based in part on the "*incentive processing* system involving the ventral striatum") (emphasis in original).

109 *See* Andersen, *supra* note 14, at 3-18; Crews, He & Hodge, *supra* note 14, at 189-99; Spear (2000), *supra* note 3, at 417-63.

110 Steinberg, *Adolescent Development, supra* note 9, at 54.

111 *Id.* at 258; *see* Luna, *supra* note 4, at 258. Moreover, "[t]here is evidence that changes in the density and distribution of receptors for dopamine ... within regions critical to incentive processing take place around the time of puberty, and that these changes coincide with a dramatic elevation in the salience of peer interactions." Chein *supra* note 15, at F8.

112 *See* Luna, *supra* note 4, at 258; R. Andrew Chambers, Jane R. Taylor & Marc N. Potenza, *Developmental Neurocircuitry of Motivation in Adolescence: A Critical Period of Addiction Vulnerability*, 160 Am. J. Psychiatry 1041 (2003).

113 Dustin Wahlstrom et al., *Neurobehavioral Evidence for Changes in Dopamine System Activity During Adolescence*, 34 Neuroscience Biobehavioral Rev. 631, 643 (2010).

114 Monique Ernst et al., *Triadic Model of the Neurobiology of Motivated Behavior in Adolescence*, 36 Psychol. Med. 299, 300-302 (2006).

115 *See* Luna, *supra* note 4, at 258; *see also* Ryan L. Muetzel et al., *The Development of Corpus Callosum Microstructure and Associations with Bimanual Task Performance in Healthy Adolescents*, 39:4 Neuroimage 1918 (2008); Elizabeth A. Olson, *White Matter Integrity Predicts Delay Discounting Behavior in Adolescents: A Diffusion Tensor Imaging Study*, 21:7 J. Cognitive Neurosci. 1406 (2008); Elizabeth A. Olson, *Delay and Probability Discounting Behavior in Healthy Adolescents: Associations with Age, Personality Style, and Other Measures of Executive Function*, 43:7 Personality And Individual Differences 1886 (2007).

116 *See* Luna, *supra* note 4, at 258.

117 *Id.*

118 Steinberg, *Adolescent Development, supra* note 9, at 56.

End of Document

76 M. R. Asato et al., *White Matter Development in Adolescence: A DTI Study*, 20:9 Cerebral Cortex 2122, 2125 (2010) ("In agreement with other studies, we found evidence for continuing maturation of white matter throughout distributed brain regions from childhood into adulthood.") (internal citations omitted); *see* Nagy, Westerberg & Klingberg, *supra* note 73, *at* 1231-32; Durston, *supra* note 37, at 1014; Sowell (1999), *supra* note 42, at 860; Adolf Pfefferbaum et al., *A Quantitative Magnetic Resonance Imaging Study of Changes in Brain Morphology from Infancy to Late Adulthood*, 51 Archives Of Neurology 874, 885 (1994) (after age 20 white matter volume did not fluctuate until about age 70; subjects of study aged 3 months to 70 years).

77 *See* Sowell (2003), *supra* note 43, at 311; Sowell (2002), *supra* note 41, at 4; Towbin & Schowalter, *supra* note 59, at 151.

78 *See* Paus, *supra* note 75, at 1908.

79 *See* Gogtay, *supra* note 38 at 8177 (noting that different parts of the brain undergo myelination and pruning at different rates, and finding that the higher-order cortices mature later than lower-order cortices."); *see also* Sowell (1999), *supra* note 42, at 859; K. Rubia et al., *Functional Frontalisation with Age: Mapping Neurodevelopmental Trajectories with fMRI*, 24 Neurosci. & Blobehav. Revs. 13 (2000) (subjects of study aged 12 to 19 and 22 to 40 years).

80 *See* Luna (2009), *supra* note 4, at 257.

81 *See id.; see also* Giedd (2009), *supra* note 62, at 467.

82 *See* Steinberg, *Adolescent Development, supra* note 9, at 56; Beatriz Luna & John A. Sweeney, *The Emergence of Collaborative Brain Function: fMRI Studies of the Development of Response Inhibition*, 1021 Annals N.Y. Acad. Sci. 296, 296309 (2004); Damien A. Fair et al., *Development of Distinct Control Networks Through Segregation and Integration*, 104 Proc. Nat'l Acad. Sci. U.S. 13507 (2007).

83 *See* Steinberg, *Adolescent Development, supra* note 9, at 56.

84 *See* Nagy, Westerberg & Klingberg, *supra* note 73, at 1231-32.

85 Kai Hwang et al., *Strengthening of Top-Down Frontal Cognitive Control Networks Underlying the Development of Inhibitory Control: A Functional Magnetic Resonance Imaging Effective Connectivity Study*, 30:46 J. Neurosci. 15535, 15543 (2010).

86 *Id.* at 15542.

87 *Id.;* Nico Dosenbach et al., *Prediction of Individual Brain Maturity Using fMRI*, 329 Science 1358, 1360-61 (2010) (brain continues to mature until 22 years of age, with region of brain most highly correlated to brain maturity was pre-frontal cortex).

88 Asato, *supra* note 76 at 2128; Petersen, *supra* note 60, at 8.

89 Asato, *supra* note 76, at 2128; *see* Spear (2011), *supra* note 15, at 391 (top-down control gradually gains a "competitive edge" over "'bottom-up' systems that express exaggerated reactivity to motivational stimuli").

90 *See* Steinberg, *Adolescent Development, supra* note 9, at 54.

91 Stephanie Burnett et al., *Development During Adolescence of the Neural Processing of Social Emotion*, 21:9 J. Cognitive Neurosci. 173 (2009); S. J. Blakemore, *Adolescent Development of the Neural Circuitry for Thinking About Intentions*, 2:2 Soc. Cognitive & Affective Neurosci. 130 (2007).

92 Amy L. Krain et al., *An fMRI Examination of Developmental Differences in the Neural Correlates of Uncertainty and Decision Making*, 47:10 J. Child Psychol. & Psychiatry 1023, 1024 (2006); *see also* Liston C. Watts et al., *Frontostriatal Microstructure Predicts Individual Differences in Cognitive Control*, 16:4 Cerebral Cortex 553 (2006); B.J. Casey et al., *Contribution of Frontostriatal Fiber Tracts to Cognitive Control in Parent-Child Dyads with ADHD*, 164:11 Am. J. Psychiatry 1729 (2007).

93 Krain, *supra* note 92; *see also* Adriana Galvan et al., *Earlier Development of the Accumbens Relative to Orbitofrontal Cortex Might Underlie Risk-Taking Behavior in Adolescents*, 26:25 J. Neurosci. 6885 (2006); *see* Hare, *supra* note 29.

94 *See* Steinberg, Adolescent Development, *supra* note 9, at 54; *see also* Monique Ernst et al., *Neurobiology of the Development of Motivated Behaviors in Adolescence: A Window into a Neural Systems Model*, 93 Pharmacology, Biochemistry & Behav. 199 (2009).

95 *See generally* Jan Gläscher & Ralph Adolphs, *Processing of the Arousal of Subliminal and Supraliminal Emotional Stimuli by the Human Amygdala*, 23 J. Neurosci. 10274 (2003); Ralph Adolphs, *Neural Systems for Recognizing Emotion*, 12 Current Opinion In Neurobio. 169 (2002); Gazzaniga, *supra* note 37, at 553-72; K. Luan Phan et al., *Functional Neuroanatomy of Emotion: A Meta-Analysis of Emotion Activation Studies in PET and fMRI*, 16 Neuroimage 331, 336 (2002); Goldberg, *supra* note 72, at 31; Kevin S. LaBar et al., *Human Amygdala Activation During Conditioned Fear Acquisition and Extinction: A Mixed-Trial fMRI Study*, 20 Neuron 937 (1998); Richard D. Lane et al., *Neuroanatomical Correlates of Pleasant and Unpleasant Emotion*, 35 Neuropsychology 1437, 1441 (1997); Hans C. Breiter et al., *Response and Habituation of the Human Amygdala During Visual Processing of Facial Expression*, 17 Neuron 875 (1996); Steinberg, *Future Orientation, supra* note 7, at 40.

96 Abigail A. Baird et al., *Functional Magnetic Resonance Imaging of Facial Affect Recognition in Children and Adolescents*, 38 J. Am. Acad. Child & Adolescent Psychiatry 1, 1 (1999) (study found that adolescents 12-17 years old showed significant

amygdala activation in response to a task that required the judgment of fearful facial affect); *see also* William D.S. Killgore & Deborah Yurgelun-Todd, *Activation of the Amygdala and Anterior Cingulate During Nonconscious Processing of Sad Versus Happy Faces*, 21 Neuroimage 1215 (2004); Phan, *supra* note 95, at 336.

97 *See* Goldberg, *supra* note 76, at 31; Phan, *supra* note 95, at 336.

98 *See* Giedd (2008), *supra* note 69, at 338.

99 *See* Mario Beauregard et al., *Neural Correlates of Conscious Self-Regulation of Emotion*, 21 J. Neurosci. 165RC (2001); Ahmad Hariri et al., *Modulating Emotional Responses: Effects of a Neocortical Network on the Limbic System*, 11 NeuroReport 43 (2000).

100 Ralph Adolphs, *The Human Amygdala and Emotion*, 5 Neuroscientist 125, 125-26 (1999); *see also* Joseph LeDoux, The Emotional Brain: The Mysterious Underpinnings Of Emotional Life 303 (1996).

101 *See* Neir Eshel et al., *Neural Substrates of Choice in Adults and Adolescents: Development of the Ventrolateral Prefrontal and Anterior Cingulate Cortices*, 45 Neuropsychology 1270, 1270-71 (2007) (reporting prefrontal brain areas associated with higher-order cognition, emotional regulation, reward values, and behavior control are some of the last to mature and that this lag in maturation may explain why adolescents demonstrate poor decision-making); *see also* Gargi Talukder, *Decision-Making Is Still a Work in Progress for Teenagers,* Report dated July 2000 at http://www.brainconnection.com; *see also* Spear (2000), *supra* note 3, at 440 (reporting Dr. YurgelunTodd's research); *see also* Ralph Adolphs et al., *Fear and the Human Amygdala*, 15 J. Neurosci. 5879, 5889 (1995).

102 Galvan, *supra* note 93, at 6890.

103 *See* Casey, *supra* note 3, at 69.

104 *See id.* at 69-70.

105 Spear (2011), *supra* note 15, at 392; *see* Chein, *supra* note 15, at F1 (the ventral striatum is part of the "incentive processing system in the brain").

106 *See* Geier, *supra* note 9, at 1625; Aarthi Padmanabhan et al., *Developmental Changes in Brain Function Underlying the Influence of Reward Processing on Inhibitory Control*, 1 Developmental Cognitive Neuroscience 517, 526 (2011), Spear (2011); *supra* note 15, at 394 (adolescents have been reported by a number of groups to show heightened activation of the ventral striatum during *receipt* of rewards relative to younger and/or older individuals").

107 Geier, *supra* note 9, at 1626; Padmanabhan, *supra* note 106, at 523.

108 Padmanabhan, *supra* note 106, at 527; Chein, *supra* note 15, at F1 ("Many research groups ... have posited that adolescents' relatively greater propensity toward risky behavior" is based in part on the "*incentive processing* system involving the ventral striatum") (emphasis in original).

109 *See* Andersen, *supra* note 14, at 3-18; Crews, He & Hodge, *supra* note 14, at 189-99; Spear (2000), *supra* note 3, at 417-63.

110 Steinberg, *Adolescent Development, supra* note 9, at 54.

111 *Id.* at 258; *see* Luna, *supra* note 4, at 258. Moreover, "[t]here is evidence that changes in the density and distribution of receptors for dopamine ... within regions critical to incentive processing take place around the time of puberty, and that these changes coincide with a dramatic elevation in the salience of peer interactions." Chein *supra* note 15, at F8.

112 *See* Luna, *supra* note 4, at 258; R. Andrew Chambers, Jane R. Taylor & Marc N. Potenza, *Developmental Neurocircuitry of Motivation in Adolescence: A Critical Period of Addiction Vulnerability*, 160 Am. J. Psychiatry 1041 (2003).

113 Dustin Wahlstrom et al., *Neurobehavioral Evidence for Changes in Dopamine System Activity During Adolescence*, 34 Neuroscience Biobehavioral Rev. 631, 643 (2010).

114 Monique Ernst et al., *Triadic Model of the Neurobiology of Motivated Behavior in Adolescence*, 36 Psychol. Med. 299, 300-302 (2006).

115 *See* Luna, *supra* note 4, at 258; *see also* Ryan L. Muetzel et al., *The Development of Corpus Callosum Microstructure and Associations with Bimanual Task Performance in Healthy Adolescents*, 39:4 Neuroimage 1918 (2008); Elizabeth A. Olson, *White Matter Integrity Predicts Delay Discounting Behavior in Adolescents: A Diffusion Tensor Imaging Study*, 21:7 J. Cognitive Neurosci. 1406 (2008); Elizabeth A. Olson, *Delay and Probability Discounting Behavior in Healthy Adolescents: Associations with Age, Personality Style, and Other Measures of Executive Function*, 43:7 Personality And Individual Differences 1886 (2007).

116 *See* Luna, *supra* note 4, at 258.

117 *Id.*

118 Steinberg, *Adolescent Development, supra* note 9, at 56.

End of Document

2012 WL 174239 (U.S.) (Appellate Brief)
Supreme Court of the United States.

Evan MILLER, Petitioner,
v.
STATE OF ALABAMA, Respondent.
Kuntrell JACKSON, Petitioner,
v.
Ray HOBBS, Respondent.

Nos. 10-9646, 10-9647.
January 17, 2012.

ON WRITS OF CERTIORARI TO THE ALABAMA COURT OF CRIMINAL APPEALS AND THE ARKANSAS SUPREME COURT

Brief for the American Psychological Association, American Psychiatric Association, and National Association of Social Workers as Amici Curiae in Support of Petitioners

Nathalie F.P. Gilfoyle
General Counsel
American Psychological Association
750 First Street, N.E.
Washington, D.C. 20002
(202) 336-5500

David W. Ogden
Danielle Spinelli
Counsel of Record
Eric F. Citron
Madhu Chugh
Wilmer Cutler Pickering Hale and Dorr LLP
1875 Pennsylvania Ave., N.W.
Washington, D.C. 20006
(202) 663-6000
danielle.spinelli@wilmerhale.com
Counsel for Amicus Curiae American Psychological Association

Aaron M. Panner
Kellogg, Huber, Hansen, Todd, Evans & Figel PLLC
Sumner Square
1615 M Street, N.W., Suite 400
Washington, D.C. 20036
(202) 326-7900
Counsel for Amicus Curiae
American Psychiatric Association

Carolyn I. Polowy

General Counsel
Sherri Morgan
Associate Counsel
National Association of Social Workers
750 First Street, N.E., Suite 700
Washington, D.C. 20002
(202) 336-8600
Counsel for Amicus Curiae
National Association of Social Workers

*i TABLE OF CONTENTS

*ii TABLE OF AUTHORITIES

CASES

OTHER AUTHORITIES

*iii

*1 INTEREST OF AMICI CURIAE [1]

The American Psychological Association is a voluntary nonprofit scientific and professional organization with more than 150,000 members and affiliates. Since 1892, the Association has been the principal organization of psychologists in the United States. Its membership includes the vast majority of U.S. psychologists holding doctoral degrees from accredited universities. [2]

An integral part of the Association's mission is to increase and disseminate knowledge regarding human behavior and to advance psychology as a science, profession, and means of promoting health, education, and human welfare. Based on the well-developed body of research distinguishing the developmental characteristics of juveniles from those of adults, the Association has endorsed the policy reflected in the United Nations Convention on the Rights of the Child, which rejects life imprisonment without possibility of parole for offenses committed by individuals under 18 years of age.

*2 The American Psychiatric Association, with roughly 35,000 members, is the principal association of physicians who specialize in psychiatry. It has an interest in this Court's understanding of the lessons of scientific study and professional experience as the Court applies constitutional principles to individuals who often are patients of the organization's members.

The National Association of Social Workers (NASW) is the largest association of professional social workers in the world, with nearly 145,000 members and 56 chapters throughout the United States and abroad. NASW conducts research, publishes books and studies, promulgates professional criteria, and develops policy statements on relevant issues of importance. NASW opposes any legislation or prosecutorial discretion permitting children to be charged and punished under adult standards.

INTRODUCTION AND SUMMARY OF ARGUMENT

In *Graham v. Florida*, 130 S. Ct. 2011 (2010), this Court held that the Eighth Amendment prohibited life sentences without the possibility of parole for juveniles convicted of non-homicide offenses. The special characteristics of juveniles that this Court identified in *Graham* - and that are supported by a large and growing body of research - apply equally to juveniles convicted of homicide offences.

In *Graham*, this Court reiterated the critical differences between juveniles and adults that it set out in *Roper v. Simmons*, 543 U.S. 551 (2005) - differences that do not absolve juveniles of responsibility for their crimes, but that do reduce their culpability and undermine any justification for definitively ending their free lives. The Court noted that juveniles lack adults' capacity ***3** for mature judgment; that they are more vulnerable to negative external influences; and that their characters are not yet fully formed. *Graham*, 130 S. Ct. at 2026-2027; *Roper*, 543 U.S. at 569-570, 573. "The susceptibility of juveniles to immature and irresponsible behavior means 'their irresponsible conduct is not as morally reprehensible as that of an adult.' " *Roper*, 543 U.S. at 570. Juveniles' vulnerability and lack of control over their surroundings "mean juveniles have a greater claim than adults to be forgiven for failing to escape negative influences in their ... environment." *Id.* And "[j]uveniles are more capable of change than are adults," meaning that "their actions are less likely to be evidence of 'irretrievably depraved character,' " even in the case of very serious crimes. *Graham*, 130 S. Ct. at 2026-2027; ***see*** *Roper*, 543 U.S. at 570. Accordingly, "[t]he juvenile should not be deprived of the opportunity to achieve maturity of judgment and self-recognition of human worth and potential" - with "no chance to leave prison before life's end" - because "[m]aturity can lead to that considered reflection which is the foundation for remorse, renewal, and rehabilitation." *Graham*, 130 S. Ct. at 2032.

As was true in *Graham*, "[n]o recent data provide reason to reconsider the Court's observations in *Roper* about the nature of juveniles." 130 S. Ct. at 2026. Rather, "developments in psychology and brain science continue to show fundamental differences between juvenile and adult minds." *Id.* In fact, an ever-growing body of research in developmental psychology and neuroscience continues to confirm and strengthen the Court's conclusions. Compared to adults, juveniles are less able to restrain their impulses and exercise self-control; less capable of considering alternative courses of action and avoiding unduly risky behaviors; and less ***4** oriented to the future and thus less attentive to the consequences of their often-impulsive actions. Research also continues to demonstrate that "juveniles are more vulnerable or susceptible to negative influences and outside pressures, including peer pressure," while at the same time they lack the freedom and autonomy that adults possess to escape such pressures. *Roper*, 543 U.S. at 569. Thus, even after their general cognitive abilities approximate those of adults, juveniles are less capable than adults of mature judgment and decision-making, especially in the social contexts in which criminal behavior is most likely to arise.

Moreover, because juveniles are still in the process of forming coherent identities, adolescent crime often reflects the "signature" - and transient - "qualities of youth" itself, *Roper*, 543 U.S. at 570, rather than an entrenched bad character. Research into adolescent development continues to confirm the law's intuition that " 'incorrigibility is inconsistent with youth.' " *Graham*, 130 S. Ct. at 2029. And although some youthful offenders will develop into criminal adults, it remains essentially impossible "even for expert psychologists to differentiate between the juvenile offender whose crime reflects unfortunate yet transient immaturity, and the rare juvenile offender whose crime reflects irreparable corruption." *Roper*, 543 U.S. at 573. As *Roper* recognized, that is true even of juvenile offenders who have committed the most serious crimes.

Recent neuroscience research suggests a possible physiological basis for these recognized developmental characteristics of adolescence. It is increasingly clear that adolescent brains are not yet fully mature in regions and systems related to higher-order executive functions such as impulse control, planning ahead, and risk avoidance. That anatomical and functional immaturity is consonant ***5** with juveniles' demonstrated psychosocial (that is, social and emotional) immaturity. During puberty, juveniles evince a rapid increase in reward- and sensation-seeking behavior that declines progressively throughout late adolescence and young adulthood. This effect is amplified by exposure to peers, and it corresponds with significant changes in certain elements of the brain's "incentive processing system" - especially the parts that process rewards and social cues. By contrast, the ability to resist emotional impulses and regulate behavior develops gradually throughout adolescence, and that behavioral development corresponds with gradual development of the brain structures and systems most involved in executive function and impulse control. The disjunction between these developmental processes - which is greatest in early and middle adolescence and narrows as individuals mature into young adulthood - is consistent with the familiar features of adolescence that this Court recognized in *Roper* and *Graham*.

In short, research continues to confirm and expand upon the fundamental insight underlying this Court's previous decisions: Juveniles' profound differences from adults undermine the possible penological justifications for punishing a juvenile offender with a sentence that "guarantees he will die in prison without any meaningful opportunity to obtain release." *Graham*, 130 S. Ct. at 2033. Nor does the scientific literature provide any reason to distinguish between homicide and non-homicide convictions in this regard. In either case, the signature qualities of adolescence reduce juveniles' culpability and increase their capacity for change. Condemning an immature, vulnerable, and not-yet-fully-formed adolescent to live every remaining day of his life in prison - whatever his crime - is thus a constitutionally disproportionate punishment.

*6 ARGUMENT

I. Research In Developmental Psychology And Neuroscience Documents Juveniles' Greater Immaturity, Vulnerability, And Changeability

In *Roper* and *Graham*, this Court concluded that "marked and well understood" developmental differences between juveniles and adults both diminish juveniles' blameworthiness for their criminal acts and enhance their prospects of change and reform. [3] *Roper*, 543 U.S. at 572. Current research continues to reinforce that conclusion, confirming that the three developmental characteristics of juveniles that this Court has identified - their immaturity, their vulnerability, and their changeability - render them, as a group, very different from adults. As this Court has recognized, those differences are central to the calculus of culpability and the proportionality of punishments imposed on juvenile offenders.

*7 A. Juveniles Are Less Capable Of Mature Judgment Than Adults

As this Court has recognized, adolescents have less capacity for mature judgment than adults, and as a result are more likely to engage in risky behaviors. "[A]s any parent knows and as ... scientific and sociological studies ... tend to confirm, '[a] lack of maturity and an underdeveloped sense of responsibility are found in youth more often than in adults and are more understandable among the young. These qualities often result in impetuous and ill-considered actions and decisions.' " *Roper*, 543 U.S. at 569.

As this Court noted in *Roper*, " 'adolescents are overrepresented statistically in virtually every category of reckless behavior.' " 543 U.S. at 569. Indeed, such behavior is "virtually a normative characteristic of adolescent development." [4] Juveniles' risky behavior frequently includes criminal activity; in fact, "numerous rigorous self-report studies have ... documented that it is statistically aberrant to refrain from crime during adolescence." [5] Both violent crimes and less serious offenses "peak sharply" in adolescence and "drop precipitously *8 in young adulthood." [6] This "age-crime curve" is "[o]ne of the most consistent findings across studies." [7]

Adolescents' striking tendency to engage in risky and even illegal behavior stems in part from their lesser capacity for mature judgment. Research has shown that adolescents' judgment and decision-making differ from adults' in several respects: Adolescents are less able to control their impulses; they weigh the risks and rewards of possible conduct differently; and they are less able to envision the future and apprehend the consequences of their actions. Even older adolescents who have developed general cognitive capacities similar to those of adults show deficits in these aspects of social and emotional maturity. [8]

1. Empirical research confirms that adolescents are less capable of self-regulation than adults and, accordingly, are less able to resist their social and emotional impulses. For example, one study of maturity of judgment found that adolescents, including 17-year-olds, scored significantly lower than adults on measures of "temperance," which included "impulse control" and *9 "suppression of aggression." [9] More recent studies confirm this result. In one example,

researchers examined differences in impulsivity between ages 10 and 30, using both self-report and performance measures, and concluded that impulsivity declined throughout the relevant period, with "gains in impulse control occur[ring] throughout adolescence" and into young adulthood. [10] In short, "adults tend to make more adaptive decisions than adolescents," in part because "they have a more mature capacity to resist the pull of social and emotional influences and remain focused on long-term goals." [11]

As explained below, *infra* pp. 25-31, researchers have an increasingly well-developed understanding of aspects of the adolescent brain that may help explain this relative deficit in mature self-control. It is now well-established that the brain continues to develop throughout adolescence and young adulthood in precisely the areas and systems that are regarded as most ***10** involved in impulse control, planning, and self-regulation. But juveniles also lack experience navigating the changing social and environmental contexts, and regulating the new emotional pressures, of adolescence. *See Roper*, 543 U.S. at 569. "[T]he developing adolescent can only learn his or her way to fully developed control by experience," and that "process will probably not be completed until very late in the teen years." [12] Thus, "expecting the experience-based ability to resist impulses ... to be fully formed prior to age eighteen or nineteen would seem on present evidence to be wishful thinking." [13]

2. Adolescents not only struggle to regulate their behavior in response to their emotional impulses, but also respond differently to perceptions of risk and reward. "In general, adolescents use a risk-reward calculus that places relatively less weight on risk, in relation to reward, than that used by adults." [14] For example, one study comparing adolescent and adult decisionmaking found that, when asked to evaluate hypothetical decisions, adolescents as old as 17 were less likely ***11** than adults to mention possible long-term consequences, to evaluate both risks and benefits, and to examine possible alternative options. [15] Similarly, a recent study that employed a gambling task to measure reward-seeking and risk-avoidance behavior in a group of more than 900 individuals aged 10 to 30 found that "adolescents and adults evince[d] significantly different patterns of approach [*i.e.*, reward-seeking] and avoidance [*i.e.*, risk-averse] behavior." [16] Whereas adolescents improved their performance over time by being drawn to the bets with the best rewards, adults improved by avoiding bets with the worst losses. The authors concluded that the "present study, as well as previous work, demonstrates that decision making ... improves throughout adolescence and into young adulthood but that this improvement may be due not to cognitive maturation but to changes in affective processing. Whereas adolescents may attend more to the potential rewards of a risky decision than to the potential costs, adults tend to consider both, even weighing costs more than rewards." [17]

Similarly, adolescents are particularly attuned to *immediate* rewards, and display much steeper "temporal ***12** discounting" than adults. [18] Juveniles are emotionally primed for spur-of-the-moment, reward- and sensation-seeking behavior without offsetting, adult sensitivities to corresponding risks and longer-term consequences. Indeed, studies have shown that perceptions of reward, not risk, are better predictors of adolescent antisocial behaviors. [19] This less mature weighing of risk and reward renders adolescents more likely to engage in criminal activity, as well as other kinds of risk-taking. [20]

3. Finally, juveniles differ from adults in their ability to foresee and take into account the consequences of their behavior. By definition, adolescents have less life experience on which to draw, making it less likely that they will fully apprehend the potential negative consequences of their actions. [21] Moreover, adolescents are less able than adults to envision and plan for the future, a capacity still developing during adolescence. [22] The study of maturity of judgment discussed above found ***13** that adolescents' future orientation is weaker than adults': Comparing over 1,000 subjects, it found that even 17-year-olds scored lower than adults on measures of "perspective," which encompassed "the ability to see short and long term consequences," as well as the ability to "take other people's perspectives into account." [23] Similarly, studies

have shown that, among 15- to 17-year-olds, realism in thinking about the future increases with age, and that the skills required for future planning continue to develop until the early 20s. [24]

The ability to resist and control emotional impulses, to gauge risks and benefits in an adult manner, and to envision the future consequences of one's actions - even in the face of environmental or peer pressures - are critical components of social and emotional maturity, necessary in order to make mature, fully considered decisions. Empirical research confirms that even older adolescents have not fully developed these abilities and hence lack an adult's capacity for mature judgment. "[I]t is clear that important progress in the development of [social and emotional maturity] occurs sometime during late adolescence, and that these changes have a profound effect on the ability to make consistently mature decisions." [25]

***14** It should be noted that multiple abilities contribute to mature judgment, and those abilities develop at different rates. Sound judgment requires both cognitive and psychosocial skills, but the former mature earlier than the latter. Studies of general cognitive capability show an increase from pre-adolescence until about age 16, when gains begin to plateau. [26] By contrast, social and emotional maturity continue to develop throughout adolescence. Thus, older adolescents (aged 16-17) often have logical reasoning skills that approximate those of adults, but nonetheless lack the adult capacities to exercise self-restraint, to weigh risk and reward appropriately, and to envision the future that are just as critical to mature judgment, [27] especially in emotionally charged settings. [28] Younger adolescents are thus doubly disadvantaged, because they typically lack not only those social and emotional skills but basic cognitive capabilities as well. [29]

***15 B. Juveniles Are More Vulnerable To Negative External Influences**

As this Court has also recognized, "juveniles are more vulnerable ... to negative influences and outside pressures, including peer pressure." *Roper*, 543 U.S. at 569. Because of their developmental immaturity, adolescents are more susceptible than adults to the negative influences of their environment, and their actions are shaped directly by family and peers in ways that adults' are not. "Adolescents are dependent on living circumstances of their parents and families and hence are vulnerable to the impact of conditions well beyond their control." [30] Difficult family and neighborhood conditions are major risk factors for juvenile crime, including ***16** homicide. [31] Yet, precisely because of their legal minority, juveniles lack the freedom to remove themselves from those negative external influences. Put simply, juveniles lack the control over themselves and their lives that adults possess, mitigating their blameworthiness for remaining in destructive or " 'criminogenic' " situations. *Roper*, 543 U.S. at 569.

Juveniles are also especially vulnerable to the negative influence of peer pressure. Research has shown that susceptibility to peer pressure to engage in antisocial behavior increases between childhood and early adolescence, peaks at around age 14, and then declines slowly during the late adolescent years, with relatively little change after age 18. [32] For instance, one major study found that exposure to peers during a risk-taking task doubled the amount of risky behavior among mid-adolescents (with a mean age of 14), increased it by 50 percent among college undergraduates (with a mean age of 19), and had no impact at all among ***17** young adults. [33] "[T]he presence of peers makes adolescents and youth, but not adults, more likely to take risks and more likely to make risky decisions." [34]

This study was recently replicated using fMRI technology, allowing researchers to measure variations in the activation of different brain areas under different experimental conditions. Because of technological constraints, the "peer pressure" variable was limited to manipulating whether test subjects were observed by peers or not while performing the task. Strikingly, mere awareness that peers were watching encouraged risky behavior among juveniles, but not adults. [35] The neuroimaging also showed different activation in different brain areas across the experimental variables. Adults showed

significantly greater activation in brain regions involved in executive functions and the regulation of impulses, whether or not they were being observed by peers. By contrast, adolescents showed significantly greater activation in brain areas associated with reward processing when they were told that their peers were watching than when they were not being observed. [36]

***18** Juveniles' lesser ability to resist peer influence affects their judgment both directly and indirectly. "In some contexts, adolescents might make choices in response to direct peer pressure, as when they are coerced to take risks that they might otherwise avoid. More indirectly, adolescents' desire for peer approval, and consequent fear of rejection, affect their choices even without direct coercion. The increased salience of peers in adolescence likely makes approval-seeking especially important in group situations." [37]

Adolescents are thus more likely than adults to engage in antisocial behavior in order to conform to peer expectations or achieve respect and status among their peers. [38] Not surprisingly, juvenile crime is significantly correlated with exposure to delinquent peers, [39] and adolescents are "far more likely than adults to commit crimes in groups." [40] "No matter the crime, if a teenager is the offender, he is usually not committing the offense alone." [41] Indeed, "[m]ost adolescent decisions to break the law take place on a social stage where the immediate pressure of peers is the real motive." [42] "A ***19** necessary condition for an adolescent to stay law-abiding is the ability to deflect or resist peer-pressure," a social skill that is not fully developed in adolescents. [43]

In short, as this Court has observed, "youth is more than a chronological fact. It is a time and condition of life when a person may be most susceptible to influence and to psychological damage." *Eddings v. Oklahoma*, 455 U.S. 104, 115 (1982). Because juveniles' developmental immaturity and legal minority render them both more susceptible to, and less capable of escaping, negative external pressures, they "have a greater claim than adults to be forgiven" for the criminal acts that result from such pressures. *Roper*, 543 U.S. at 570.

C. Juveniles Have A Greater Capacity For Change And Reform

Finally, as this Court has recognized, "the character of a juvenile is not as well formed as that of an adult," and "[t]he personality traits of juveniles are more transitory, less fixed." *Roper*, 543 U.S. at 570. Accordingly, "[j]uveniles are more capable of change than are adults, and their actions are less likely to be evidence of 'irretrievably depraved character.' " *Graham*, 130 S. Ct. at 2026. A defining aspect of adolescence is that character is not yet fully formed, and adolescents' signature qualities - including their susceptibility to peer influence and weaknesses in self-regulation - reflect their incomplete identity or "sense of self." Thus, what may be perceived as fixed personality traits in juveniles may in fact result from malleable factors such as present maturity level or social ***20** context, rather than engrained or enduring aspects of personality or worldview. Research has shown that personality traits change significantly during the developmental transition from adolescence to adulthood, [44] and the process of identity-formation typically remains incomplete until at least the early twenties. [45] Juveniles are simply more likely than adults to change.

This Court recognized in *Roper* that because "juveniles still struggle to define their identity, ... it is less supportable to conclude that even a heinous crime committed by a juvenile is evidence of irretrievably depraved character." 543 U.S. at 570. And it reaffirmed in *Graham* that " 'from a moral standpoint it would be misguided to equate the failings of a minor with those of an adult, for a greater possibility exists that a minor's character deficiencies will be reformed.' " 130 S. Ct. at 2026-2027.

In fact, juveniles do typically outgrow their antisocial behavior as the "'impetuousness and recklessness' " of youth subside in adulthood. *Roper*, 543 U.S. at 570. Adolescent criminal conduct frequently results from ***21** experimentation

with risky behavior and not from deep-seated moral deficiency reflective of "bad" character.[46] For most juveniles, therefore, antisocial behavior will " 'cease with maturity as individual identity becomes settled.' " *Id.* at 570. Only a small proportion of adolescents who experiment with illegal activities will develop an entrenched pattern of criminal behavior that persists into adulthood; "the vast majority of adolescents who engage in criminal or delinquent behavior desist from crime as they mature."[47]

As this Court has previously observed, moreover, even experts have no reliable way to predict whether a particular juvenile offender will continue to commit crimes as an adult. *See Roper*, 543 U.S. at 573. The positive predictive power of juvenile psychopathy assessments, for instance, remains poor. One study found that only 16% of young adolescents who scored in the top quintile on a juvenile psychopathy measure would eventually be assessed as psychopathic at age 24.[48] The authors concluded that "most individuals identified as psychopaths at age 13 will not receive such a diagnosis" as adults.[49] A recent study of 75 male juvenile offenders found that assessments of psychopathic characteristics ***22** did not predict general or violent reconvictions over a 10-year follow-up period.[50] And another recent study showed no correlation between a youthful homicide offense and the basic psychological measures of persistent antisocial personality such as "cruelty to people and callous-unemotional behavior."[51]

To be sure, research has identified certain childhood risk factors, or "predictors," that show a statistically significant association with adult criminality. But such studies do not suggest that anyone could reliably determine, *ex ante*, whether particular juvenile offenders will reoffend. To the contrary, the same research makes clear that such predictions cannot be made with any accuracy. Simply put, while many criminals may share certain childhood traits, the great majority of juvenile offenders with those traits will not be criminal adults. For example, a major longitudinal study of Pittsburgh inner-city boys successfully identified, *ex post*, childhood risk factors, including various forms of antisocial behavior and crime, that were correlated with future homicide convictions. But it also found that, even among the subgroup of boys with the greatest number of risk factors, only a small minority were eventually convicted of homicide: Using the authors' model to attempt to identify juveniles who would be future homicide offenders yielded a very high false positive rate of 87%.[52]

***23** In fact, researchers have consistently concluded that the behavior of juveniles who will and will not continue as criminal offenders through adulthood is "often indistinguishable during adolescence."[53] In first distinguishing between adolescence-limited and persistent offenders, researchers recognized that they could not "effectively assign individual delinquent adolescents to meaningful subtypes on the basis of ... their antisocial behavior during adolescence."[54] And those who have dedicated their careers to identifying risk factors associated with persistent criminality continue to acknowledge that "[t]he results show very imperfect predictions of which offense trajectory individuals will follow over time," and to warn against the "danger that policy makers will start to use less than good predictions as a rationale for harsh punishments and severe legal sanctions."[55]

***24** Moreover, it is just as difficult to predict future criminality among adolescents convicted of the most serious crimes.[56] A recent, major effort to identify risk factors for recidivism among serious adolescent offenders confirmed the "good news ... that even within a sample ... limited to those convicted of the most serious crimes, the percentage who continue to offend consistently at a high level is very small," while acknowledging the "bad news" that the ability to predict future criminality remains "exceedingly limited."[57] Most strikingly, when the homicide study discussed above limited its effort to predict future homicide offenses to boys who had already committed an act of violence, it "did not significantly improve predictive accuracy."[58] In fact, the false-positive rate *increased* from 87% to 89%.[59]

In sum, juveniles are still developing their character and identity, and it is quite likely that a juvenile offender ***25** will desist from crime in adulthood. *See Roper*, 543 U.S. at 570. Juvenile crime is likely to be the product of the "signature

qualities of youth," *id.*; there is no reliable way to determine that a juvenile's offenses are the result of an irredeemably corrupt character; and there is thus no reliable way to conclude that a juvenile - even one convicted of an extremely serious offense - should be sentenced to life in prison, without any opportunity to demonstrate change or reform.

D. Juveniles' Psychosocial Immaturity Is Consistent With Recent Research Regarding Adolescent Brain Development

Neuroscientists continue to accumulate evidence that the adolescent brain is not yet fully developed in critical respects. By now, "[t]here is incontrovertible evidence of significant changes in brain structure and function during adolescence," and "[a]lthough most of this work has appeared just in the last 10 years, there is already strong consensus among developmental neuroscientists about the nature" of these changes. [60] While research continues into the precise meaning and effect of the changes in the brain during adolescence, they are consistent with and suggest the possible physiological basis for adolescents' observed psychosocial immaturity.

The most noteworthy features of adolescent brain development relate to changes occurring within the brain's frontal lobes - in particular the prefrontal cortex - and in the connections between the prefrontal ***26** cortex and other brain structures. These areas and interconnections are critical to "executive" functions such as planning, motivation, judgment, and decisionmaking, including the evaluation of future consequences, the weighing of risk and reward, the perception and control of emotions, and the processing and inhibition of impulses. [61] Four related changes in these brain systems during adolescence merit special attention.

First, early adolescence (especially the period immediately after puberty) coincides with major changes in the "incentive processing system" of the brain involving neurotransmitters like dopamine. [62] "[R]eward-related regions of the brain and their neurocircuitry undergo particularly marked developmental changes ***27** during adolescence." [63] These pubertal changes are seen in other species, and "have been linked to changes in reward-directed activity" among adolescents, especially the willingness to engage in risky and socially motivated behaviors. [64] The observed spike in risk-taking, reward-seeking, and peer-influenced behaviors among adolescents correlates with this normal aspect of adolescent brain development.

Second, during childhood and early adolescence the brain undergoes substantial synaptic "pruning" - the paring away of unused synapses - leading to more efficient neural connections. [65] During adolescence, this pruning is more characteristic of the prefrontal cortex than other brain regions, consistent with the observation that adolescence is a time of marked improvement in executive functions. [66]

***28** *Third*, the adolescent brain undergoes substantial myelination, the process through which neural pathways are insulated with a white fatty tissue called myelin. That insulation "speeds ... neural signal transmission," making "communication between different parts of the brain faster and more reliable." [67] "[M]yelination is ongoing well into late adolescence and early adulthood." [68] And this "improved connectivity within the prefrontal cortex is important for higher order functions subserved by multiple prefrontal areas, including many aspects of executive function, such as response inhibition, planning ahead, weighing risks and rewards, and the simultaneous consideration of multiple sources of information." [69]

Fourth, "well into late adolescence" there is "an increase in connections not only among cortical areas but between cortical and subcortical regions" that are "especially important for emotion regulation." [70] As the ***29** brain matures, that self-regulation is "facilitated by the increased connectivity between regions important in the processing of emotional and social information and regions important in cognitive control processes." [71] This developmental pattern is consistent

with adults' superior ability to make mature judgments about risk and reward, and to exercise cognitive control over their emotional impulses, especially in circumstances that adolescents would react to as socially charged. [72]

In short, the brain systems that govern many aspects of social and emotional maturity, such as impulse control, risk avoidance, planning ahead, and coordination of emotion and cognition, continue to mature throughout adolescence. [73] Importantly, these changes occur at different times, with the rapid, pubertal changes in the brain's incentive and social processing systems outpacing the slower, steadier, and later-occurring changes in areas related to executive function ***30** and self-control. [74] Indeed, studies have shown that the prefrontal cortex is among the last areas in the brain to mature fully. [75] These findings suggest a

basic framework, articulated in slightly different versions by many writers ... posit[ing] that middle adolescence is a time of heightened vulnerability to risky and reckless behavior because of the temporal disjunction between the rapid rise in dopaminergic activity around the time of puberty, which leads to an increase in reward-seeking, and the slower and more gradual maturation of the prefrontal cortex and its connections to other brain regions, which leads to improvements in cognitive control and in the coordination of affect and cognition. As dopaminergic activity declines from its early adolescent peak, and as self-regulatory systems become increasingly mature, risk-taking begins to decline. [76]

"From this perspective, middle adolescence (roughly 14-17) should be a period of especially heightened vulnerability to risky behavior, because sensation-seeking is high and self-regulation is still immature. And in fact, many risk behaviors follow this pattern, including unprotected sex, criminal behavior, attempted suicide, and reckless driving." [77]

***31** Although the precise relationships between particular aspects of brain development and adolescent behavior continue to be studied, these findings regarding the neuroscience of adolescent development reinforce and expand upon the well-established behavioral findings discussed in *Roper* and *Graham.* They demonstrate that, even in late adolescence, important aspects of brain maturation remain incomplete. And those normal patterns of adolescent physiological development are correlated with the poor judgment and particular vulnerability to negative social influences that characterize adolescence and then subside in young adulthood. Unlike adults, juveniles may thus be expected to change as they age and their brains mature, evincing both fewer impulses toward reckless and criminal behavior and an increased ability to restrain such impulses.

II. Sentencing Juveniles To Lifelong Imprisonment With No Opportunity To Demonstrate Reform Is A Disproportionate Punishment

In *Graham*, this Court determined that a sentence of life without parole for juvenile offenders convicted of non-homicide offenses was constitutionally disproportionate punishment for two related reasons - both of which are equally powerful as applied to juveniles convicted of homicide.

First, juveniles' immaturity, vulnerability, and changeability - while in no way excusing their crimes - substantially lessen their culpability and undermine any justification for definitively ending their free lives. *Graham*, 130 S. Ct. at 2026; *Roper*, 543 U.S. at 569-570. The Court thus reaffirmed in *Graham* that "from a moral standpoint it would be misguided to equate the failings of a minor with those of an adult." ***32** 130 S. Ct. at 2026-2027. At the same time, the Court recognized that "[l]ife without parole is an especially harsh punishment for a juvenile," because "a juvenile offender will on average serve more years and a greater percentage of his life in prison than an adult offender." *Id.* at 2028. "A 16-year-old and a 75-year-old each sentenced to life without parole receive the same punishment in name only." *Id.* In fact, a juvenile sentenced to life in custody not only serves a greater percentage of his life in prison, but suffers a unique deprivation: He will never experience adulthood - or the ability "to attain a mature understanding of his own humanity," *Roper*, 543 U.S. at 574 - as a free person.

Sentences that foreclose any possibility of eventual release are thus particularly draconian for juveniles. Although adolescents can be expected to mature and reform as they age, such a sentence "means denial of hope; it means that good behavior and character improvement are immaterial; it means that whatever the future might hold in store for the mind and spirit of the convict, he will remain in prison for the rest of his days." *Graham*, 130 S. Ct. at 2027. Juvenile crimes are committed "while [the offender is] a child in the eyes of the law," *id.* at 2033, meaning that most juvenile offenders are sentenced to life imprisonment without ever having been initiated into such elementary aspects of adult society as voting, driving, marriage, parenthood, profession - even high-school graduation. For adolescent offenders, a sentence of "[l]ife in prison without the possibility of parole gives no chance for fulfillment outside prison walls, no chance for reconciliation with society, no hope." *Id.* at 2032. Given juveniles' reduced culpability and increased likelihood of reform, such a severe sanction - foreclosing any willingness even to consider release in the future - is manifestly ***33** disproportionate to the penological justifications for imposing it.

Neither this Court's precedent nor the research into adolescent development provides any reason why this analysis should be different in the case of juvenile homicide offenders. This Court first recognized the reduced culpability of adolescent offenders in the context of prohibiting the death penalty for juvenile homicide offenses, finding that even for older adolescents, and "even [for] a heinous crime," the immaturity, vulnerability, and changeability of juvenile offenders made it "less supportable to conclude that ... [a] crime committed by a juvenile is evidence of irretrievably depraved character." *Roper*, 543 U.S. at 570. To be sure, more serious crimes call for more serious punishments. But there is no reason why the reduction in culpability associated with adolescence should vary according to the severity of the offense. Indeed, the best available research indicates that even serious juvenile offenders are far more likely than not to desist from criminality as they mature, and that it is equally true of the most serious offenders that "expert psychologists [cannot] differentiate between the juvenile offender whose crime reflects unfortunate yet transient immaturity, and the rare juvenile offender whose crime reflects irreparable corruption." *Id.* at 573. [78]

Accordingly, the penological justifications for a sentence of life imprisonment without parole are weakened for juveniles who commit homicide, just as they are for other juvenile offenders. The retributive purpose of such a punishment is attenuated because "culpability ***34** or blameworthiness is diminished, to a substantial degree, by reason of youth and immaturity." *Roper*, 543 U.S. at 571. Likewise, the same characteristics of juveniles that render them less culpable - their impulsivity, rash decision-making, biased attention to anticipated immediate rewards rather than longer-term costs, and lesser ability to consider and evaluate the future consequences of their actions - substantially weaken the deterrence justification for such punishment. *Id.* [79] Life without parole will unquestionably incapacitate a juvenile offender, but the Court rightly noted in *Graham* that justifying "life without parole on the assumption that the juvenile offender forever will be a danger to society requires the sentencer to make a judgment that the juvenile is incorrigible," when "[t]he characteristics of juveniles make that judgment questionable." 130 S. Ct. 2029. And it is particularly inappropriate to "forswear[] altogether the rehabilitative ideal," *id.* at 2030, with respect to offenders who are far more likely than any others to reform as both their character and their physical brain structure mature into adulthood.

***35** In short, this Court has recognized what research confirms: Adolescence is transitory, and juveniles change. Indeed, most adolescents who commit crimes will desist from criminal activity in adulthood. Because the adolescent self is not yet fully formed, there is no way reliably to conclude that an adolescent's crime is the expression of an entrenched and irredeemably malign character that might justify permanent incarceration. And, even in the case of the most serious offenses, there is no reliable way to distinguish the juvenile offender who might become a hardened criminal from the far more common offender whose crime is a product of the transient influences of adolescence itself. Sentencing a juvenile to life imprisonment "without any meaningful opportunity to obtain release, no matter what he might do to demonstrate that the bad acts he committed as a teenager are not representative of his true character, even if he spends the next half century attempting to atone for his crimes and learn from his mistakes," *Graham*, 130 S. Ct. at 2033, disregards entirely the signature characteristics of youth. And sentencing such an immature and less culpable juvenile to spend his entire

adult life in prison, notwithstanding the likelihood that "[m]aturity can lead to … remorse, renewal, and rehabilitation," *id.* at 2032, is grossly disproportionate punishment.

*36 CONCLUSION

The judgments below should be reversed.

Footnotes

1 The parties have consented to the filing of this brief. Pursuant to Rule 37.3(a), letters of consent are on file with the Clerk of the Court. No counsel for a party authored this brief in whole or in part, and no person, other than amici curiae, their members, and their counsel, made a monetary contribution to the preparation or submission of this brief.

2 Amici acknowledge the assistance of Elizabeth Cauffman, Ph.D., Thomas Grisso, Ph.D., Terrie Moffitt, Ph.D., Laurence Steinberg, Ph.D., and Jennifer Woolard, Ph.D., in the preparation of this brief.
Research cited in this brief includes data from studies conducted using the scientific method. Such research typically is subject to critical review by outside experts, usually during the peer-review process preceding publication in a scholarly journal.

3 We use the terms "juvenile" and "adolescent" interchangeably to refer to individuals aged 12 to 17. Science cannot, of course, draw bright lines precisely demarcating the boundaries between childhood, adolescence, and adulthood; the "qualities that distinguish juveniles from adults do not disappear when an individual turns 18." *Roper*, 543 U.S. at 574. Likewise, younger adolescents differ in some respects from 16- and 17-year-olds. Nonetheless, because adolescents generally share certain developmental characteristics that mitigate their culpability, and because "the age of 18 is the point where society draws the line for many purposes between childhood and adulthood," this Court's decisions have recognized age 18 as a relevant demarcation point. *Graham*, 130 S. Ct. at 2030; *see Roper*, 543 U.S. at 574. The research discussed in this brief accordingly applies to adolescents under age 18, including older adolescents, unless otherwise noted.

4 Jeffrey Arnett, *Reckless Behavior in Adolescence: A Developmental Perspective*, 12 Developmental Rev. 339, 344 (1992).

5 Terrie Moffitt, *Adolescent-Limited and Life-Course-Persistent Antisocial Behavior: A Developmental Taxonomy*, 100 Psychol. Rev. 674, 685-686 (1993). Moffitt posits that there are two groups of adolescent offenders who may engage in similar antisocial behavior: a majority whose offending is limited to adolescence, and a minority who will persist into adulthood.

6 *Id.* at 675 & fig. 1 (depicting age-crime curve with steep peak in late adolescence); Arnett, *supra* note 4, at 343; Terrie Moffitt, *Natural Histories of Delinquency*, in *Cross-National Longitudinal Research on Human Development and Criminal Behavior* 3, 29 (Elmar Weitekamp & Hans-Jürgen Kerner eds., 1994).

7 Rolf Loeber et al., *Violence and Serious Theft* 77 (2008); *see also* Moffitt, *supra* note 6, at 7; Kathryn Monahan et al., *Trajectories of Antisocial Behavior and Psychosocial Maturity from Adolescence to Young Adulthood,* 45 Developmental Psychol. 1654, 1654 (2009).

8 Laurence Steinberg, *Adolescent Development and Juvenile Justice*, 5 Ann. Rev. Clinical Psychol. 47, 55-56 (2008).

9 Elizabeth Cauffman & Laurence Steinberg, *(Im)maturity of Judgment in Adolescence*, 18 Behav. Sci. & L. 741, 748-749, 754 & tbl. 4 (2000).

10 Laurence Steinberg et al., *Age Differences in Sensation Seeking and Impulsivity as Indexed by Behavior and Self-Report*, 44 Developmental Psychol. 1764, 1774-1776 (2008).

11 Dustin Albert & Laurence Steinberg, *Judgment and Decision Making in Adolescence,* 21 J. Research on Adolescence 211, 220 (2011); *see also* Adriana Galvan et al., *Risk Taking and the Adolescent Brain*, 10 Developmental Sci. F8, F13 (2007) (finding, in study of individuals aged 7 to 29, that impulse control continues to develop throughout adolescence and early adulthood); Rotem Leshem & Joseph Glicksohn, *The Construct of Impulsivity Revisited*, 43 Personality & Individual Differences 681, 684-686 (2007) (reporting significant decline in impulsivity from ages 14-16 to 20-22).

12 Franklin Zimring, *Penal Proportionality for the Young Offender*, in *Youth on Trial* 271, 280 (Thomas Grisso & Robert Schwartz eds., 2000).

13 *Id.* at 282.

14 Laurence Steinberg & Elizabeth Scott, *Less Guilty by Reason of Adolescence: Developmental Immaturity, Diminished Responsibility, and the Juvenile Death Penalty*, 58 Am. Psychologist 1009, 1012 (2003); *see* Arnett, *supra* note 4, at 350-353 (summarizing evidence that adolescent recklessness relates to poor "probability reasoning"); Susan Millstein & Bonnie

Halpern-Felsher, *Perceptions of Risk and Vulnerability*, in *Adolescent Risk and Vulnerability* 15, 34-35 (Baruch Fischoff et al. eds., 2001).

15 Bonnie Halpern-Felsher & Elizabeth Cauffman, *Costs and Benefits of a Decision: Decision-Making Competence in Adolescents and Adults*, 22 J. Applied Developmental Psychol. 257, 265, 268 (2001). Even greater differences prevailed between adults and younger adolescents. *See id. at* 268.

16 Elizabeth Cauffman et al., *Age Differences in Affective Decision Making as Indexed by Performance on the Iowa Gambling Task*, 46 Developmental Psychol. 193, 204 (2010).

17 *Id.* at 204, 206.

18 Laurence Steinberg et al., *Age Differences in Future Orientation and Delay Discounting*, 80 Child Dev. 28, 39 (2009); Steinberg, *supra* note 8, at 58.

19 Louk Peters et al., *A Review of Similarities Between Domain-Specific Determinants of Four Health Behaviors Among Adolescents*, 24 Health Educ. Research 198, 216 (2009).

20 Arnett, *supra* note 4, at 344, 350-351 (relating skewed adolescent risk- and reward-perception to fact that 50% or more of adolescents report drunk driving, unprotected sex, illegal drug use, or some form of criminal activity).

21 *Id.* at 351-352; Zimring, *supra* note 12, at 280.

22 *See* Jari-Erik Nurmi, *How Do Adolescents See Their Future? A Review of the Development of Future Orientation and Planning*, 11 Developmental Rev. 1, 28-29 (1991); Steinberg et al., *supra* note 18, at 35-36.

23 Cauffinan & Steinberg, *supra* note 9, at 746, 748, 754 & tbl. 4.

24 Nurmi, *supra* note 22, at 28-29; *see* Steinberg et al., *supra* note 18, at 35-36.

25 Cauffman & Steinberg, *supra* note 9, at 741, 756, 758 (noting that the most dramatic increase in psychosocial maturity occurs between ages 16 and 19); *see* Halpern-Felsher & Cauffinan, *supra* note 15, at 271 ("[I]mportant progress in the development of decisionmaking competence occurs sometime during late adolescence[.]").

26 *See, e.g.*, Thomas Grisso et al., *Juveniles' Competence to Stand Trial*, 27 Law & Hum. Behav. 333, 343-344 (2003) (16- to 17-year-olds did not differ from 18- to 24-year-old adults but performed significantly better than 14- to 15-year-olds on test of basic cognitive abilities); Daniel Keating, *Cognitive and Brain Development*, in *Handbook of Adolescent Psychology* 45, 64 (Richard Lerner & Laurence Steinberg eds., 2d ed. 2004) (cognitive functions exhibit robust growth at earlier ages but approach a limit in the 14- to 16-year-old group).

27 Cauffman & Steinberg, *supra* note 9, at 743-745; Halpern-Felsher & Cauffman, *supra* note 15, at 264-271; Steinberg, *supra* note 8, at 55-59.

28 Albert & Steinberg, *supra* note 11, at 216-220.

29 The dissent in *Roper* criticized the American Psychological Association for taking allegedly inconsistent positions regarding adolescent maturity with respect to severe criminal sanctions for juveniles (in *Roper*) and the competence of minor females to obtain abortions absent parental notification (in *Hodgson v. Minnesota*, 497 U.S. 417 (1990)). *See* 543 U.S. at 617-618 (Scalia, J., dissenting). These are different questions concerning distinct aspects of mature judgment. *Hodgson* addressed *competence* to make medical decisions that can be made in a relatively unhurried manner in consultation with medical professionals, and the Association's brief thus focused on adolescents' *cognitive* abilities, which approximate those of adults by mid-adolescence. The questions presented in *Roper, Graham*, and this case concern the degree of *culpability* and *reformability* of adolescents who commit criminal acts that often evince impulsivity and ill-considered choices resulting from *psychosocial* immaturity. *See* Laurence Steinberg et al., *Are Adolescents Less Mature Than Adults? Minors' Access to Abortion, the Juvenile Death Penalty, and the Alleged APA "Flip-Flop,"* 64 Am. Psychologist 583, 592-593 (2009); Elizabeth Scott et al., *Evaluating Adolescent Decision Making in Legal Contexts*, 19 Law & Hum. Behav. 221, 226-235 (1995).

30 Alan Kazdin, *Adolescent Development, Mental Disorders, and Decision Making of Delinquent Youths*, in *Youth on Trial, supra* note 12, at 47.

31 *Id.* at 47-48; *see* Rolf Loeber & David Farrington, *Young Homicide Offenders and Victims: Risk Factors, Prediction, and Prevention from Childhood* 61 & tbl. 4.1 (2011) (noting high likelihood that homicide offenders came from broken family or bad neighborhood); Jeffrey Fagan, *Contexts of Choice by Adolescents in Criminal Events*, in *Youth on Trial, supra* note 12, at 372, 389-391.

32 Elizabeth Scott & Laurence Steinberg, *Rethinking Juvenile Justice* 38 (2008); Thomas Berndt, *Developmental Changes in Conformity to Peers and Parents*, 15 Developmental Psychol. 608, 612, 615-616 (1979); Laurence Steinberg & Susan Silverberg, *The Vicissitudes of Autonomy in Early Adolescence*, 57 Child Dev. 841, 848 (1986); Fagan, *supra* note 31, at 382-384 (discussing coercive effect of social context on adolescents).

33 Margo Gardner & Laurence Steinberg, *Peer Influence on Risk Taking, Risk Preference, and Risky Decision Making in Adolescence and Adulthood*, 41 Developmental Psychol. 625, 626-634 (2005).

34 *Id.* at 634; *see* Laurence Steinberg & Kathryn Monahan, *Age Differences in Resistance to Peer Influence*, 43 Developmental Psychol. 1531, 1538 (2007) (same).

35 Jason Chein et al., *Peers Increase Adolescent Risk Taking By Enhancing Activity in the Brain's Reward Circuitry*, 14 Developmental Sci. F1, F7 (2011).

36 *Id.* at F5-F8.

37 Scott & Steinberg, *supra* note 32, at 38-39; *see also* Moffitt, *supra* note 5, at 686; Zimring, *supra* note 12, at 280-281.

38 *See* Moffitt, *supra* note 5, at 686.

39 *See id.* at 687-688.

40 Scott & Steinberg, *supra* note 32, at 39.

41 Zimring, *supra* note 12, at 281; *see* Joan McCord & Kevin Conway, *Co-Offending and Patterns of Juvenile Crime* 5 (2005) (finding that "[c]o-offending violence increased throughout adolescence").

42 Zimring, *supra* note 12, at 280.

43 *Id.* at 280-281.

44 *See* Brent Roberts et al., *Patterns of Mean-Level Change in Personality Traits Across the Life Course*, 132 Psychol. Bull. 1, 14-15 (2006).

45 *E.g.*, Alan Waterman, *Identity Development from Adolescence to Adulthood*, 18 Developmental Psychol. 341, 355 (1982) ("The most extensive advances in identity formation occur during the time spent in college."); Laurence Steinberg & Robert Schwartz, *Developmental Psychology Goes to Court*, in *Youth on Trial, supra* note 12, at 9,27 ("[M]ost identity development takes place during the late teens and early twenties."); Scott & Steinberg, *supra* note 32, at 52 ("[C]oherent integration of ... [identity] does not occur until late adolescence or early adulthood. ... [T]he final stages of this process often occur during the college years.").

46 Moffitt, *supra* note 5, at 686, 690; *see also* Arnett, *supra* note 4, at 344, 366-367.

47 Steinberg & Scott, *supra* note 14, at 1014-1015; *see also* Moffitt, *supra* note 5, at 685-686; Monahan et al., *supra* note 7, at 1654, 1655.

48 Donald Lynam et al., *Longitudinal Evidence That Psychopathy Scores in Early Adolescence Predict Adult Psychopathy*, 116 J. Abnormal Psychol. 155, 160 (2007).

49 *Id.* at 162.

50 *See* John Edens & Melissa Cahill, *Psychopathy in Adolescence and Criminal Recidivism in Young Adulthood*, 14 Assessment 57, 60 (2007).

51 Loeber & Farrington, *supra* note 31, at 158.

52 *Id.* at 75.

53 Monahan et al., *supra* note 7, at 1655; *see also, e.g.*, John Edens et al., *Assessment of "Juvenile Psychopathy" and Its Association with Violence*, 19 Behav. Sci. & L. 53, 59 (2001) (collecting evidence that psychopathy assessments may "tap construct-irrelevant variance associated with relatively *normative* and *temporary* characteristics of adolescence rather than deviant and stable personality features"); Edward Mulvey & Elizabeth Cauffman, *The Inherent Limits of Predicting School Violence*, 56 Am. Psychologist 797, 799 (2001) ("Assessing adolescents ... presents the formidable challenge of trying to capture a rapidly changing process with few trustworthy markers."); Thomas Grisso, *Double Jeopardy: Adolescent Offenders with Mental Disorders* 64-65 (2004) (noting discontinuity and disappearance of mental disorders identified in adolescence).

54 Moffitt, *supra* note 5, at 678.

55 Loeber et al., *supra* note 7, at 333.

56 *See id.* (distinguishing, throughout, between serious and less serious forms of violence and theft).

57 Edward Mulvey et al., *Trajectories of Desistance and Continuity in Antisocial Behavior Following Court Adjudication Among Serious Adolescent Offenders*, 22 Dev. & Psychopathology 453, 468-470 (2010); *see also* Monahan et al., *supra* note 7 (finding that only 6% of serious juvenile offenders persisted in high levels of antisocial behavior into adulthood).

58 Loeber & Farrington, *supra* note 31, at 88.

59 *Id.* at 89; *see also* Alex Piquero et al., *Violence in Criminal Careers: A Review of the Literature from a Developmental Life-Course Perspective*, Aggression & Violent Behav. (forthcoming 2012) (concluding that "most youths who become violent do so in adolescence and their violent involvement is limited to the late teen/early 20s" and that "attempt[ing] to correctly predict the violent recidivist is virtually impossible").

60 Laurence Steinberg, *Should the Science of Adolescent Brain Development Inform Public Policy?*, 64 Am. Psychologist 739, 742 (2009).

61 *E.g.*, Elkhonon Goldberg, *The Executive Brain: Frontal Lobes and the Civilized Mind* 23, 24, 141 (2001); B.J. Casey et al., *Structural and Functional Brain Development and its Relation to Cognitive Development*, 54 Biological Psychol. 241, 244-246 (2000); Elizabeth Sowell et al., In Vivo *Evidence for Post-Adolescent Brain Maturation in Frontal and Striatal Regions*, 2 Nature Neurosci. 859, 860 (1999); Antonio Damasio & Steven Anderson, *The Frontal Lobes*, in *Clinical Neuropsychology* 404, 434-435 (Kenneth Heilman & Edward Valenstein eds., 4th ed. 2003) (one "hallmark of frontal lobe dysfunction is difficulty making decisions that are in the long-term best interests" of the individual).

62 *E.g.*, Chein et al., *supra* note 35, at F2; Linda Spear, *The Behavioral Neuroscience of Adolescence* 149-150 (2009); Dustin Wahlstrom et al., *Developmental Changes In Dopamine Neurotransmission in Adolescence: Behavioral Implications and Issues in Assessment*, 72 Brain & Cognition 146, 150-151 (2010); Monique Ernst et al., *Neurobiology of the Development of Motivated Behaviors in Adolescence: A Window into a Neural Systems Model*, 93 Pharmacology Biochem. & Behav. 199, 206-208 (2009); Albert & Steinberg, *supra* note 11, at 217.

63 Tamara Doremus-Fitzwater et al., *Motivational Systems in Adolescence: Possible Implications for Age Differences in Substance Abuse and Other Risk-Taking Behaviors*, 72 Brain & Cognition 114, 116 (2010); Steinberg, *supra* note 60, at 743.

64 Laurence Steinberg, *A Behavioral Scientist Looks at the Science of Adolescent Brain Development*, 72 Brain & Cognition 160, 161 (2010); Spear, *supra* note 62, at 18-19; Linda Van Leijenhorst et al., *What Motivates the Adolescent? Brain Regions Mediating Reward Sensitivity Across Adolescence*, 20 Cerebral Cortex 61, 67 (2010).

65 Casey et al., *supra* note 61, at 242-243; Nitin Gogtay et al., *Dynamic Mapping of Human Cortical Development During Childhood Through Early Adulthood*, 101 Proc. Nat'l Acad. Sci. 8174, 8175 (2004); Spear, *supra* note 62, at 81-90; Peter Huttenlocher, *Neural Plasticity: The Effects of Environment on the Development of the Cerebral Cortex* 41, 46-47, 52-58, 67 (2002).

66 *E.g.*, Nitin Gogtay & Paul Thompson, *Mapping Gray Matter Development*, 72 Brain & Cognition 6, 7 (2010); Neir Eshel et al., *Neural Substrates of Choice Selection in Adults and Adolescents*, 45 Neuropsychologia 1270, 1270-1271 (2007); Spear, *supra* note 62, at 87-90.

67 Goldberg, *supra* note 61, at 144.

68 Steinberg, *supra* note 60, at 743; *see* Rhoshel Lenroot et al., *Sexual Dimorphism of Brain Developmental Trajectories During Childhood and Adolescence*, 36 Neuroimage 1065, 1065 (2007).

69 Steinberg, *supra* note 60, at 743; *see* Casey et al., *supra* note 61, at 245-246; Elizabeth Sowell et al., *Mapping Continued Brain Growth and Gray Matter Density Reduction in Dorsal Frontal Cortex: Inverse Relationships During Postadolescent Brain Maturation*, 21 J. Neurosci. 8819, 8828 (2001).

70 Steinberg, *supra* note 60, at 743; Spear, *supra* note 62, at 119-120, 125-126; Thomas Eluvathingal et al., *Quantitative Diffusion Tensor Tractography of Association and Projection Fibers in Normally Developing Children and Adolescents*, 17 Cerebral Cortex 2760, 2763-2764 (2007).

71 Steinberg, *supra* note 60, at 743; Leah Somerville et al., *A Time of Change: Behavioral and Neural Correlates of Adolescent Sensitivity to Appetitive and Aversive Environmental Cues*, 72 Brain & Cognition 124, 128-129 (2010) (noting importance of white-matter development and the "functional network [in] mediat[ing] the ability to exert control in the face of emotion").

72 Chein et al., *supra* note 35, at F7-F8; Steinberg, *supra* note 64, at 162; Spear, *supra* note 62, at 121-126.

73 *See, e.g.*, Eshel et al, *supra* note 66, at 1270-1271; Kathryn Modecki, *Addressing Gaps in the Maturity of Judgment Literature: Age Differences and Delinquency*, 32 Law & Hum. Behav. 78, 79-80 (2008); Steinberg et al., *supra* note 10, at 1765.

74 Steinberg, *supra* note 64, at 161.

75 Gogtay & Thompson, *supra* note 66, at 7; Casey et al., *supra* note 61, at 243; Spear, *supra* note 62, at 87-88.

76 Steinberg, *supra* note 64, at 161; *see* Somerville et al., *supra* note 71, at 126-127.

77 Steinberg, *supra* note 64, at 162.

78 *See supra* p. 24 & nn. 56-59.

79 Indeed, empirical studies evaluating the deterrent effect of laws mandating that juvenile offenders be transferred to the adult criminal justice system for certain crimes have concluded that the threat of adult criminal sanctions had no measurable effect on juvenile crime. *E.g.*, Simon Singer & David McDowall, *Criminalizing Delinquency: The Deterrent Effects of the New York Juvenile Offender Law*, 22 Law & Soc'y Rev. 521, 526-532 (1988) (comparing juvenile arrest statistics before and after enactment of New York's transfer legislation and finding little measurable impact on serious juvenile crime); Eric Jensen & Linda Metsger, *A Test of the Deterrent Effect of Legislative Waiver on Violent Juvenile Crime*, 40 Crime & Delinq. 96,100-102 (1994) (same for Idaho).

End of Document

APPENDIX B

IN THE CIRCUIT COURT FOR MCMINN COUNTY, TENNESSEE
AT ATHENS

AMOS BROWN (TDOC #287845),	)	FILED
	)	MAY 07 2018
Petitioner,	)	RHONDA [illegible]
	)	CIRCUIT COURT CLERK
	)	BY [illegible] D.C.
vs.	)	**No. 4-CR-64**
	)	
STATE OF TENNESSEE	)	
	)	
Respondent.	)	
	)	

PETITIONER'S NOTICE OF FILING OF DECLARATION OF DR. MICHAEL FREEMAN

Plaintiff Amos Brown gives notice that he is filing the attached Declaration of Dr. Julie A. Gallagher, an epidemiologist, in support of his claims in this case. Dr. Freeman's Declaration regards his expert opinion concerning the average life expectancy of prisoners serving life sentences in Tennessee, in support of Petitioner's claim that a 51-year mandatory minimum sentence of juvenile offenders such as Mr. Brown violates the Cruel and Unusual Punishment Clauses and other Clauses of the United States and Tennessee Constitutions.

Respectfully submitted,

BRADLEY A. MACLEAN, BPR #009562
Counsel for Plaintiff
1702 Villa Place
Nashville, TN 37212
Phone: 615-943-8716
Email: brad.maclean9@gmail.com

CERTIFICATE OF SERVICE

I hereby certify that on this 1st day of May, 2018, a true copy of the foregoing was served by first class mail and by email on:

Stephen D. Crump, Esq.
District Attorney General
93 Ocoee Street, Suite 2
Cleveland, TN 37311

Courtney N. Orr, Esq.
Assistant Attorney General
Criminal Appeals Division
P.O. Box 20207
Nashville, TN 37202-3491

Stephen Ross Johnson, Esq.
Ritchie, Dillard, Davies & Johnson, PC
606 W. Main Street, Suite 300
Knoxville, TN 37902

Bradley A. MacLean

IN THE CIRCUIT COURT FOR MCMINN COUNTY, TENNESSEE
AT ATHENS

AMOS BROWN (TDOC #287845),	)	
	)	
Petitioner,	)	
	)	
v.	)	**No.: 4-CR-64**
	)	
	)	
STATE OF TENNESSEE,	)	
	)	
	)	
Respondents.	)	
	)	

DECLARATION OF DR. MICHAEL FREEMAN

Pursuant to Tenn. R. Evid. 703 and Tenn. R. Civ. Pro. 72, Dr. Michael Freeman declares as follows:

1. I have been retained by Bradley A. MacLean, counsel for Petitioner Amos Brown, as an independent expert in the above-captioned matter. I make this Declaration based on my own personal knowledge and, if called as a witness, I could and would testify competently to the truth of the matters set forth herein.

2. A true and correct copy of my expert report, dated December 20, 2017, along with my current CV, is attached hereto. The information in my report and CV is true and correct to the best of my knowledge, information and belief.

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

Executed on December 20, 2017.

Michael D. Freeman

Dr. Michael Freeman

Forensic Research + Analysis

December 20, 2017

Bradley A. MacLean
1702 Villa Place
Nashville, Tennessee
Phone: (615) 943-8716
Email: brad.maclean9@gmail.com

RE: *Amos Brown v. State of Tennessee*
Amos Brown v. Tennessee Department of Correction, et al.

Dear Mr. MacLean,

I am in receipt of your correspondence and materials regarding the above-named actions. My report in this matter is in response to the questions that you have posed regarding sentencing practices in the Tennessee penal system. Specifically, I am responding to your questions regarding the 51-year life sentence, and how this mandatory minimum sentence affects the proportion of inmates with a life sentence who will die while serving the 51-year sentence (while incarcerated).

My methods and opinions in this case pertain to the field of epidemiology. Epidemiology is defined as the scientific study of disease and injury in populations, including prevalence, risk, and incidence in specific populations, and includes the study of survival and mortality risk. The methods applied in this report are consistent with those outlined in the Reference Guide on Epidemiology, from the Reference Manual on Scientific Evidence, published by the Federal Judicial Center and the National Academies of Science (3rd Edition, 2011), as well as in the text Forensic Epidemiology: Principles and Practice, published by Elsevier (2016).

Qualifications

I am a doctor of medicine and an epidemiologist, and my field of expertise is forensic medicine and forensic epidemiology. I hold the following academic degrees: a doctor of medicine degree from Umeå University, a Ph.D. in public health with a major focus in epidemiology from Oregon State University, and an MPH in epidemiology and biostatistics, also from Oregon State University, *inter alia*. I have completed a 2-year post-doctoral fellowship in forensic pathology at Umeå University in Sweden, and am currently an affiliate medical examiner with the Allegheny County Medical Examiner's office, a fellow of the Pathology section of the American Academy of Forensic Sciences (AAFS), and vice chair of of the US national standards board for medicolegal

death investigation for the AAFS.

I am a Fulbright Fellow, and hold a 3-year appointment (2017-20) with the United States Department of State as a Fulbright Specialist in the field of forensic medicine.

I serve as an Associate Professor of Forensic Epidemiology at Maastricht University Medical Center, an Affiliate Professor of Psychiatry at Oregon Health and Science University (OHSU) School of Medicine, and an Adjunct Professor of Forensic Medicine and Epidemiology in the Faculty of Health Sciences at Aarhus University. I have taught courses for the past 17 years in forensic medicine, forensic epidemiology, and injury epidemiology at OHSU.

I currently serve or have served as an associate editor or editorial board member of 13 peer-reviewed scientific journals, and have published approximately 180 scientific papers, abstracts, book chapters and books on topics largely related to scientific methods of causal evaluation. I am the editor of the textbook *Forensic Epidemiology: Principles and Practice* (Elsevier, 2016), the most comprehensive authority on the topic, and co-authored the chapter on survival analysis in that textbook.

I have provided testimony in more than 300 civil and criminal trials in state and Federal courts throughout the United States, Canada, Australia, and Europe. Please see my CV for further details.

Materials reviewed:

In forming my opinions in this matter, I have reviewed the following case-specific documents:

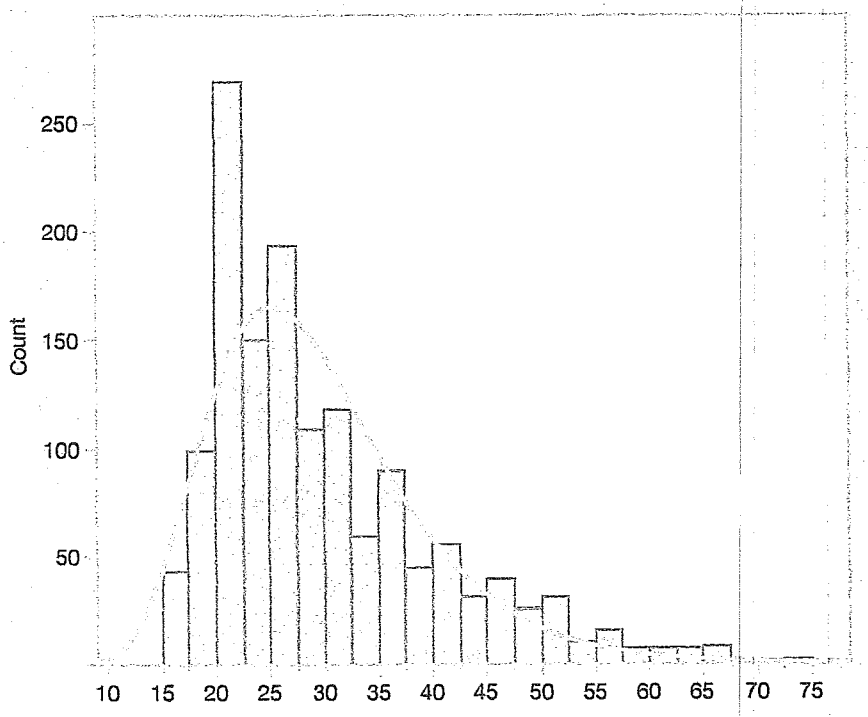
- The complaints in the subject matter
- Tennessee Department of Correction data on deaths while in custody
- Tennessee Department of Correction data on age at time of incarceration

Opinions

Following my review and analysis of data that I understand to be from the Tennessee Department of Correction (TDOC), I have arrived at the following opinions:

Age at time of incarceration among inmates sentenced to life

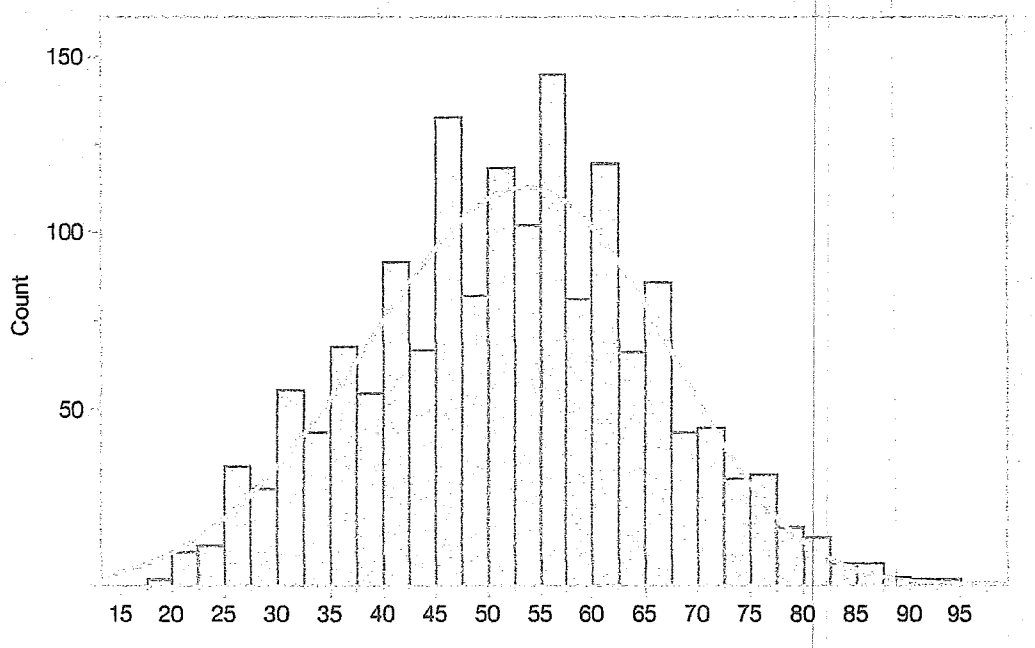
The average age at time of conviction among 1,395 inmates with a life sentence in the TDOC system is 29.5 years. It can be seen in the chart below that the distribution of age at time of sentencing is right skewed, meaning that tail of the curve stretches farther to the right of the average than to the left. The median age at time of life sentence is 27 (meaning that 50% of sentences are higher age and 50% are lower age); 25% are over the age of 35 and 10% are over 45.



Age of death among TDOC inmates

The average age of death among 1,575 inmates who have died in prison is 52 (tracked since 1991, and through 2015, and both prior to and after the 51 year life sentence minimum, enacted in 1995). Among these deaths, the median is also 52 years of age, and the top 25th and 10th percentiles are 61 and 69 years. Black inmates comprise 34% of the deaths, and almost all of the remaining deaths were among white inmates. Most (82%) of the deaths were ruled natural, 9% were due to unspecified illness, 3% suicided, 2% were murdered, and 1% died of HIV/ AIDS complications.

See the chart below for the distribution of age at time of death:



Conclusions:

A minimum 51-year sentence added to an average age at time of incarceration of 29.5 years results in an average age at release of 80.5 years among TDOC inmates. There is an approximately 1.5% probability that a TDOC inmate will live to this age, and thus a 1.5% that the average TDOC inmate sentenced to life will survive long enough to be released, and conversely, a 98.5% probability that they will not survive to release date.

Questions:

In the following section of this report I have responded to specific questions that you have posed.

Life Expectancy: Prison v. General Population

- Is life expectancy lower for individuals in prison compared to the general population in the United States? By how much? What is the average life expectancy for prisoners in the United States?

  Response: **Life expectancy is defined as the average age of death for a population. The precise life expectancy of the prison population is unknown, largely because most people who have been in prison will die after release, and the duration of incarceration has an effect on survival. Research conducted on the Georgia prison population has indicated an approximately 43% increased 15-year risk of death versus the never incarcerated population,[1] but this value doesn't translate to a life expectancy for the incarcerated population, as most inmates are released prior to death. A study of the prison population with a life sentence in Michigan reported an overall life expectancy of 58.1 years for all prisoners (56.0 for African-American males and 60.1 years for white males), and 50.6 years for prisoners sentenced as children, based on 400 deaths.[2] These life expectancies are approximately 15 years less than for the non-incarcerated population of the same race and gender.[3] Other researchers have reported similar findings; for every year spent behind bars roughly 2 years of life expectancy is lost in the imprisoned and paroled population,[4] and this life shortening effect is more pronounced in younger prisoners.[5]**

- Is there any reason to believe that life expectancy for inmates in TDOC custody would be longer than the average life expectancy for prisoners in the United States generally? Shorter?

  Response: **Based on the above reported data analysis, the average age at death (an approximation of life expectancy) is substantially less for TDOC inmates than**

[1] Spaulding AC et al. Prisoner survival inside and outside of the institution: implications for health-care planning. Am J Epidemiol. 2011;173(5):479-87.
[2] http://fairsentencingofyouth.org/wp-content/uploads/2010/02/Michigan-Life-Expectancy-Data-Youth-Serving-Life.pdf accessed January 2, 2017.
[3] https://www.cdc.gov/nchs/data/nvsr/nvsr64/nvsr64_11.pdf
[4] Patterson EJ. The dose-response of time served in prison on mortality: New York State, 1989-2003. Am J Public Health. 2013;103(3):523-8.
[5] Kouyoumdjian FG, Andreev EM, Borschmann R, Kinner SA, McConnon A. Do people who experience incarceration age more quickly? Exploratory analyses using retrospective cohort data on mortality from Ontario, Canada. PLoS One. 2017;12(4):e0175837.

inmates in the Michigan study. Part of this difference may be due to the fact that the Tennessee population has an approximately 2 year shorter life expectancy than the Michigan population, and part of it may be due to the fact that such a large proportion of inmates sentenced to life in prison in the TDOC are under the age of 25 at the time of incarceration (refer to the first chart above).

- What is the life expectancy for inmates in TDOC custody?

  Response: **See the previously described analysis. The only available proxy for life expectancy that is available for the TDOC life sentence population is the average age at death.**

- By how much is life expectancy lower for inmates in TDOC custody compared to the general population in Tennessee and the United States?

  Response: **The general population in Tennessee has a life expectancy of approximately 76 years, whereas the US population life expectancy is nearly 79 years. The average age at death of a TDOC inmate is 24 years less than for the general population in Tennessee.**

51 Year Sentence

- Given that individuals are required to serve 51 years prior to release, what percent of individuals serving that sentence would be expected to live longer than their sentence? Said otherwise, what is the likelihood that an individual sentenced at age 18 will survive his 51-year sentence? What is the likelihood for a 25 year old? A 35 year old?

  Response: **An individual sentenced at age 18 would be released at age 69, and 90% of the deaths in the TDOC occur prior to this age. Thus, there is a 10% probability that an 18 year old individual would survive to see their release. For a 25 and 35 year old the probabilities are less than 5% and 1%, respectively, that the inmates will survive to release date.**

- Is a 51-year sentence the equivalent of a life without parole sentence (i.e. is it the equivalent to a sentence under which the inmate will die in prison). Said otherwise, is there a meaningful difference between a 51 year sentence and a life without parole sentence?

Response: **A 1.5% average probability of surviving to release is the same thing as a 98.5% probability of dying before release. Thus, on average, in 98.5% of cases a 51-year sentence is equivalent to a life without parole sentence.**

- If an inmate lives beyond the 51 years to which he is sentenced, how many years do you estimate he will live?

  Response: **As mentioned above, the probability that the average inmate would survive to 81 years of age (average age of release) is approximately 1.5%. The US Life Tables indicate that an 81 year-old man has a life expectancy of approximately 8 years. The 1.5% of prisoners who survive 51 years to their release would thus live 8 or fewer years after release, on average.**

- Does a 51-year sentence provide a meaningful opportunity for release?

  Response: **As the opportunity for release is available to fewer than 1 in 65 prisoners sentenced to life, the answer is no. A 51-year life sentence is 98.5% identical to a life sentence without the possibility of parole.**

The preceding opinions and responses were given as reasonable medical and scientific probabilities.

Very truly yours,

Michael D. Freeman, MedDr PhD MPH FAAFS
Forensic Medicine and Epidemiology

CURRICULUM VITAE

MICHAEL D. FREEMAN

December 2017

ADDRESS
4500 Kruse Way, Plaza I, Suite 385
Lake Oswego, Oregon 97035
Tel 971-255-1008 Fax 971-255-1046
e-mail: forensictrauma@gmail.com
m.freeman@maastrichtuniversity.nl

EDUCATION

Doctor of Medicine (Med.Dr.)
Faculty of Medicine, Umeå University, Umeå, Sweden

Doctor of Philosophy (Ph.D.) Public Health/ Epidemiology
Oregon State University, Corvallis, Oregon

Master of Public Health (M.P.H.), Epidemiology/ Biostatistics
Oregon State University, Corvallis, Oregon

Doctor of Chiropractic (D.C.)
University of Western States, Portland, Oregon

Bachelor of Science (B.S.) General Science
University of Oregon, Eugene, Oregon

FELLOWSHIPS

Fulbright Specialist Roster
Bureau of Educational and Cultural Affairs and World Learning,
United States Department of State, 2017-2020 tenure

Postdoctoral Fellowship
Forensic Pathology
Section of Forensic Medicine, Department of Community Medicine and Rehabilitation, Umeå University, Umeå, Sweden 2014-2015

ACADEMIC POSITIONS

Associate Professor of Forensic Medicine – 2015-2018
CAPHRI School for Public Health and Primary Care
Maastricht University Medical Center
Maastricht, The Netherlands

Affiliate Professor of Epidemiology – 2010 to 2015
Department of Public Health and Preventive Medicine
School of Medicine, Oregon Health & Science University
Portland, Oregon

Affiliate Professor of Psychiatry – 2011 to present
Department of Psychiatry
School of Medicine, Oregon Health & Science University

Portland, Oregon
Clinical/Affiliate Associate Professor – 2005-10
Department of Public Health and Preventive Medicine
School of Medicine, Oregon Health & Science University
Portland, Oregon
Clinical Assistant Professor – 1997-2005
Department of Public Health and Preventive Medicine
School of Medicine, Oregon Health & Science University
Portland, Oregon
Adjunct Professor of Forensic Epidemiology and Traumatology – 2012-17
Department of Forensic Medicine
Faculty of Health Sciences, Aarhus University
Aarhus, Denmark
Adjunct/Honorary Associate Professor of Epidemiology and Traumatology – 2012-17
Department of Forensic Medicine
Faculty of Health Sciences, Aarhus University
Aarhus, Denmark
Adjunct Associate Professor of Forensic Medicine and Epidemiology – 2005-12
Institute of Forensic Medicine
Faculty of Health Sciences, Aarhus University
Aarhus, Denmark
Adjunct Professor – 2015 to present.
University of Western States
Portland, Oregon

EDITORIAL ACTIVITIES

Co-Editor in Chief:
Journal of Whiplash-Related Disorders 1999-2006
Associate Editor:
OA Epidemiology, 2014-present
J of Forensic Biomechanics, 2010-present
The Spine Journal 2007-present
PM&R, official scientific journal of the American Academy of Physical Medicine and Rehabilitation, 2008-present
Scandinavian Journal of Forensic Medicine, 2012-present
Editorial Board Member:
The Spine Journal 2004-present
International Research Journal of Medicine and Medical Sciences
Egyptian Journal of Forensic Sciences 2010-present
Journal of Case Reports Practice 2014-present
Austin Journal of Public Health & Epidemiology 2014-2016
Edorium Journal of Public Health 2014-present
Editorial Committee Member:
Spineline 2004-2009
Peer reviewer:
BMC Public Health
BMC Research Notes
Annals of Epidemiology (outstanding reviewer status 2015)
Orthopedics
Spine
The Spine Journal
Lancet
Mayo Clinic Proceedings
Annals of Biomechanical Engineering

Journal of the American Board of Family Medicine
Journal of Forensic and Legal Medicine
Acta Neurologica Scandanavica
Medical Science Monitor
Pain Research & Management
Journal of Back and Musculoskeletal Rehabilitation
American Society for Testing and Materials (ASTM)
Biosecurity & Bioterrorism
Annals of Medical and Health Sciences Research
Neurorehabilitation and Neural Repair
International Research Journal of Medicine and Medical Sciences
Jurimetrics
Law, Probability, and Risk
International Journal of Molecular Sciences
Journal of Rehabilitation Medicine
Arthritis
BMC Pediatrics
Journal of Back and Musculoskeletal Rehabilitation
Diagnostic and Interventional Radiology
Healthcare

COURSES TAUGHT

PHPM 574 Forensic & Trauma Epidemiology
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 2006-2013

Principles of Forensic Medicine and Forensic Epidemiology
Forensic Psychiatry Fellowship
Department of Psychiatry
Oregon Health & Science University School of Medicine
Portland, Oregon – 2011 to present

PHPM 503 Thesis Advising
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 2005-present

PHPM 507 Injury and Trauma Epidemiology
Department of Public Health and Preventive Medicine
Oregon Health & Science University School of Medicine
Portland, Oregon 1999 – 2005

Forensic Epidemiology and Bioterrorism
Charles County Department of Public Health
College of Southern Maryland, Waldorf, Maryland 2014

ACTIVITIES and HONORS

Keynote speaker, Gran Sesión de Epidemiología Forense. November 18, 2016 Universidad Libre, Seccional Cali, Colombia.

Vice Chair, American Academy of Forensic Sciences Standards Board Medicolegal Death Investigation Consensus Body – 2016-present

Member, American Academy of Forensic Sciences Standards Board Medicolegal Death Investigation Consensus Body – 2016-present

Affiliate Medical Examiner, Allegheny County, Pennsylvania, 2014-present

Member, Scientific Advisory Board, International Conference on Forensic Inference and Statistics. August 2014, Leiden, The Netherlands

Reviewer, National Aeronautical Space Administration (NASA) 2011
Past president, International Cellular Medicine Society, 2009 to 2012
Founding member, International Cellular Medicine Society, 2009
Member, Research Planning Committee, North American Spine Society 2007-2009
Member, Complementary Medicine Committee, North American Spine Society 2007-2009
Special Deputy Sheriff (Forensics), Vehicular Homicide Investigator, Clackamas County, Oregon, 2007-2009
Member, Crash Reconstruction and Forensic Technology (CRAFT) multidisciplinary law enforcement fatal crash investigation team, Clackamas County, Oregon, 2002-2013
Consultant Forensic Trauma Epidemiologist to the Medical Examiner Division of the Oregon Department of State Police – Occupant Kinematics, 1999-2006
Deputy Medical Examiner, Marion County, Oregon. 2000-2005
Moderator, Engineering sciences section, American Academy of Forensic Sciences 62nd Annual Meeting, Seattle, WA 2010
Co-Chair, International Whiplash Trauma Congress V, Lund, Sweden. 2011
Co-Chair, International Whiplash Trauma Congress IV, Miami, FL. October 2007.
Co-Chair, International Whiplash Trauma Congress III, Portland, OR. June 2006.
Co-Chair, International Whiplash Trauma Congress II, Breckenridge, CO. February 2005.
Co-Chair, International Whiplash Trauma Congress I, Denver, CO. October, 2003
Co-Chair, Forensic Section, International Traffic Medicine Association. Budapest, Hungary. September, 2003
Member, Blue Ribbon Panel Congressional Task Force on roller coaster-induced brain injury. Funded by a grant from the National Institute of Child Health and Human Development 2002-2003
President, Spinal Injury Foundation. Denver, CO 2002-2009
Member, Marion-Polk County C.R.A.S.H. Team - Occupant Kinematics Consultant 1999-2004
Scientific Chair, North American Whiplash Trauma Congress. Victoria, British Columbia 1999

BOARD CERTIFICATION AND ORGANIZATIONS

American Academy of Forensic Sciences, Pathology/ Biology section
Fellow (2016-present)
Member (2008-2016)
Faculty of Forensic & Legal Medicine, Royal College of Physicians, Affiliate Member
ACTAR Accredited Crash Reconstructionist, Accreditation Commission for Traffic Accident Reconstruction, Accreditation #1581
Crash Data Retrieval Technician I & II
Certification in basic and advanced crash reconstruction - Northwestern University
Diplomate, American Academy of Pain Management
Member, American College of Epidemiology
Member, Association for the Advancement of Automotive Medicine
Member, Sigma Xi Scientific Honor Society
Member, Society of Automotive Engineers
Past member, International Traffic Medicine Association
Fellow, International College of Chiropractic
Inactive member, North American Spine Society
Past member, Forensic Accident Reconstructionists of Oregon

GRANTS

2017-2020 Fulbright scholarship, Fulbright Specialist program, Bureau of Educational and Cultural Affairs and World Learning, United States Department of State.
2015 National Science Foundation Industry/University Cooperative Research Centers Program, NSF 13-594 Planning Grant: I/UCRC for Advanced Research in Forensic Science, National Center for Research on Forensic Epidemiology. Principal Investigator.

2011-2013 World Health Organization – research grant for Rwandan study of relationship between genocide and suicide and homicide victimization and offending. $50,000. Project No: AFRWA 1005685, Award No: 53975.

2010-2015 Centers for Disease Control (Administered by National University of Rwanda and OHSU) SPH/CDC $200,000 over 4 years.

2002-2003 National Institute of Child Health and Human Development – Blue Ribbon Task Force on Roller Coaster Associated Brain Injury. $75,000.

DISSERTATION SUPERVISION/MENTORING

Paul Nolet MPH, MSc, DC – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2017 to present)

Huijie Wang B.Med,, M.Med. – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2017 to present)

Dritan Bijko MD MSc – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2017 to present)

Putri Dianita MD – PhD candidate, CAPHRI School for Public Health and Primary Care, Maastricht University Medical Center (2015 to present)

Frank Franklin Ph.D., J.D. (2013), Earle Mack School of Law, Drexel University

Bonnie Colville-Ebeling MD – PhD candidate (2012 to present) University of Copenhagen, Faculty of Health Sciences, Department of Forensic Medicine

Dimitrios Papadakis BSc, MRes, Dr.rer.nat. (2012-present) independent mentoring

Wendy Leith MS – MPH (2015) Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Konrad Dobbertin – MPH (2011) Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Apostolo Alexandridis – MPH (2011) - Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Wilson Rubanzana MD – PhD (2016) National University of Rwanda, School of Public Health, Kigali, Rwanda

Catherine Maddux-Gonzalez – MPH (2009) – Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

Laura Criddle MS, RN – PhD (2008) Oregon Health & Science University School of Medicine, School of Nursing

Peter Harmer PhD – MPH (2006) Department of Public Health & Preventive Medicine, Oregon Health & Science University School of Medicine

PUBLICATIONS

Peer-reviewed journal articles

1. **Freeman MD,** Leith WM. The epidemiology of tire failure-related traffic crashes (in review).

2. **Freeman MD.** A practicable and systematic approach to medicolegal causation. *Orthopedics* (in press)

3. Centeno C, Markle J, Dodson E, Stemper I, **Freeman MD.** A prospective multi-site study of treatment for lumbar radiculopathy using epidural injection of platelet lysate (in review).

4. Centeno C, Markle J, Dodson E, Stemper I, Hyzy M, Williams C, **Freeman MD.** The safety and efficacy of using lumbar epidural injection of platelet lysate for treatment of radicular pain. J Exp Orthopaedics (*in press*)

5. Centeno C, Markle J, Dodson E, Stemper I, Williams C, Hyzy M, Ichim T, **Freeman MD.** Treatment of lumbar degenerative disc disease-associated radicular pain with culture-expanded autologous mesenchymal stem cells *J Translational Medicine* (in press).

6. Williams KE. **Freeman MD.** The role of the medical examiner/ coroner system in creating a public database for surveillance and information sharing on drug overdose deaths. *Academic Forensic Pathology.* 2017;7(1):60-72.

7. Leith W, Lambert W, Boenhlein J, **Freeman MD.** The association between gabapentin and suicidality in bipolar patients (in review).

8. Centeno C, Markle J, Dodson E, Stemper I, Williams C, Hyzy M, **Freeman MD.** Symptomatic anterior cruciate ligament tears treated with percutaneous injection of autologous bone marrow concentrate: a non-controlled prospective registry study. *BMC Musculoskeletal Disorders* (in review).

9. Centeno C, Markle J, Dodson E, Stemper I, **Freeman MD.** A prospective multi-site study of treatment for lumbar radiculopathy using epidural injection of platelet lysate. *J Pain Research* (in review).

10. **Freeman MD,** Goodyear S, Leith W. Risk factors for neonatal brachial plexus injury; a multistate epidemiologic study of matched maternal and newborn discharge records. *Int J Gynecology & Obstetrics* 2017;136(3):331-336.

11. **Freeman MD, Zeegers M.** Forensic Epidemiology: An evidence-based system for analyzing individual causation in a medicolegal setting. *Austin J Public Health Epidemiol* 3(3):2016. ISSN: 2381-9014

12. Westergren H, Larson L, Carlsson A, Joud A, **Freeman MD,** Malmstrom E-M. Sex-based differences in chronic pain distribution in a cohort of patients with post-traumatic neck pain. *Disabil Rehabil* 2017 DOI: 10.1080/09638288.2017.1280543

13. Nyström A, **Freeman MD.** Central sensitization is modulated following trigger point anesthetization in patients with chronic pain following whiplash trauma. A double-blind, placebo-controlled, cross-over study. *Pain Med* 2017;0:1-6.

14. **Freeman MD,** Zeegers M. Principles and applications of forensic epidemiology in the medicolegal setting. *Law, Probability, & Risk* 2015; doi:10.1093/lpr/mgv010.

15. Centeno CJ, Al-Sayegh H, **Freeman MD** et al. A multi-center analysis of adverse events among 2,372 adult patients undergoing adult autologous stem cell therapy for orthopedic conditions. *International Orthopedics* DOI 10.1007/s00264-016-3162-y.

16. **Freeman MD.** Medicolegal causation analysis of a lumbar spine fracture following a low speed rear impact traffic crash. *J Case Rep Prac* 2015; 3(2): 23-29.

17. Uhrenholt L, **Freeman MD,** Webb A, Pedersen M, Thorup-Boel LW. Fatal subarachnoid hemorrhage associated with internal carotid artery dissection resulting from whiplash trauma. *Forens Sci Med Path* 2015;11(4):564-9.

18. Rubanzana W, Hedt-Gauthier B, Ntanganira J, **Freeman MD.** Exposure to effects of genocide as a risk factor for homicide perpetration in Rwanda: A population-based case-control study. *J Interpersonal Violence* 2015;pii: 0886260515619749. [Epub ahead of print] PubMed PMID: 26681788.

19. Centeno CJ, Al-Sayegh H, Bashir J, **Freeman MD.** A prospective study of the safety and efficacy of autologous bone marrow concentrate for the treatment of rotator cuff tears and shoulder osteoarthritis *J Pain Res* 2015;8:1-8.

20. Centeno CJ, Al-Sayegh H, Bashir J, **Freeman MD.** A dose response analysis of bone marrow concentrate injections for knee osteoarthritis. *BMC Musculoskeletal Disorders (Section: Orthopedics and biomechanics)* 2015;16:258. doi: 10.1186/s12891-015-0714-z.

21. Rubanzana W, Ntanganira J, **Freeman MD,** Hedt-Gauthier B, Risk factors for homicide victimization in post-genocide Rwanda: a population -based case- control study. *BMC Public Health* 2015;15(1):809.

22. Rubanzana W, Hedt-Gauthier B, Ntanganira J, **Freeman MD**. Exposure to genocide as a risk factor for suicide in Rwanda. *J Epidemiol Community Health* 2015 Feb;69(2):117-22.

23. Westergren H, **Freeman MD,** Malmström E-M. The whiplash enigma: still searching for answers. *Scand J Pain* 2014; http://dx.doi.org/10.1016/ j.sjpain.2014.08.003.

24. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Efficacy and Safety of Bone Marrow Concentrate for Osteoarthritis of the Hip; Treatment Registry Results for 196 Patients. *J Stem Cell Res Ther* 2014;4:242. doi: 10.4172/2157-7633.1000242

25. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Efficacy of autologous bone marrow concentrate for knee osteoarthritis with and without adipose graft. *Biomed Res Int* 2014. doi:10.1155/2014/370621

26. Centeno CJ, Pitts J, Al-Sayegh H, **Freeman MD.** Anterior cruciate ligament tears treated with percutaneous injection of autologous bone marrow nucleated cells; a pilot study. *J Pain Res* 2015;8:1–11.

27. **Freeman MD**, Cahn PJ, Franklin FA. Applied forensic epidemiology. Part 1: medical negligence. *OA Epidemiology* 2014;2(1):2.

28. Koehler S, **Freeman MD**. Forensic epidemiology; a methodology for investigating and quantifying specific causation. *Forens Sci Med Path* 2014 Jun;10(2):217-22.

29. Centeno CJ, **Freeman MD**. Percutaneous injection of autologous, culture-expanded mesenchymal stem cells into carpo-metacarpal hand joints: A case series with an untreated comparison group. *Wien Med Wochenschr* 2013:DOI 10.1007/s10354-013-0222-4

30. **Freeman MD**, Eriksson A, Leith W. Head and neck injury patterns in fatal falls: epidemiologic and biomechanical considerations. *J Forensic Legal Med* 2014;21:64-70.

31. Colville-Ebeling B, **Freeman MD**, Banner J, Lynnerup N. Autopsy practice in forensic pathology – evidence-based or experience-based? A review of autopsies performed in a case of multiple, simultaneous deaths. *J Forensic Legal Med* 2014;22:33-6.

32. **Freeman MD**, Eriksson A, Leith W. Injury pattern as an indication of seat belt failure in ejected vehicle occupants *J Forensic Sci* 2014; 59(5):1271-4.

33. Dobbertin KM, **Freeman MD**, Lambert WE, Lasarev MR, Kohles SS. The relationship between vehicle roof crush and head, neck and spine injury in rollover crashes. *Accid Anal Prev* 2013;58:46-52.

34. Centeno CJ, Schultz JR, Cheever M, **Freeman M**, Faulkner S, Robinson S. A Case Series of Percutaneous Treatment of Non-Union Fractures with Autologous, Culture Expanded, Bone Marrow Derived, Mesenchymal Stem Cells and Platelet Lysate. *J Bioengineer & Biomedical Sci S2:007* doi:10.4172/2155- 9538.S2-007

35. Woodham M, Woodham A, Skeate JG, **Freeman MD**. Long-Term Lumbar Multifidus Muscle Atrophy Changes Documented With Magnetic Resonance Imaging; A Case Series. *Radiology Case Reports* 2014;8(5):27-34

36. Wendlova J, **Freeman MD**. The Slovak Regression Model of Fall-Related Femoral Neck Fracture Risk. *Journal of Forensic Biomechanics* Vol. 4 2013), Article ID 235595, 5 pages doi:10.4303/jfb/235595

37. **Freeman MD**, Dobbertin K, Kohles SS, Uhrenholt L, Eriksson A. Serious head and neck injury as a predictor of occupant position in fatal rollover crashes. *Forensic Sci Int* 2012;222:228–33.

38. **Freeman MD**, Kohles SS. An examination of the threshold criteria for the evaluation of specific causation of mesothelioma following a history of significant exposure to chrysotile asbestos-containing brake dust, *Int J Occ Env Hlth* 2012;18(4):329-36.

39. **Freeman MD**, Fuerst M. Does the FDA have regulatory authority over adult autologous stem cell therapies? FDCA 21 CFR 1271 and the Emperor's New Clothes. *J Transl Med* 2012;10(1):60.

40. **Freeman MD**, Everson T, Kohles SS. Forensic epidemiologic and biomechanical analysis of a pelvic cavity blowout injury associated with ejection from a personal watercraft (jet-ski). *J Forens Sci* 2012 doi: 10.1111/j.1556-4029.2012.02250.x

41. **Freeman MD**, Kohles SS. Plasma levels of polychlorinated biphenyls, non-Hodgkin lymphoma, and causation. *J Environ Public Health* 2012;2012:258981. doi: 10.1155/2012/258981. Review.

42. Centeno CJ, Fuerst M, Faulkner SJ, **Freeman MD**. Is cosmetic platelet-rich plasma a drug to be regulated by the Food and Drug Administration? *J Cosm Derm* 2011;10:171–3.

43. Centeno CJ, Schultz JR, Cheever M, **Freeman M**, Faulkner S, Robinson S, Hanson R. Safety and Complications Reporting Update on the Re-Implantation of Culture-Expanded Mesenchymal Stem Cells Using Autologous Platelet Lysate Technique. *Cur Stem Cell Res & Ther* 2011;6(4):XX

44. **Freeman MD**, Kohles SS. Application of the Hill Criteria to the Causal Association of Post-Traumatic Headache and Assault. *Egypt J Forensic Sci* 2011;1:35-40.

45. **Freeman MD**, Kohles SS. Application of the Bradford-Hill Criteria for Assessing Specific Causation in Post-Traumatic Headache. *Brain Inj Prof* 2011;8(1):26-8.

46. **Freeman MD**, Kohles SS. An Evaluation of Applied Biomechanics as an adjunct to systematic specific causation in forensic medicine. *Wien Med Wochenschr* 2011;161:1-11.

47. Uhrenholt L, **Freeman MD**, Jurik AG, Jensen LJ, Gregersen M, Boel LW, Kohles SS, Thomsen AH. Esophageal injury in fatal rear-impact collisions. *Forensic Sci Int* 2011;206(1-3):e52-7.

48. **Freeman MD**. A Bayesian assessment of unexplained fracture as a forensic test of child abuse; quantification of uncertainty using the Error Odds approach. *Acta Medicinae Legalis et Socialis* 2010:179-84.

49. Nystrom NA, Champagne LP, **Freeman MD**, Blix E. Surgical fasciectomy of the trapezius muscle combined with neurolysis of the spinal accessory nerve; results and long-term follow-up in 30 consecutive cases of refractory chronic whiplash syndrome. *J Brachial Plexus and Peripheral Nerve Injury* 2010:5;7.

50. Centeno CJ, Schultz J, Cheever M, Robinson B, **Freeman MD**, Marasco W. Safety of autologous MSC transplantation: an in vivo MRI study of transplanted MSCs culture-expanded using a novel, platelet-lysate technique. *Cur Stem Cell Res & Ther* 2010;5:81-93.

51. Dagenais S, Gay RE, Tricco A, Mayer, JM, **Freeman MD**. North American Spine Society Contemporary Concepts in Spine Care: Spinal Manipulation Therapy for Acute Low Back Pain *Spine J* 2010 Oct;10(10):918-40.

52. Uhrenholt L, Schumacher B, **Freeman MD**. Road traffic fatalities in Aarhus Police District in 2000-2004 - medical investigations and legal consequences. *Ugeskr Laeger*. 2010 Sep 27;172(39):2683-2687. Danish

53. **Freeman MD**, Woodham M, Woodham A. The role of the lumbar multifidus in chronic low back pain; a review. *PM R* 2010 Feb;2(2):142-6.

54. **Freeman MD**, Centeno CJ, Kohles SS. A systematic approach to clinical determinations of causation in symptomatic spinal disc injury following motor vehicle crash trauma. *PM R* 2009;1(10):951-6.

55. **Freeman MD**, Rosa S, Harshfield D, Smith F, Bennett R, Centeno CJ, Kornel E, Nystrom A, Heffez D, Kohles SS. A case-control study of cerebellar tonsillar ectopia and head/neck (whiplash) trauma. *Brain Injury* 2010;24(7-8):988-94.

56. **Freeman MD**, Kohles SS. Applications and limitation of forensic biomechanics; a Bayesian perspective. *J Forensic Legal Med* 2010;17:67-77.

57. **Freeman MD**, Nystrom A, Centeno C, Hand M. Chronic whiplash and central sensitization; do a trigger points play an important role in pain modulation? *J Brachial Plex Peripher Nerve Inj* 2009 Apr 23;4:2.

58. **Freeman MD**, Hand ML, Rossignol AM. Applied Forensic Epidemiology: A Bayesian evaluation of forensic evidence in a vehicular homicide investigation. *J Forensic Legal Med* 2009;16(2):83-92.

59. Centeno CJ, Busse D, Kisiday J, Keohan C, **Freeman M**, Karli D Regeneration of meniscus cartilage in a knee treated with percutaneously implanted autologous mesenchymal stem cells. *Med Hypothese*. 2008 Dec;71(6):900-8.

60. Centeno CJ, Schultz J, **Freeman M**. Sclerotherapy of Baker's cyst with imaging confirmation of resolution. *Pain Physician* 2008 Mar-Apr;11(2):257-61.

61. Centeno CJ, Busse D, Kisiday J, Keohan C, **Freeman M**, Karli D. Increased knee cartilage volume in degenerative joint disease using percutaneously implanted, autologous mesenchymal stem cells. *Pain Physician* 2008 May;11(3):343-53.

62. **Freeman MD**, Centeno CJ. A fatal case of secondary gain; a cautionary tale. *Amer J Case Reports* 2008;9:97-103.

63. Centeno CJ, Elkins W, **Freeman M**, Elliott J, Sterling M, Katz E. Total Cervical Translation as a Function of Impact Vector as Measured by Flexion-Extension Radiography *Pain Physician* 2007 Sep;10(5):667-71.

64. **Freeman MD**, Rossignol AC, Hand M. Forensic Epidemiology: A systematic approach to probabilistic determinations in disputed matters. *J Forensic Legal Med* 2008;15(5):281-90.

65. Centeno CJ, Kisiday J, **Freeman MD**, Shultz JR. Partial regeneration of the human hip via autologous bone marrow nucleated cell transfer: a case study. *Pain Physician* 2006;9:135-7.

66. Croft AC, **Freeman MD**. Correlating crash severity with injury risk, injury severity, and long-term symptoms in low velocity motor vehicle collisions. *Med Sci Monit* 2005 Oct;11(10):RA316-21. Epub 2005 Sep 26.

67. **Freeman MD**, Croft AC, Nicodemus CN, Centeno CJ, Welkins WL. Significant spinal injury resulting from low-level accelerations: A case series of roller coaster injuries. *Arch Phys Med Rehab* November 2005;86:2126-30.

68. **Freeman MD**, Croft AC, Rossignol AC, Elkins W. Chronic neck pain and whiplash: a case/control study of the relationship between acute whiplash injuries and chronic neck pain. *Pain Res Manag* 2006;11(2):79-83.

69. Centeno CJ, **Freeman MD**, Welkins WL. A review of the literature refuting the concept of minor impact soft tissue injury. *Pain Res Manag* 2005;10(2):71-4.

70. Centeno C, Elliot J, Elkins W, **Freeman M**. A prospective case series of fluoroscopically guided cervical prolotherapy for instability with blinded pre and post radiographic reading. *Pain Physician* 2005;8(1):

71. Centeno CJ, Elkins WL, **Freeman M**. Waddell's signs revisited? *Spine* 2004 Jul 1;29(13):1392

72. **Freeman MD**, Nelson C. Injury Pattern Analysis as a means of driver identification *Laboratory Medicine* 2004;35(8):502-5.

73. **Freeman MD**, Olson D. Hemifacial tic following a low-speed motor vehicle crash. *J Whiplash Rel Dis* 2004;3(1).

74. **Freeman MD**, Nelson C. Injury pattern analysis as a means of driver identification in a vehicular homicide; a case study. *Forensic Examiner* Spring 2004;13(1):24-8.

75. Croft AC, Haneline MT, **Freeman MD**. Low speed frontal crashes and low speed rear crashes: is there a differential risk for injury? *Annu Proc Assoc Adv Automot Med. 2002;46:79-91.*

76. Croft AC, Herring P, **Freeman MD**, Haneline MT: The neck injury criterion (NIC): future considerations. *Accid Anal Prev* 2002;34(2):247-55.

77. **Freeman MD**, Croft AC, Rossignol AM. A critical evaluation of the methodology of a low back pain clinical trial *J Manipulative Physio Ther* 2000; 23(5):363-4.

78. **Freeman MD**, Croft AC, Rossignol AM, Weaver DS, Reiser M. A review and methodologic critique of the literature refuting whiplash syndrome. *Spine* 1999;24(1):86-98.

79. **Freeman MD**, Croft AC, Rossignol AM. Whiplash Associated Disorders (WAD) - Redefining Whiplash and its Management" by the Quebec Task Force: A Critical Evaluation. *Spine* 1998;23(9):1043-9.

80. **Freeman MD**, Fox DD, Richards TR. The superior intracapsular ligament of the sacroiliac joint: confirmation of Illi's ligament. *J Manipulative Physiol Ther* 1990;13(7):374-90.

Non-peer-reviewed publications

1. **Freeman MD**. The problem with probability. *Trial* March 2006, 58-61.

2. Croft AC, **Freeman MD**. Auto insurers and their new role in whiplash prevention—new rules, new risks, new tests. *Forum* 35(3):9-13, 2005.

3. O'Shanick G, Varney N, **Freeman MD**, et al. Blue Ribbon Panel Review of the Correlation Between Brain Injury and Roller Coaster Rides (Report to US Congress, Funded by the National Institute of Child Health and Human Development). February 25, 2003.

4. Croft AC, Herring P, **Freeman MD**, Centeno C, Haneline MT, Baric JJ: Late (chronic) whiplash injury. Public health perspectives amidst a controversial literature. JACA 40(8):26-32, 2003.

5. **Freeman MD**. Don't fall for defense fallacies. *Trial* 2000.

6. Seroussi R, **Freeman MD**. A review of original research by Brault et al. "Clinical response of human subjects to rear-end automobile collisions" *Injury Forum* 2000;2(5):49-50.

7. **Freeman MD**. The epidemiology of acute and chronic whiplash injury in the U.S. Proceedings of HWS-Distorsion (Schleudetrauma) & Leichte Traumatische, Hirnverletzung. Invaliditat und Berufliche Reintegration. Basel, Switzerland. June 29-30, 2000.

8. **Freeman MD**. Meta-analysis of whiplash prognosis studies. Proceedings of Whiplash 2000, Bath, England. May 16-18, 2000. pp 102-24.

9. **Freeman MD**, Croft AC, Reiser M. [reprint of A review and methodologic critique of the literature refuting whiplash syndrome] *Trial* March 1999

10. Croft AC, **Freeman MD**. From railway spine to whiplash. *Topics in Clinical Chiropractic* (Trauma) 1998;5(3):54-61.

11. **Freeman MD**, Croft AC, Reiser M. Die epidemiologie des Schleudertraumas - wo liegt die Schelle Zur Verletzung? (The epidemiology of whiplash - is there a reliable threshold for whiplash injury?) *HWS-Distortion (Schleudetrauma) & Leichte Traumatische, Hirnverletzung.* Edited by Ettlin TM and Mürner J. June 25-6, 1998:99-118.

12. **Freeman MD**. The first CIREN conference: motor vehicle crash-related trauma and biomechanical engineering. *JACA* 1998;35(4):54-61.

Theses

1. **Freeman MD.** *The Role of Forensic Epidemiology in Evidence-Based Forensic Medical Practice.* Thesis for completion of Doctor of Medicine degree, Umeå University. Responsible publisher under Swedish law: the Dean of the Medical Faculty of Umeå University, Printed by: Print och Media, Umeå, Sweden 2013. ISBN: 978-91-7459-729-5 ISSN: 0346-6612

2. **Freeman MD.** *A study of chronic neck pain and whiplash injuries.* Thesis for completion of Doctor of Philosophy degree, Oregon State University. *UMI Dissertation services*, Ann Arbor, MI. 1998:9820108.

3. **Freeman MD.** *Analysis of lumbar spinal strength using the doubly multivariate repeated measures design.* Thesis for completion of Master of Public Health degree, Oregon State University. 1995 (unpublished)

Books

1. **Freeman MD**, Zeegers M, Eds. Forensic Epidemiology: Principles and Practice. Elsevier, Amsterdam, NL. 2016

2. Nordhoff L, **Freeman MD**, Siegmund GP. Human Subject Crash Testing: Innovations and Advances. Society of Automotive Engineers, Detroit MI 2007

3. Berardinelli D, **Freeman MD**, DeShaw A. From Good Hands to Boxing Gloves: How Allstate Changed Casualty Insurance in America. Trial Guides 2008

4. **Freeman MD**. Litigating Major Auto Injury and Death Cases; Forensic Science (volume 2 of Litigating Major Auto Injury and Death Cases, Koehler K, Freeman MD). ThomsonWest:2006

5. **Freeman MD**. Litigating Minor Impact Soft Tissue Cases; Forensic Medicine (volume 2 of Litigating Minor Impact Soft Tissue Cases, Koehler K and Freeman MD). ThomsonWest 2001.

Book Chapters

1. **Freeman MD**. Cervical Sprain and Strain. Medscape. Updated April 2016. Available at: http://emedicine.medscape.com/.

2. **Freeman MD**, Franklin FA. Criminal Investigation. Chapter 15, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

3. **Freeman MD**, Franklin FA, Cahn P. Medical Negligence Investigation. Chapter 14, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

4. **Freeman MD**, Franklin FA. Consumer Product Defect Investigation. Chapter 13, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

5. **Freeman MD**. Motor Vehicle Defect Investigation. Chapter 12, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

6. **Freeman MD**. Traffic Injury Causation Investigation. Chapter 11, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

7. Tolley HD, Barnes J, **Freeman MD**. Survival Analysis. Chapter 10, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

8. Faure M, Visscher L, Zeegers M, **Freeman MD**. The Role of the Expert Witness. Chapter 5, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

9. Zeegers M, Bours M, **Freeman MD**. Methods used in Forensic Epidemiologic Analysis. Chapter 3, in Forensic Epidemiology; Principles and Practice. Freeman M, Zeegers M, Eds. Elsevier, Amsterdam, NL. 2016.

10. **Freeman MD**, Haneline M. Unintentional and Intentional Injuries. Chapter 7, in An Introduction to Public Health in Chiropractic, Jones & Bartlett.

11. **Freeman MD**, Centeno CJ, Kornel E. Acute cervical spine trauma. British Medical Journal, *BMJ in Practice*. Available online at: https://online.epocrates.com/noFrame/showPage.do?method=diseases&MonographId=944

12. Hunter OK, **Freeman MD**. Cervical Sprain and Strain. eMedicine from WebMD. Updated July 15, 2009. Available at: http://emedicine.medscape.com/article/306176-overview.

13. **Freeman MD**. Forensic considerations in the mild traumatic brain injury case. In: Mild Traumatic Brain Injury: Onset, Consequences, & Outcomes Eds. O'Shanick G, Varney N. Springer (in press)

14. **Freeman MD**. The epidemiologist as forensic scientist pp 178-9. In: Principles and Practice of Epidemiology: An Engaged Approach, Rossignol AM. McGraw-Hill, NY, NY 2007

15. **Freeman MD**, Nordoff LS. Crash Injury Thresholds, (Ch. 14) in Nordhoff LS. Motor vehicle collision injuries: Biomechanics, diagnosis & management. Jones and Bartlett Publishing, Boston MA 2005.

16. Murphy D, **Freeman MD**. Management of neck pain and related disorders. In Principles of Chiropractic Text. Haldeman S, Editor in Chief. Third Edition, 2004: 969-998.

17. **Freeman MD**, Croft AC. The Controversy over Late Whiplash: Are Chronic Symptoms after Whiplash Real? in: Whiplash Injuries Edited by M. Szpalski and R. Gunzburg. Lippencott-Raven. September 1997

Scientific and Peer-reviewed Conference Proceedings and Abstracts

1. Freeman MD, Williams K, Eriksson A. Ballistic analysis of an attempted murder using a porcine model. *Proceedings of 70th Annual Meeting of the American Academy of Forensic Sciences* 2018 (in review).

2. Freeman MD, Freeman EM. A probabilistic analysis of the cause of a traffic death following 2 crashes using national crash data. *Proceedings of 70th Annual Meeting of the American Academy of Forensic Sciences* 2018 (in review).

3. **Freeman MD,** Lukasevic T, Williams K, Eriksson A. Characteristics of traffic crash related blunt traumatic aortic injury. *Proceedings of 68th Annual Meeting of the American Academy of Forensic Sciences* 2016 Feb 22-26: Las Vegas, NV H61:773-5.

4. **Freeman MD**. Concussion risk associated with head impact; an analysis of pooled data from helmeted sports. *Proceedings of the 12th Annual Conference of the North American Brain Injury Society* J Head Trauma Rehab 2015;30(3):E72-3.

5. Franklin F, **Freeman MD.** An analysis of the causal relationship between maternal/ prenatal cocaine use and stillbirth: results of a national hospital database study. *Proceedings of 67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL H154:975-6.

6. **Freeman MD**. Biomechanical, Mechanical, and Epidemiologic Characteristics of Low Speed Rear Impact Collisions. *Proceedings of 67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL. D11:517-8.

7. **Freeman MD**, Cahn P. An unusual case of commotio cordis resulting from a side impact airbag deployment. *Proceedings of the AAFS Scientific Session of the World Forensic Festival*, October 12-18, 2014, Seoul, Korea

8. Williams K, **Freeman MD,** et al. The investigation of a cluster of fentanyl overdose deaths: how the use of epidemiologic surveillance and outbreak methods resulted in the rapid identification of the source of a public health crisis. *Acad Forensic Pathol* 2014;4(Suppl):S-4.

9. **Freeman MD**. Forensic Applications of Epidemiology in Civil and Criminal Litigation. *9th International Conference on Forensic Inference and Statistics* August 19-22, 2014, Leiden, NL

10. Rubanzana W, **Freeman MD**, Hedt-Gauthier B. Exposure to genocide and risk of suicide in Rwanda: a population-based case-control study. *20th IEA World Congress of Epidemiology.*

August 2014, Anchorage, AK.

11. **Freeman MD**, Uhrenholt L. Investigation of a disputed mechanism of diffuse axonal injury following a low speed frontal crash. *Proceedings of 66th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle (WA). 1984:367-8.

12. **Freeman MD**, Cahn P. Cause of death following opiate overdose; a forensic epidemiologic investigation of comparative risk. *Proceedings of 66th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle (WA). 1984:396.

13. Rubanzana W, **Freeman MD**, Hedt-Gauthier B. Risk factors for homicide victimization in Rwanda, post Genocide: A forensic epidemiological investigation. *Proceedings of 66th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle (WA). 1984:564-5.

14. Colville-Ebeling B, **Freeman MD.** An Evaluation of the Discriminatory Power of Clinical Diagnostic Findings of Abusive Head Trauma in U.S. Hospitals. *Proceedings of 66th Annual Meeting of the American Academy of Forensic Sciences* 2014 Feb 17-22: Seattle (WA). 1984:346-7.

15. Hatten B, **Freeman MD,** Horowitz Z. Coral Snake Envenomations 2001-2011: Antivenin Use and Outcomes. Proceedings of the Society for Academic Emergency Medicine. 2013, Annual Meeting in Atlanta, Georgia, May 14-18, 2013.

16. Rubanzana W, **Freeman MD.** Exposure to effects of genocide as a risk factor for intentional death in Rwanda: a forensic epidemiological investigation. *Scand J Forens Med* 2012;18(1):117.

17. **Freeman MD**, Dobbertin K, Kohles SS, Uhrenholt L, Eriksson A. Serious head and neck injury as a predictor of occupant position in fatal rollover crashes. *Scand J Forens Med* 2012;18(1):34-5.

18. **Freeman MD**, Uhrenholt L. Self-defense or attempted murder? A combined ballistic and traffic crash reconstruction of a Texas shooting. *Scand J Forens Med* (2012;18(1):51.

19. **Freeman MD.** Applied forensic epidemiology: the evaluation of individual causation in wrongful death cases using relative risk. *Scand J Forens Med* (2012;18(1):25.

20. Uhrenholt L, Webb A, Pedersen M, Christensen HW, **Freeman MD.** Does whiplash trauma result in somatic injury. *Scand J Forens Med* (2012;18(1):121

21. **Freeman M**, Uhrenholt L. Rollover collisions; the effect of restraint use on skull vault fractures. 2011. Poster session presented at Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

22. Uhrenholt L, **Freeman M**, Jurik AG, Jensen LL, Gregersen MEG, Boel LWT et al. Evidence of somatic injury in rear-impact collisions - esophagus injuries. 2011. Poster session presented at Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

23. Uhrenholt L, **Freeman M.** How microscopy can explain traffic crash-related cervical spine injury. 2011. Poster session presented at Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse [The Danish Traffic Medicine Society of

the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

24. Nystrom NA, **Freeman MD.** "Trigger Point" Surgery for Soft Tissue Pain in Chronic Whiplash Syndrome. *J Rehab Medicine* 2011;Suppl 50:27.

25. **Freeman MD**, Kohles SS. Scientific and Legal Criteria for Evaluating Injury Causation Following Whiplash Trauma. *J Rehab Medicine* 2011;Suppl 50:20.

26. Rosa S, **Freeman MD**, Harshfield D. Restoration of Normal Cerebrospinal Fluid Flow in 2 Cases of Confirmed Cerebellar Tonsillar Ectopia with Long-Term Headaches, Following Use of The Atlas Orthogonal Instrumented Manipulation Technique. *J Rehab Medicine* 2011;Suppl 50:14.

27. **Freeman MD**, Centeno CJ. "Whiplash-Associated Disorders [WAD]" – the persisting lexicon of a failed venture. *J Rehab Medicine* 2011;Suppl 50:6-7

28. Kohles SS, **Freeman MD**. Mathematical Models Characterizing the Probability of Trigger Event, Ambient-Risk, and Coincidental Influences on Inductive and Abductive Conclusions of Specific Causation. *Annals of Epidemiology* 2010;20(9):713-4.

29. Centeno MD, **Freeman MD**, Schultz J, Cheever M, Faulkner S, Hanson R, Kohles S. Clinical Percutaneous Implantation of Autologous, Culture-Expanded MSCs into Peripheral Joints. *Orthopedic Research Society*, 2011 Annual Meeting in Long Beach, California, January 13-16.

30. Nystrom A, **Freeman MD**. Central sensitization is a reversible response to focal soft-tissue neck pain in chronic whiplash. *2010 American Academy of Orthopedic Surgeons Annual Meeting* March 9-13, 2010, New Orleans, LA.

31. **Freeman MD**. The Error Odds method of objectively assessing bioengineering based claims of causation; a Bayesian approach to test validity quantification (Special joint session of Jurisprudence and Engineering Sciences) *Proceedings of 62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 2010, Seattle, Washington.

32. Nystrom A, **Freeman MD**. Central sensitization is an immediately reversible phenomenon in chronic pain after whiplash. A double blind, placebo controlled study. *XXVIII European Society for Regional Anaesthesia Annual Congress* Salzburg, Austria, September 9-12, 2009

33. **Freeman MD**, Rosa S, Harshfield D, Smith F, Bennett RM, Centeno CJ, Kornel E, Nystrom A, Heffez D, Kohles SS. A case-control study of cerebellar tonsillar ectopia and cervical spine trauma. *European Congress of Radiology,* March 4-8, 2010, Vienna, Austria.

34. Uhrenholt L, **Freeman MD**. The Role of Microscopic Post-Mortem Study in Explaining Traffic-Crash Related Neck Injury; A Review. *Proceedings of 62nd Annual Meeting of the American Academy of Forensic Sciences*. Feb 2010, Seattle, Washington.

35. **Freeman MD**, Uhrenholt L, Newgard C. The effect of restraint use on skull vault fractures in rollover crashes. *Proceedings of 62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 2010, Seattle, Washington.

36. **Freeman MD**, Uhrenholt L, Newgard C. Head injuries in lower speed collinear collisions; an analysis of the National Automotive Sampling System database. *Proceedings of 62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 2010, Seattle, Washington.

37. **Freeman MD**, Rosa S, Harshfield D, Smith F, Bennett RM, Centeno CJ, Kornel E, Nystrom A, Heffez D, Kohles SS. A case-control study of cerebellar tonsillar ectopia and cervical spine trauma. *XXI Congress of the International Academy of Legal Medicine* May 2009 Lisbon, Portugal

38. **Freeman MD**. The Error Odds assessment of accuracy for tests in forensic medicine; a simple application of Bayes' Law. *XXI Congress of the International Academy of Legal Medicine* May 2009 Lisbon, Portugal

39. Uhrenholt L, Schumacher B, **Freeman MD**. A cross-sectional study of road traffic fatalities and vehicular homicide investigation practices in Denmark for 2000-2004. *Proceedings of 61st Annual Meeting of the American Academy of Forensic Sciences*. Feb 2009, Denver, Colorado.

40. **Freeman MD**, Centeno CJ. Etiologic and demographic characteristics of traffic crash-related disc injuries. *Spine J* doi:10.1016/j.spinee.2008.06.373.

41. **Freeman MD**. Bayesian analysis of predictive characteristics in suicidal versus homicidal hanging deaths: A case study in forensic epidemiology. *Proceedings of 59th Annual Meeting of the American Academy of Forensic Sciences* February 19-24, 2007, San Antonio, Texas 2007;13:304.

42. **Freeman MD**. Probability and pathological findings in suicide versus homicidal hanging deaths; a case study. *Proceedings of 16th Nordic Conference on Forensic Medicine* June 15-17, 2006, Turku, Finland 2006:15-6.

43. **Freeman MD**. Injury Pattern Analysis as a means of driver determination in a vehicular homicide investigation. *Proceedings of 16th Nordic Conference on Forensic Medicine* Turku, Finland June 15-17 2006:38-9.

44. **Freeman MD**. Injury Pattern Analysis in Fatal Traffic Crash Investigation. *Proceedings of 57th Annual Meeting of American Academy of Forensic Sciences* New Orleans, Louisiana. February 24, 2005.

45. **Freeman MD**, Croft AC, Centeno C. Fatal head injury cases in a rural Oregon county. *Proceedings of the 19th World Congress of the International Traffic Medicine Association* Budapest, Hungary, September 14-17, 2003.

46. Croft AC, Haneline MT, **Freeman MD**. Differential Occupant Kinematics and Forces Between Frontal and Rear Automobile Impacts at Low Speed: Evidence for a Differential Injury Risk, *International Research Council on the Biomechanics of Impact (IRCOBI)* Munich, Germany September 18-20, 2002:365-6.

47. Sparr L, **Freeman MD**. The Uses and Abuses of Psychiatric IMEs: An Ethical Dilemma? *American Psychiatric Association Annual Meeting* San Francisco, California May 2003.

48. Croft AC, Haneline MT, **Freeman MD**. Low speed frontal crashes and low speed rear crashes: is there a differential risk for injury? *Proceedings of the 46th Association for the Advancement of Automotive Medicine (AAAM) Annual Scientific Conference* Tempe, Arizona, September 29-October 2, 2002:79-91.

49. Croft AC, Lord S, **Freeman MD**. Whiplash Injury: Mechanisms of Injury, Pathophysiology, and Treatment *10th World Congress on Pain, International Association for the Study of Pain* San Diego, August 17-22, 2002:482.

50. **Freeman MD**, Centeno C, Croft AC, Nicodemus CN: Significant spinal injury resulting from low-level accelerations: a comparison with whiplash. *International Congress on Whiplash-Associated Disorders* Berne, Switzerland, March 9-10, 2001.

51. Croft AC, Haneline MT, **Freeman MD**. Differential occupant kinematics and head linear acceleration between frontal and rear automobile impacts at low speed: evidence for a differential injury risk. I*nternational Congress on Whiplash-Associated Disorders* Berne, Switzerland, March 9-10, 2001.

52. Croft AC, Haneline MT, **Freeman MD**. Automobile crash reconstruction in low speed rear impact crashes utilizing a momentum, energy, and restitution (MER) method. *International Congress on Whiplash-Associated Disorders* Berne, Switzerland, March 9-10, 2001.

53. Centeno C, **Freeman MD**, Croft AC. A comparison of the functional profile of an international cohort of whiplash injured patients and non-patients: an internet study. *International Congress on Whiplash-Associated Disorders* Berne, Switzerland, March 9-10, 2001.

54. **Freeman MD**, Sapir D, Boutselis A, Gorup J, Tuckman G, Croft AC, Centeno C, Phillips A. Whiplash injury and occult vertebral fracture: a case series of bone SPECT imaging of patients with persisting spine pain following a motor vehicle crash. *Cervical Spine Research Society 29th Annual Meeting* Monterey, CA, Nov 29-Dec 1, 2001.

55. Johansson BH, **Freeman MD**. The prevalence of symptomatic cervical disc herniation in the Swedish population with asymptomatic degenerative disc disease (a cross-sectional study). *International Congress on Whiplash Associated Disorders* March, 2001. Berne, Switzerland.

56. **Freeman MD**, Centeno C, Croft AC, Nicodemus C. Significant spinal injuries resulting from low-level accelerations: a case series of roller coaster injuries. *Proceedings of Cervical Spine Research Society 28th Annual Meeting,* November 30-December 2, 2000:110-1.

57. Croft AC, **Freeman MD**. An evaluation of the neck injury criterion; recommendations for future consideration. *Association for the Advancement of Automotive Medicine*, San Antonio, TX October, 2000.

58. **Freeman MD**, Croft AC, Rossignol AM. The prevalence of whiplash-associated chronic cervical pain among a random sample of patients with chronic spine pain. *Proceedings of 27th Annual Cervical Spine Research Society Annual Meeting*. Seattle, WA December 13-15, 1999.

59. **Freeman MD**, Croft AC, Rossignol AM. Late whiplash risk factor analysis of a random sample of patients with chronic spine pain. *World Whiplash Associated Disorders Congress*, February 8-10, 1999, Vancouver, British Columbia

Scientific Commentary/Editorials/Letters

1. Uhrenholt L, Webb A, **Freeman MD**. Letter to the Editor regarding "Do X-ray-occult fractures play a role in chronic pain following a whiplash injury?" *Eur Spine J* DOI 10.1007/s00586-014-3362-3.

2. **Freeman MD**. Clinical Practice Guidelines versus Systematic Reviews; which serves as the best basis for evidence based spine medicine? Invited commentary. *Spine J* 2010 Jun;10(6):512-3.

3. **Freeman MD**, Centeno CJ, Katz E. MR imaging of whiplash injury in the upper cervical spine; controversy or confounding? *Spine J* 2009 Sep;9(9):789-90. Epub 2009 Jun 17

4. Centeno CJ, **Freeman M**. Re: Are smooth pursuit eye movements altered in chronic whiplash-associated disorders? A cross-sectional study. *Clin Rehabil* 2008 Apr;22(4):377-8.

5. Centeno CJ, **Freeman MD**. Editorial Submission on Kongsted, A., et al., Are smooth pursuit eye movements altered in chronic whiplash associated disorders? A cross-sectional study. *Clin Rehabil* 2007;21(11):1038-49.

6. **Freeman MD**. Crash Test Dummy? *New Scientist* June 23, 2007:22-3.

7. **Freeman MD**, Centeno CJ, Merskey H, Teasell R, Rossignol AM. Greater injury leads to more treatment for whiplash: no surprises here. *Arch Int Med* 2006;166(11):1238-9.

8. Centeno C, **Freeman MD**. Alberta rodeo riders do not develop late whiplash. *J Rheumatol* 2007 Feb;34(2):451-2.

9. **Freeman MD**, Centeno C. Alar, Transverse and Apical Ligament Strain due to Head-Turned Rear Impact. *Spine* 2006;31(17):2030.

10. **Freeman MD**. Cervical disc herniation following motor vehicle crash trauma. Invited commentary. *Spine J* 2005 Nov-Dec;5(6):644.

11. **Freeman MD**, Centeno C. Whiplash and Peer Review *JWRD* 2003;2(2):1-3.

12. **Freeman MD**, Centeno C. Whiplash and Secondary Gain *JWRD* 2003;2(1):1-4.

13. **Freeman MD**, Centeno C. "Placebo" Collisions and Whiplash *JWRD* 2002;1(2):1-8.

14. **Freeman MD**. Biomechanics of minor automobile accidents. *J South Orthop Assoc* 2001 Summer;10(2):95-6.

15. **Freeman MD**. Are demolition derby drivers a valid proxy for the population at risk for whiplash injury? *Arch Neurol* 2001 Apr;58(4):680-1.

16. **Freeman MD**, Rossignol AM. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims. *NEJM* 2000 Oct 12;343 (15):1118-9.

17. **Freeman MD**. Letter to the editor. *Cranio* 1999;17(3):160-1.

18. Croft AC, **Freeman MD**. Commentary on "Pain after whiplash: a prospective controlled inception cohort study." *The Back Letter* 1999;14(4):43-5.

19. **Freeman MD**, Croft AC. Late Whiplash Syndrome, 3rd reply. *Lancet* 1996 Jul 13;348(9020):125.

SCIENTIFIC PRESENTATIONS

1. Freeman MD. Evidence-based practice in Forensic Medicine; Principles of Forensic Epidemiology. Faculty of Medicine, University of Nijmegan, October 9, 2017, Nijmegen, Netherlands.

2. Freeman MD. Incidence and risk factors for neonatal falls US Hospitals, 2003-2012. *Health Science Research,* Doernbecher Childrens' Hospital, Oregon Health & Science University, March 13, 2017, Portland, Oregon.

3. Freeman MD. Incidence and risk factors for neonatal falls US Hospitals, 2003-2012. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, January 31, 2017, Portland, Oregon.

4. Freeman MD. Evidence-based practice in Forensic Medicine. Invited presentation to the Dutch National Forensic Institute (NFI). December 6, 2016 Maastricht University, Maastricht, Netherlands.

5. Freeman MD. Forensic Epidemiology: Principals & Practice Part 2: Investigation of specific causation. Gran Sesión de Epidemiología Forense. November 18, 2016 Universidad Libre, Cali, Colombia.

6. Freeman MD. Forensic Epidemiology: Principals & Practice Part 1: Investigation of specific causation. Gran Sesión de Epidemiología Forense. November 18, 2016 Universidad Libre, Cali, Colombia.

7. Freeman MD. Fatal crash investigation. World Reconstruction Exposition (WREX 2016). May 2-6, 2016. Orlando, Florida.

8. Freeman MD. Trends in police use-of-force related hospitalizations; an analysis of Nationwide Inpatient Sample data for 1998-2012. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, November 10, 2015, Portland, Oregon.

9. Freeman MD. Concussion risk associated with head impact; an analysis of pooled data from helmeted sports. *12th Annual Conference of the North American Brain Injury Society,* April 29-May 1, 2015 San Antonio, Texas

10. Freeman MD. The role of risk in assessing cause in forensic investigation of injury and death. *American Medical Response biennial EMS training.* April 17, 2015, Mt. Hood, Oregon.

11. Freeman MD. Development of a pediatric fatal head trauma registry. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, April 7, 2015, Portland, Oregon.

12. Freeman MD. Fatal crash investigation: methods and case presentations. Washington County CART Team training lecture. Tualatin Police Department, Tualatin, Oregon. March 4, 2015.

13. Freeman MD. An analysis of the causal relationship between maternal/ prenatal cocaine use and stillbirth: results of a national hospital database study. *67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL

14. Freeman MD. Biomechanical, Mechanical, and Epidemiologic Characteristics of Low Speed Rear Impact Collisions. *67th Annual Meeting of the American Academy of Forensic Sciences* 2015 Feb 16-21: Orlando, FL.

15. Freeman MD. Sexual abuse in the Boy Scouts: a preliminary analysis of Boy Scout ineligible volunteer files from 1945 to 2004. *Research in Progress,* Department of Sociology, Portland State University. December 18, 2014.

16. Freeman MD. Understanding chronic pain after whiplash trauma. *Lund University Hospital, Department of Rehabilitation Medicine.* December 11, 2014, Lund, Sweden.

17. Freeman MD. Forensic Applications of Epidemiology in Criminal and Civil Settings. *Richard Doll Building, Nuffield College, Oxford University.* December 10, Oxford, UK.

18. Freeman MD. The Efficacy of tPA in Preventing Long Term Poor Outcome After Ischemic Stroke: A Reanalysis of NINDS Data. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, November 25, 2014, Portland, Oregon.

19. Freeman MD. Forensic Epidemiology and Bioterrorism. Full day course for public health and law enforcement. A joint training for public health, law enforcement, and emergency services. Sponsored by Charles County Department of Public Health and funded through a grant from the Centers for Disease Control and Prevention, Public Health Preparedness Cooperative Agreement. College of Southern Maryland. June 10, 2014. Waldorf, Maryland.

20. Freeman MD. Maternal cocaine exposure and still birth risk. *Research in Progress,* Department of Internal Medicine, Oregon Health & Science University School of Medicine, May 20, 2014, Portland, Oregon.

21. Freeman MD. Forensic Applications of Epidemiology in Civil and Criminal Litigation. *9th International Conference on Forensic Inference and Statistics* August 19-22, 2014

22. Freeman MD. Investigation of a disputed mechanism of diffuse axonal injury following a low speed frontal crash. *65th Annual Meeting of the American Academy of Forensic Sciences,* Feb 21, 2014, Seattle, Washington.

23. Freeman MD. Public defense of dissertation for Doctor of Medicine degree, "The role of forensic epidemiology in evidence based forensic medical practice." *Section of Forensic Medicine, Department of Community Medicine and Rehabilitation, Faculty of Medicine,*

Umeå University. November 6, 2013, Umeå, Sweden.

24. Freeman MD. Case studies in applied forensic epidemiology. Invited lecture, *University of Maastricht, Department of Complex Genetics and Epidemiology,* Maastricht, The Netherlands. October 31, 2013.

25. Freeman MD. The relationship between Chiari malformation, trauma, and chronic pain. *Karolinska Institute,* September 27, 2012, Stockholm, Sweden.

26. Freeman MD. Serious head and neck injury as a predictor of occupant position in fatal rollover crashes. *18th Nordic Conference on Forensic Medicine*, June 13-16, 2012 Aarhus Denmark.

27. Freeman M. Self-defense or attempted murder? A combined ballistic and traffic crash reconstruction of a Texas shooting. *18th Nordic Conference on Forensic Medicine*, June 13-16, 2012 Aarhus Denmark.

28. Freeman MD. Applied forensic epidemiology: the evaluation of individual causation in wrongful death cases using relative risk. *18th Nordic Conference on Forensic Medicine,* June 13-16, 2012 Aarhus Denmark.

29. Freeman MD. Forensic Epidemiologic Investigation of Traffic Crash-Related Homicide. *Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse* [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

30. Freeman MD. Traffic Crash Injuries 1960 to the present; how far we've come. Keynote address, *Årsmøde i Dansk Selskab for Retsmedicin og Dansk Selskab for Ulykkes- og Skadeforebyggelse* [The Danish Traffic Medicine Society of the Danish Society for Forensic Medicine] November 3-5, 2011] Grenå, Denmark.

31. Freeman MD. Is there a place for forensic biomechanics in evaluation of Probability of Causation? *8th International Conference on Forensic Inference and Statistics (ICFIS),* July 19-21, 2011; University of Washington, Seattle, Washington.

32. Freeman MD. Case studies in forensic epidemiology. *8th International Conference on Forensic Inference and Statistics (ICFIS)*, July 19-21, 2011; University of Washington, Seattle, Washington.

33. Freeman MD. The Error Odds method of objectively assessing bioengineering based claims of causation; a Bayesian approach to test validity quantification. Invited lecture; joint session of Jurisprudence and Engineering Sciences. *62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 25, 2010, Seattle, Washington.

34. Freeman MD, Uhrenholt L, Newgard C. The effect of restraint use on skull vault fractures in rollover crashes. Engineering Sciences section, *62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 26, 2010 Seattle, Washington.

35. Freeman MD, Uhrenholt L, Newgard C. Head injuries in lower speed collinear collisions; an analysis of the National Automotive Sampling System database. Engineering Sciences section, *62nd Annual Meeting of the American Academy of Forensic Sciences* Feb 26, 2010 Seattle, Washington.

36. Freeman MD. The Error Odds assessment of accuracy for tests in forensic medicine; a simple application of Bayes' Law. Invited presentation; *XXI Congress of the International Academy of Legal Medicine* May 2009, Lisbon, Portugal

37. Freeman MD. Forensic Epidemiology and Traumatic Brain Injury. Invited presentation; *VII World Congress on Brain Injury, International Brain Injury Association* April 2008 Lisbon, Portugal.

38. Freeman MD, Hand M. Bayesian analysis of predictive characteristics in suicidal versus homicidal hanging deaths: A case study in forensic epidemiology. *59th Annual Meeting of the American Academy of Forensic Sciences* February 19-24, 2007, San Antonio, Texas.

39. Freeman MD. Probability and pathologic findings in suicidal versus homicidal hanging deaths; a case study *16th Nordic Conference on Forensic Medicine* June 15, 2006, Turku, Finland.

40. Freeman MD. Injury Pattern Analysis as a means of driver determination in a vehicular homicide investigation *16th Nordic Conference on Forensic Medicine* June 16, 2006, Turku, Finland.

41. Freeman MD. Probability and pathologic findings in suicidal versus homicidal hangings; a case study. Grand Rounds *Institute of Forensic Medicine, Aarhus University, Aarhus, Denmark.* October 27, 2005.

42. Freeman MD. Road Traffic Crashes- mechanisms, injuries and analysis. Invited lecture (Keynote address) *Danish Society for Automotive Medicine* Aarhus, Denmark. October 27, 2005.

43. Freeman MD. The Defense Medical Evaluation: Issues, Ethics and Pitfalls. *2nd Annual International Whiplash Trauma Congress* Breckenridge, Colorado. February 26, 2005.

44. Freeman MD. Injury Pattern Analysis in Fatal Traffic Crash Investigation *American Academy of Forensic Sciences' 57th Annual Meeting* New Orleans, Louisiana. February 24, 2005.

45. Freeman MD. Independent Medical Evaluations and secondary gain. Grand Rounds, *Department of Psychiatry, Oregon Health & Science University School of Medicine* November 2, 2004.

46. Freeman MD. The epidemiology of crash-related trauma. Invited lecture. Grand Rounds *Peace Health Hospital* Longview, Washington. March 30, 2004.

47. Freeman MD. Injury pattern analysis: the practical application to the investigation of crash related death. Grand Rounds Department of Pathology, *Oregon Health Sciences University* Portland, Oregon. January 21, 2004.

48. Freeman MD. Literature critique, Whiplash Updates. Invited lecture. *British Columbia Chiropractic Association* Vancouver, British Columbia, Canada. October 23, 2003.

49. Freeman MD. Catastrophic crash cases and probability. Invited lecture. *Paris American Legal Institute* Florence, Italy. September 22, 2003.

50. Freeman MD. Injury pattern analysis as a means of driver identification in a vehicular homicide; a case study. *International Traffic Medicine Association Annual Meeting.* Budapest, Hungary. September 17, 2003.

51. Freeman MD. Fatal head injury crashes in a rural Oregon county, 1990-1999. *International Traffic Medicine Association Annual Meeting.* Budapest, Hungary. September 16, 2003.

52. Freeman MD. Crash reconstruction and forensic science. Invited lecture. *CRASH 2003* Spine Research Institute of San Diego. San Diego, California. August 22, 2003.

53. Freeman MD, Sparr L. The uses and abuses of psychiatric IMEs: an ethical dilemma. *American Psychiatric Association Annual Meeting.* San Francisco, California. May 21, 2003.

54. Freeman MD. Crash-related trauma. Invited lecture. THRI Neuroscience meeting. *Texas Back Institute* St. Mary's Hospital. Plano, Texas. February 28, 2003.

55. Freeman MD. Whiplash injury and occult spinal fracture. *International Association for the Study of Pain* 10th World Congress on pain. San Diego, California. August 20, 2002.

56. Freeman MD. Crash Reconstruction and forensic science. *CRASH 2002* Spine Research Institute of San Diego. San Diego, California. August 8, 2002.

57. Freeman MD. Epidemiologic and medical aspects of whiplash injury. *Swedish Orthopedic Society* Stockholm, Sweden. May 17, 2002.

58. Freeman MD. Epidemiologic considerations of whiplash injuries. Invited lecture. *European Chiropractic Union Annual Congress* Oslo, Norway. May 9, 2002.

59. Freeman MD. The role of cervical manipulation in neck pain. Invited lecture. *Cervical Spine Research Society 29th Annual Meeting* Instructional Course, Monterey, CA, Nov 29-Dec 1, 2001

60. Freeman MD. Whiplash injury and occult vertebral fracture: a case series of bone SPECT imaging of patients with persisting spine pain following a motor vehicle crash. *Cervical Spine Research Society 29th Annual Meeting* Monterey, CA, Nov 29-Dec 1, 2001

61. Freeman MD. Interpreting the medical literature with a focus on bias and confounding/Minimal Damage Crash Reconstruction. Invited lecture. *CRASH 2001* Spine Research Institute of San Diego. San Diego, CA. August 2001.

62. Freeman MD. Injury Pattern Analysis and Forensic Trauma Epidemiology in vehicular homicide investigation. *Washington State Patrol* Lacy, WA, June 20, 2001

63. Freeman MD. Case studies in multidisciplinary spine care. *Chiropractic Association of Oregon* Portland OR, April 28, 2001

64. Freeman MD. Injury Pattern Analysis and Forensic Trauma Epidemiology in vehicular homicide investigation. *Washington State Patrol* Vancouver, WA, February 13, 2001

65. Freeman MD. The role of cervical manipulation in neck pain. Invited lecture. *Cervical Spine Research Society 28th Annual Meeting* Instructional Course. Charleston, South Carolina, December 1, 2000

66. Freeman MD. Significant spinal injuries resulting from low-level accelerations: a case series of roller coaster injuries. *Cervical Spine Research Society 28th Annual Meeting* Charleston, South Carolina, December 1, 2000

67. Freeman MD. Injury Pattern Analysis and Forensic Trauma Epidemiology in vehicular homicide investigation. *Medical Examiner Division, Oregon State Police.* Salem, OR. November 28, 2000

68. Freeman MD. Minimal damage motor vehicle crash reconstruction. Invited lecture. Spine Research Institute of San Diego. *CRASH 2000* Spine Research Institute of San Diego. San Diego CA. August 11-13, 2000

69. Freeman MD. Analysis of the whiplash literature with emphasis on research out of Quebec and Saskatchewan. *Saskatchewan Medical Group and Coalition Against No-Fault.* Saskatoon, Saskatchewan. September 2000.

70. Freeman MD. Forensic applications of crash reconstruction. Invited lecture. *CRASH 2000* Spine Research Institute of San Diego.. San Diego, CA. August 11, 2000.

71. Freeman MD. Injury Pattern Analysis and Forensic Trauma Epidemiology; practical application in the forensic setting. Washington County CART Team training lecture, on behalf of *Medical Examiner Division, Oregon State Police.* Lake Oswego, Oregon. July 13, 2000.

72. Freeman MD. The epidemiology of acute and chronic whiplash injury in the U.S. Invited lecture. *HWS-Distorsion (Schleudetrauma) & Leichte Traumatische, Hirnverletzung. Invaliditat und Berufliche Reintegration.* Basel, Switzerland. June 29-30, 2000.

73. Freeman MD. Whiplash injury risk factors. Invited lecture. *Whiplash 2000.* Bath, England. May 18, 2000.

74. Freeman MD. How many whiplash injuries could there be? Invited lecture. *Whiplash 2000* Bath, England. May 17, 2000.

75. Freeman MD. Whiplash injury and occupant kinematics; the results of human volunteer crash testing. Invited lecture. *Society for Road Traffic Injuries (LFT).* Oslo, Norway. April 3, 2000.

76. Freeman MD. Epidemiology of Whiplash Injuries. Invited lecture. *Swedish Orthopedic Society* Stockholm, Sweden. March 31, 2000.

77. Freeman MD. Methodologic pitfalls in epidemiological and clinical research, with examples from whiplash research. Invited lecture. *Arvetsinstitut (Institute for Musculoskeletal Medicine Research) Umeå University,* Umeå, Sweden. March 30, 2000.

78. Freeman MD. The prevalence of whiplash-associated chronic cervical pain among a random sample of patients with chronic spine pain. *Cervical Spine Research Society 27th Annual Meeting* Seattle, WA December 13-15, 1999.

79. Freeman MD. High speed videography of occupant movement during human volunteer crash testing; searching for an injury threshold. *North American Whiplash Trauma Congress* November 12, 1999.

80. Freeman MD. Scientific Chair Address. *North American Whiplash Trauma Congress* November 12, 1999.

81. The science of whiplash injuries: common mistakes in the reconstruction of low speed crashes. Invited lecture. *Forensic Accident Reconstructionists of Oregon* Eugene, Oregon, April 1, 1999.

82. Freeman MD. Late whiplash risk factor analysis of a random sample of patients with chronic spine pain. *Whiplash Associated Disorders World Congress* Vancouver, B.C. February 9, 1999.

83. Freeman MD. The epidemiology of whiplash injuries; critiquing the literature. Grand rounds, *Department of Public Health and Preventive Medicine, Oregon Health Sciences University* Portland, Oregon. December 17, 1998.

84. Freeman MD. The scientific appraisal of motor vehicle crash-related injuries. Invited lecture. *Managing the Cost of Auto Injuries.* Orlando, FL. December 8, 1998.

85. Freeman MD. Risk factors for chronic pain following acute whiplash injury. Invited lecture. *Managing the Cost of Auto Injuries* Orlando, FL. December 7, 1998.

86. Freeman MD. The epidemiology of whiplash injuries. Current Issues in Public Health, *Department of Public Health and Preventive Medicine, Oregon Health Sciences University* Portland, Oregon. October 7, 1998

87. Freeman MD. The epidemiology of whiplash - is there a reliable threshold for whiplash injury? Invited lecture. *HWS-Distortion (Schleudetrauma) & Leichte Traumatische Medico-Legal Congress.* Basel, Switzerland, June 26, 1998.

88. Freeman MD. The Epidemiology of Late Whiplash. Invited lecture. *HWS-Distortion (Schleudetrauma) & Leichte Traumatische Medico-Legal Congress.* Basel, Switzerland, June 25, 1998.

89. Freeman MD. Methodologic error in the whiplash literature. Invited lecture. *Whiplash '96* Brussels, Belgium, November 15-16, 1996

90. Freeman MD. Conservative therapy for spinal disorders *St. Francis Hospital*, San Francisco, CA. September 1994

91. Freeman MD. The history of chiropractic. Invited lecture. *White Plains Hospital*, White Plains, NY. December 1993