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IN THE SUPREME COURT OF OHIO

STATE OF OHIO, Plaintiff-Appellee, v. BRANDON MOORE, Defendant-Appellant.	: : : : : : : : : : :	CASE NO. 2014-0120 ON DISCRETIONARY APPEAL FROM THE MAHONING COUNTY COURT OF APPEALS, SEVENTH APPELLATE DISTRICT, CASE NO. 08MA20

BRIEF OF *AMICI CURIAE* DR. BEATRIZ LUNA, DR. CHARLES ALEXANDER NELSON III, DR. SILVIA BUNGE, DR. ADRIANA GALVÁN, AND DR. LINDA PATIA SPEAR IN SUPPORT OF NEITHER PARTY

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STATEMENT OF INTEREST

Amici are leading neuroscientists whose research focuses on cognitive development in youth and adolescents.

Dr. Beatriz Luna is the Staunton Professor of Pediatrics and Psychiatry, a Professor of Psychology, and the Director of the Laboratory of Neurocognitive Development at the University of Pittsburgh. Her work, which was cited extensively in *amicus* briefs submitted to the U.S. Supreme Court by the American Medical Association and the American Academy of Child and Adolescent Psychiatry in *Graham v. Florida*, 560 U.S. 48, 130 S.Ct. 2011, 176 L.Ed.2d 825 (2010) and *Miller v. Alabama*, __ U.S. __, 132 S. Ct. 2455, 183 L.Ed.2d 407 (2012), focuses on brain mechanisms that support the transition to adult-level cognition control of behavior.

Dr. Charles Alexander Nelson III is Professor of Pediatrics and Neuroscience and of Psychology in Psychiatry at Harvard Medical School; he is also Professor of Education at Harvard University. Dr. Nelson holds the Richard David Scott Chair in Pediatric Developmental Research at Boston Children's Hospital, where he also serves as Director of Research in Developmental Medicine. His work addresss a variety of problems in developmental cognitive neuroscience, including the role of experience in influencing the course of brain development.

Dr. Silvia Bunge is a Professor in the Department of Psychology and the Helen Wills Neuroscience Institute at the University of California at Berkeley, and the Director of the Building Blocks of Cognition Laboratory. Among other projects, the laboratory is conducting a large study intended to track neural changes that underlie improvements in reasoning ability from childhood through adolescence. Professor Bunge has contributed to several amicus briefs regarding life without parole sentencing for juveniles, and has served as an expert witness on adolescent brain development in the California Senate with regard to CA Senate Bill 9, 2011.

Her work on the developing human brain was cited in *Graham v. Florida*, 560 U.S. 48, 130 S.Ct. 2011, 176 L.Ed.2d 825 (2010).

Dr. Adriana Galván is an Associate Professor of Developmental Psychology at the University of California, Los Angeles, an Executive Committee Member of the Staglin Center for Cognitive Neuroscience, and the Director of the Developmental Neuroscience Laboratory at UCLA. Her work centers on adolescent brain development, and in particular on how changes in brain maturation during adolescence relate to adolescent behavior and decision-making. Her work was cited in *amicus* briefs submitted to the U.S. Supreme Court in *Graham v. Florida*, 560 U.S. 48, 130 S.Ct. 2011, 176 L.Ed.2d 825 (2010) and *Miller v Alabama*, __ U.S. __, 132 S. Ct. 2455, 183 L.Ed.2d 407 (2012). Dr. Galván appeared as an *amicus* in the latter case.

Dr. Linda Patia Spear is a SUNY-Distinguished Professor of Behavioral Neuroscience in the Department of Psychology at Binghamton University and serves as Director of the Developmental Exposure Alcohol Research Center funded by the National Institute of Alcohol and Alcohol Abuse. Her work assesses neurobehavioral development during adolescence, neural and biological underpinnings of adolescent behavior, and lasting consequences of drugs and stressors experienced at that time on later neurobehavioral function.

Amici are committed to the advancement of science rather than legal advocacy and do not take a position on whether the sentence in this case violates the Eighth Amendment to the United States Constitution. *Amici* are aware, however, that in recent years, courts considering legal constraints on juvenile sentencing frequently have looked to neuroscience to inform their consideration of juvenile offenders' moral culpability and capacity for reform. This attention corresponds to, and likely reflects, important and relatively recent advances in non-invasive

techniques that allow scientists to more accurately assess brain structure and function in people of all ages, including during adolescence.

In the considered view of *amici*, the accumulated scientific evidence demonstrates that "the adolescent's brain is different from both the child's brain and the adult's brain. It is different with respect to both morphology and function, and at the levels of brain structures, regions, circuits, and systems. . . . Indeed, it appears that the brain changes characteristic of adolescence are among the most dramatic and important to occur during the human lifespan." Laurence Steinberg, *A Behavioral Scientist Looks at the Science of Adolescent Brain Development*, 72 Brain and Cognition 160, 160 (2010). *Amici* believe that these scientific findings have important implications for understanding adolescent behavior, but also are cognizant of the risk that these implications can be overstated or misunderstood. *Amici* accordingly provide this brief, which was prepared by the undersigned attorneys, to advise the Court of the current state of neuroscience as it relates to brain development in adolescents.

STATEMENT OF FACTS

To comply with Ohio Supreme Court Rules of Practice 16.02(B) (1)(b) and 16.05, *amici* formally adopt the statement of facts presented in the brief for appellant Brandon Moore.

Amici wish to make clear, however, that the findings described in this brief apply in general terms to adolescents, considered as a class. Given the current state of technology and scientific understanding, *amici* do not believe it is possible for neuroscientists to predict the future developmental trajectory of any single individual or to establish causal links between brain maturation processes and specific instances of past behavior.

ARGUMENT

I. PROPOSITION OF LAW OF *AMICI CURIAE*: COURTS MAY TAKE ACCOUNT OF SCIENTIFIC EVIDENCE ABOUT ADOLESCENT BRAIN DEVELOPMENT WHEN ADDRESSING CONSTITUTIONAL LIMITATIONS ON JUVENILE SENTENCING.

Science alone cannot resolve the normative questions that confront judges who must make legal determinations about juvenile sentencing. But science can aid judges by providing deeper insight into the age-old observation, deeply imbued in the criminal law of Ohio and other American jurisdictions, that adolescents "'often lack the experience, perspective, and judgment expected of adults.'" *See Eddings v. Oklahoma*, 455 U.S. 104, 115-16, 102 S.Ct. 869, 71 L.Ed.2d 1 (1982); *see also In re C.P.*, 131 Ohio St.3d 513, 2012-Ohio-1446, 967 N.E.2d 729, ¶ 39 (Ohio law reflects the "self-evident" assumption that "children are not as culpable for their acts as adults"); *State v. Long*, 138 Ohio St.3d 478, 2014-Ohio-849, 8 N.E.3d 890, ¶ 29 ("juveniles who commit criminal offenses are not as culpable for their acts as adults are and are more amenable to reform.").

In fact, in recent years, the U.S. Supreme Court consistently has looked to neuroscience to illuminate differences between adolescents and adults when addressing legal constraints on how juvenile offenders may be punished. In *Roper v. Simmons*, 543 U.S. 551, 569, 125 S.Ct. 1183, 161 L.Ed.2d 1 (2005), the Court held that the Eighth Amendment bars States from imposing the death penalty for juvenile offenses. That holding rested in part on "scientific and sociological studies" which "tend[ed] to confirm" that 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.'" *Id.* In *Graham v. Florida*, 560 U.S. 48, 68, 130 S.Ct. 2011, 176 L.Ed.2d 825 (2010), the U.S. Supreme Court emphasized that "developments in psychology and brain science continue to show fundamental differences between juvenile and adult minds,"

including that "parts of the brain involved in behavior control continue to mature through late adolescence." Based in part on this scientific evidence, the Court concluded that "juveniles have lessened culpability," are "less deserving of the most severe punishments," *id.*, and, ultimately, that "the Eighth Amendment forbids a State from imposing a life without parole sentence on a juvenile nonhomicide offender." *Id.* at 75. Similarly, when the U.S. Supreme Court later held that "the Eighth Amendment forbids a sentencing scheme that mandates life in prison without possibility of parole for juvenile offenders," *Miller*, 132 S.Ct. at 2469, it observed that the evidence supporting the view that adolescent minds are saliently different from adult brains had grown "even stronger" in the short span since its decisions in *Roper* and *Graham*. *See id.* at 2464 n.5.

Judges have also considered brain development evidence in other related settings. For example, a federal trial judge in Iowa took account of general information about human brain development when sentencing a young adult offender. *United States v. Gall*, 374 F. Supp. 2d 758, 762 & n.2 (S.D. Iowa 2005); *see also Gall v. United States*, 552 U.S. 38, 57-58, 128 S.Ct. 586, 169 L.Ed.2d 445 (2007) (noting that consideration with evident approval). Similarly, a federal district judge sitting in Ohio "conducted a review of the scientific literature" and found "compelling evidence that the judicial system's longstanding principle of treating youth offenders differently than adult offenders is justified in part based on the unformed nature of the adolescent brain." *United States v. Stern*, 590 F. Supp. 2d 945, 952-53 (N.D. Ohio 2008) (O'Malley, J.) (noting that the defendant, a young adult, had begun to engage in relevant criminal behavior at age 14).

The recent attention to neuroscience in the courts reflects in part significant recent advances in the field. A generation ago, neuroscientists knew "[s]urprisingly little" about

"human anatomical brain development between the ages 4 and 18." Jay Giedd et al., *Quantitative Magnetic Resonance Imaging of Human Brain Development*, 6 Cerebral Cortex 551, 551 (1996). In fact, some of what scientists once believed about brain development in youth and adolescents has since been disproved. "Whereas it was once believed that the human brain was largely developed by the onset of puberty, it has now been established that the brain continues to develop throughout adolescence and well into adulthood." Alecia D. Schweinsburg et al., *fMRI Reveals Alteration of Spatial Working Memory Networks Across Adolescence*, 11 J. Int'l Neuropsychological Soc'y 631, 631 (2005). The emergence of magnetic resonance imaging (MRI) techniques contributed significantly to these advances. MRI-based technologies use high-strength magnetic fields to produce high-resolution images of human tissues, and thus allow researchers to examine the brain without using invasive or otherwise risky procedures, such as surgery, radiation, or injections. Alongside other methods, neuroscientists use three basic categories of MRI-based technologies to study the brain.

1. *"Structural" MRI studies*: These studies seek to understand how particular features of the brain change over the course of human development, taking advantage of the capacity of MRI to produce "higher spatial resolution, superior contrast and soft-tissue imaging capability, improved fine gray–white matter distinctions and greater differentiation of white matter changes when compared with other techniques."[1] These studies give scientists an important window into "age-related changes in total brain volume and in the volumes of various cortical and subcortical structures."[2]

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[1] Deborah Yurgelun-Todd, *Emotional and Cognitive Changes During Adolescence*, 17 Current Opinion Neurobiology 251, 252 (2007).

[2] Elizabeth Sowell et al., *Development of Cortical and Subcortical Brain Structures in Children and Adolescence: A Structural MRI Study*, 44 Developmental Med. & Child Neurology 4, 4 (2002).

2. *"Functional MRI" studies (fMRI)*: When a region of the brain is activated in connection with cognitive activity, that activity "results in increased metabolic demand and that metabolic demand, in turn, brings increased blood flow to the region."[3] That shift in blood flow produces magnetic changes that are detectable by MRI and, in turn, "can be used to provide a detailed picture of brain regions involved in performing a particular cognitive task." *Id.* In essence, then, fMRI studies permit scientists to gain insight about which parts of the brain are active when a person is engaged in a specific activity – and they have produced an extensive literature characterizing cognitive regions, including the prefrontal cortex, that engage differently during adolescence compared to adulthood.[4]

3. *Diffusion Tensor Imaging (DTI)*: DTI studies directly measure the diffusion of water within the brain. Water diffuses in a more coherent fashion within the confines of white matter tracts, which link the areas of the brain that are responsible for cognitive function.[5] Tracking the flow of water molecules within the brain allows scientists to draw inferences about the growth and maturation of white matter connectivity. DTI studies consistently show that white matter connections that support cognition, emotion, and social behavior are not mature in adolescence.[6]

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[3] Beatriz Luna & John A. Sweeney, *Studies of Brain and Cognitive Maturation Through Childhood and Adolescence: A Strategy for Testing Neurodevelopmental Hypotheses*, 27 Schizophrenia Bull. 443, 448 (2001).

[4] Beatriz Luna, Aarthi Padmanabhan & Kirsten O'Hearn, *What has fMRI told us about the development of cognitive control through adolescence?* 72 Brain & Cognition 101 (2010); Sarah J. Ordaz et al., *Longitudinal Growth Curves of Brain Function Underlying Inhibitory Control Through Adolescence*, 33 J. Neuroscience 18109, 18109–24 (2013).

[5] *See* M.R. Asato et al., *White Matter Development in Adolescence: A DTI Study*, 20 Cerebral Cortex 2122, 2123 (2010).

[6] Daniel J. Simmonds et al., *Developmental Stages And Sex Differences Of White Matter And Behavioral Development Through Adolescence: A Longitudinal Diffusion Tensor Imaging (DTI) Study*, 92 NeuroImage 356, 356–57 (2014); Asato, *supra*, 20 Cerebral Cortex at 2122–31; C.

To be sure, these investigations do not directly test the complex cognitive behaviors involved in the full range of human activity, let alone criminal conduct specifically. Nonetheless, such MRI studies have in recent years contributed significantly, alongside other research, to the discovery of important foundational information about human brain development, and in particular about how human minds develop and mature during adolescence. Below, we summarize some of the key findings of the neuroscientific community in this regard.

A. THE HUMAN BRAIN UNDERGOES SIGNIFICANT STRUCTURAL, FUNCTIONAL, AND CHEMICAL CHANGES DURING ADOLESCENCE.

During adolescence, the brain is still a work in progress, with significant physical, functional, and chemical changes underway. These changes are essential for carrying out the "high-level executive functions" that are characteristic of adult cognition: the ability to plan, to consider and evaluate multiple pieces of information, to control emotions and impulses, to make decisions, and to evaluate consequences. And while adolescent minds tend to be advanced in these respects in comparison to children,"'[i]t is increasingly clear that adolescent brains are not yet fully mature in regions and systems related to higher-order executive functions.'" *Miller*, 132 S.Ct. at 2464 n.5; *see also Graham*, 560 U.S. at 68 ("parts of the brain involved in behavior control continue to mature through late adolescence").

1. Significant Changes In Brain Structure Take Place During Adolescence.

The brain undergoes significant physical changes during adolescence, continuing the path of development that begins much earlier in life. During childhood, the brain typically attains

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Lebel et al., *Diffusion Tensor Imaging of White Matter Tract Evolution Over the Lifespan*, 60 NeuroImage 340, 347 (2012).

adult size and weight.[7] Subdivided regional functions for cognition, basic vitals, reflexes, and other functions also develop during childhood. However, the brain continues to mature throughout adolescence.[8] Among the more notable physical changes in the brain during adolescence are, first, pruning of excess connections (synapses) between nerve cells and, second, myelination of long-range fibers that connect distant brain regions.[9] These structural changes enable the prefrontal cortex to more effectively communicate with the brain's other regions.[10] These improvements are believed to underlie the increased ability to control impulses, plan ahead, and avoid risk[11]—abilities that are not fully realized until late adolescence. Indeed, the prefrontal cortex—an area that plays a primary role in impulse control, risk avoidance, planning, and cognition—is among the last regions of the brain to complete its development.[12]

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[7] Jay N. Giedd, *The Teen Brain: Insights from Neuroimaging*, 42 J. of Adolescent Health 335, 336 (2008); B.J. Casey et al., *The Adolescent Brain*, 1124 Annals N.Y. Acad. Sci. 111, 114 (2008).

[8] Casey, *supra*, 1124 Annals N.Y. Acad. Sci. at 114.

[9] Laurence Steinberg, *Adolescent Development and Juvenile Justice*, 5 Ann. Rev. Clinical Psychol. 47, 54 (2009).

[10] Beatriz Luna, *The Maturation of Cognitive Control and the Adolescent Brain* in *From Attention to Goal-Directed Behavior* 257–58 (Francisco Aboitiz & Diego Cosmelli eds, 2009).

[11] Steinberg, *supra*, 5 Ann. Rev. Clinical Psychol. at 54; Luna, *supra*, The *Maturation of Cognitive Control and the Adolescent Brain* at 253.

[12] Nitin Gogtay et al., *Dynamic Mapping of Human Cortical Development During Childhood Through Early Adulthood*, 101 Proc. Nat'l Acad. Sci. 8174, 8177 (2004); 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. Neuroscience 8819, 8828 (2001); B.J. Casey et al., *Structural and Functional Brain Development and its Relation to Cognitive Development*, 54 Biological Psychol. 241, 245–46 (2000); Allan L. Reiss et al., *Brain Development, Gender and IQ in Children: A Volumetric Imagining Study*, 119 Brain 1763, 1770 (1996).

a. *Synaptic Pruning*

Synaptic pruning is a term that scientists use to describe the process of eliminating unnecessary synaptic connections.[13] Synapses are the connecting points in the brain that enable one neuron to communicate with others by transmitting chemical signals. Somewhat paradoxically, the brain has an excess of these connections at birth,[14] and shedding underutilized synapses is believed to support adaptation to an individual's environmental demands. The pruning of redundant or otherwise extraneous synaptic connections is believed to improve information processing and the fidelity of neural responses.[15] In that sense, "[s]ynaptic pruning enhances the ability to support complex computations necessary for higher-order behavior, such as the ability to enact plans that allow for response inhibition and retaining representations of goals in working memory."[16] Regions of the brain that control vision and hearing undergo reductions in gray matter that are completed by ages 7 and 10, respectively.[17] Synaptic pruning in the prefrontal cortex and other regions of the brain involved in executive functioning,

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[13] Casey, *supra*, 54 Biological Psychol. at 242-43; Gogtay, *supra*, 101 Proc. Nat'l Acad. Sci. at 8178; Linda Spear, *The Behavioral Neuroscience of Adolescence* 81-90 (2010); Peter Huttenlocher, *Neural Plasticity: The Effects of Environment on the Development of the Cerebral Cortex* 41, 46–47, 52–58, 67 (2002).

[14] *See, e.g.*, Luna, *supra*, *The Maturation of Cognitive Control and the Adolescent Brain* at 257.

[15] *Id.*; Casey, *supra*, 54 Biological Psychol. at 242–43; Gogtay, *supra*, 101 Proc. Nat'l Acad. Sci. at 8178; Spear, *supra*, *The Behavioral Neuroscience of Adolescence* at 81–90; Huttenlocher, *supra*, Neural *Plasticity: The Effects of Environment on the Development of the Cerebral Cortex* at 41, 46–47, 52–58, 67.

[16] Luna, *supra*, *The Maturation of Cognitive Control and the Adolescent Brain* at 267.

[17] Peter R. Huttenlocher & A.S. Dabholkar, *Regional Differences in Synaptogenesis in Human Cerebral Cortex*, 387 J. Comp. Neurology 167, 175 (1997); Peter R. Huttenlocher, *Morphometric Study of Human Cerebral Cortex Development*, 28 Neuropsychologia 517, 523 (1990).

however, does not reach adult levels until after adolescence.[18] Other regions of the brain that support higher-level functioning—such as areas relevant to social cognitive functions and motivation—also are not generally pruned to adult levels until late in adolescence.[19] The delayed maturation of brain processes underlying cognition likely contributes to adolescents' diminished cognitive capacity, in relation to adults, to process complex demands.[20]

b. *Myelination*

A second major physical change that occurs in the adolescent brain is myelination—the process by which the brain's neural pathways become surrounded by a white fatty tissue called myelin.[21] Much as insulation helps facilitate the transmission of electricity across wires, myelination improves the transmission of neuronal signals, allowing, for example, for the prefrontal cortex to more rapidly communicate with distant parts of the brain.[22] Myelination also assists in the integration of information needed for emotional and cognitive control systems.[23] "Improved connectivity within the prefrontal cortex," one study notes, "should be associated

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[18] Zdravko Petanjek et al., *Extraordinary Neoteny of Synaptic Spines in the Human Prefrontal Cortex*, 108 Proc. Nat'l Acad. Sci. 13281, 13281-86 (2011); Luna, *supra*, *The Maturation of Cognitive Control and the Adolescent Brain* at 257-58; Steinberg, *supra*, 5 Ann. Rev. Clinical Psychol. at 54-55; Laurence Steinberg, *A Social Neuroscience Perspective on Adolescent Risk-Taking*, 28 Developmental Rev. 78, 94 (2008).

[19] *See* Jay N. Giedd, *supra*, 42 J. Adolescent Health at 338-39.

[20] Steinberg, *supra*, 5 Ann. Rev. Clinical Psychol. at 54-55.

[21] Steinberg, *supra*, 28 Developmental Rev. at 94; Elkhorn Goldberg, *The Executive Brain: Frontal Lobes and the Civilized Mind* 144 (2001).

[22] The insulation provided by myelination "speeds . . . neural signal transmission" and "communication between different regions of the brain [are] faster and more reliable." Goldberg, *supra*, *The Executive Brain: Frontal Lobes and the Civilized Mind*, at 144; Luna, *supra*, *The Maturation of Cognitive Control and the Adolescent Brain* at 257.

[23] Steinberg, *supra*, 5 Ann. Rev. Clinical Psychol. at 54-55; Luna, *supra*, *The Maturation of Cognitive Control and the Adolescent Brain* at 253.

with subsequent improvements in 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."[24] The process of myelination continues into early adulthood.[25]

2. Significant Changes in Brain Function Take Place During Adolescence.

a. *Functional Brain Maturation and Executive Function*

Research findings also indicate that adolescents' ability to plan and carry out behaviors in order to achieve an identified goal is still developing and continues to do so into adulthood. This reduced capacity in adolescents compared to adults reflects developments in two aspects of cognitive function. First, adolescents are less able than adults to exercise high levels of inhibitory control—the ability to voluntarily suppress reflexive or enticing responses. Second, relative to adults, adolescents generally have lower working memory capacity, which is commonly described as "the ability to hold task-relevant information in mind long enough to use it to attain a task-relevant goal."[26]

Inhibitory control relies on the ability of "top" executive brain systems to influence "bottom" response systems. For example, one test of inhibitory control is known as the antisaccade task. In antisaccade experiments, subjects are presented with a screen on which a visual stimulus (such as a colored dot) suddenly appears, and are asked to look away from the

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[24] Steinberg, *supra*, 28 Developmental Rev. at 94; *see also* Luna, *supra*, *The Maturation of Cognitive Control and the Adolescent Brain* at 267.

[25] Paul I. Yakovlev & Andre-Roch Lecours, *The Myelogenetic Cycles of Regional Maturation of the Brain* in *Regional Development of the Brain in Early Life* 3–70 (AlexandreMinkowski ed., 1967).

[26] K. Suzanne Scherf, John A. Sweeney & Beatriz Luna, *Brain Basis of Developmental Change in Visuospatial Working Memory*, 18 J. Cognitive Neuroscience 1045, 1045 (2006).

stimulus rather than at it. This requires test subjects to engage their executive brain systems to suppress the natural and reflexive impulse to look at an image that has just come into view. A similar test of inhibitory control is the go/no go task, in which subjects are asked to press a button in response to repeated stimuli but to refrain from pressing the button in response to a specific, infrequently occurring stimulus. Even young children have the ability to suppress reflexive responses such as these, but studies have shown that this ability continues to develop and improve throughout adolescence and into adulthood.[27]

Studies show similar development patterns for working memory. In working memory tasks, subjects are given a goal and must keep it in working memory over a period of time, sometimes even as a new source of stimulus intervenes. Again, although working memory is available in childhood, performance on these tasks improves through adolescence and into adulthood.[28]

Studies using fMRI support the conclusion that executive function continues to develop in adolescence. These studies focus on the prefrontal cortex, which has unique patterns of interconnectivity with the rest of the brain and is crucial to executive function. By gauging activity in the prefrontal cortex during working memory and inhibitory response tasks, fMRI studies provide insight into the development of executive function. These studies show that although even children are able to engage the prefrontal cortex during working memory tasks, patterns of activity in the prefrontal cortex change with age for more difficult working memory

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[27] Beatriz Luna et al., *Maturation of Cognitive Processes from Late Childhood to Adulthood*, 75 Child Development1357, 1369 (2004).

[28] *Id.*

tasks.[29] Developmental differences have also been found in prefrontal cortex activity in inhibitory control tasks.[30] And a recent longitudinal fMRI study, in which individuals were tested yearly, showed strong age-related differences in the activation of a region within the prefrontal cortex that is involved in monitoring performance and error correction. The results of the study suggested that although adolescents are able to exercise inhibitory control, they do not yet have a fully developed capacity to supervise their responses and correct their own errors.[31]

b. *Functional Brain Maturation and Reward Processing*

Scientists have also demonstrated that there are developmental differences between adolescents and adults in the way in which the brain perceives and processes rewards.[32] These differences appear to be rooted in the ventral striatum, a region of the brain "that has consistently been associated with all phases of the processing of rewards, including detection, anticipation, and outcome and may underlie bias for immediate over future rewards."[33] When asked to complete tasks linked to monetary rewards, adolescents display greater activity in the ventral

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[29] Charles F. Geier et al., *Development of Working Memory Maintenance*, 101 J. Neurophysiology 84, 84-99 (2009); Eveline A. Crone et al., *Neurocognitive Development of the Ability to Manipulate Information in Working Memory*, 103 Proc. Nat'l Acad. Sci. 9315, 9315–20 (2006).

[30] Beatriz Luna et al., *Maturation of Widely Distributed Brain Function Subserves Cognitive Development*, 13 Neuroimage 786, 786 (2001); Silvia A. Bunge et al., *Immature Frontal Lobe Contributions to Cognitive Control in Children: Evidence from fMRI*, 33 Neuron 301, 301–11 (2002).

[31] Ordaz, *supra*, 33 J. Neuroscience at 18119–20.

[32] Adriana Galván, *The Teenage Brain: Sensitivity to Rewards*, 22 Current Directions Psychol. Sci. 88, 88–91 (2013).

[33] Aarthi Padmanabhan, et al. *Developmental Changes In Brain Function Underlying The Influence of Reward Processing on Inhibitory Control*, 1 Developmental Cognitive Neuroscience 517 (2011) (citations omitted).

striatum than do adults and children.[34] The same exaggerated pattern of activation is observed in adolescents compared to adults when asked to complete non-monetary reward tasks.[35] In addition, when obtaining the reward requires exercising cognitive control, adolescents show increased activity both in the ventral striatum and in the areas of the brain that are key to performing the cognitive task.[36]

At the same time, adolescents have less developed abilities to integrate inputs from cortical and subcortical brain regions, which may impede their ability to choose a delayed reward over a more immediate one.[37] And during reward-oriented tasks, adolescents show decreased activity of the orbitofrontal cortex, a region of the prefrontal cortex that is involved in assessing the significance of a reward compared to the costs of obtaining it.[38] This combination of increased sensitivity to rewards and decreased ability to value rewards versus costs suggests that adolescents may be more inclined to accept large risks in pursuing comparatively insignificant returns.[39] Indeed, research suggests that there is a strong relationship between youth who exhibit

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[34] *Id.*; Adriana Galvan et al., *Earlier Development of the Accumbens Relative to Orbitofrontal Cortex Might Underlie Risk-Taking Behavior in Adolescents*, 26 J. Neuroscience 6885, 6885–91 (2006).

[35] Adriana Galván & Kristine M. McGlennen, *Enhanced Striatal Sensitivity to Aversive Reinforcement in Adolescents Versus Adults*, 25 J. Cognitive Neuroscience 284, 290 (2013).

[36] Padmanabhan, *supra*, 1 Developmental Cognitive Neuroscience 517.

[37] Charles Geier & Beatriz Luna, *The Maturation of Incentive Processing and Cognitive Control*, 93 Pharmacology Biochemistry & Behav., 212, 212-18 (2009).

[38] Galvan et al., *supra*, 26 J. Neuroscience at 6885-92; Padmanabhan, *supra*, 1 Developmental Cognitive Neuroscience 517.

[39] Emily Barkley-Levenson & Adriana Galván, *Neural Representation of Expected Value in the Adolescent Brain*, 111 Proc. Nat'l Acad. Sci. 1646, 1648-49 (2014).

enhanced reward-related activation in the ventral striatum and self-reported real-life risky behavior.[40]

c. *Functional Brain Maturation and Socio-Emotional Cognition*

Finally, developmental studies show that adolescents have greater difficulty in processing socioemotional stimuli and, in comparison to adults, are more likely to be influenced by the presence of peers. One area of the brain that plays a primary role in processing emotional stimuli is the amygdala. Adolescents demonstrate weaker connections between the amygdala and prefrontal cortex than do adults, which may explain the distinctions in how they process socioemotional stimuli.[41] For example, fMRI studies show that adolescents have a harder time assessing other people's intentions and are less able to engage executive control regions to process rejection.[42]

The presence of peers has a stronger influence on the reward regions of the brain in adolescents than in adults.[43] Adolescents are more willing than adults to take risks when in the presence of peers, and demonstrate correspondingly increased activity in the brain's reward processing regions when peers are present.[44] Moreover, when receiving peer approval,

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[40] Adriana Galván et al., *Risk-Taking and the Adolescent Brain: Who is at Risk?* 10 Developmental Sci. F8, F12-13 (2007); Eva H. Telzer et al., *Meaningful Family Relationships: Neurocognitive Buffers of Adolescent Risk Taking*, 25 J. Cognitive Neuroscience 374, 383 (2013).

[41] Leah H. Somerville, Rebecca M. Jones & B.J. Casey, *A Time of Change: Behavioral and Neural Correlates of Adolescent Sensitivity to Appetitive and Aversive Environmental Cues*, 72 Brain and cognition 124, 126 (2010).

[42] B. Gunther Moor et al., *Do You Like Me? Neural Correlates of Social Evaluation and Developmental Trajectories*, 5 Soc. Neuroscience, 461, 461–82 (2010).

[43] Jason Chein et al., *Peers Increase Adolescent Risk Taking by Enhancing Activity in the Brain's Reward Circuitry*, 14 Developmental Sci. F1, F2 (2011).

[44] *Id.*

adolescents show greater activity in brain regions involved in action, which may indicate that positive reinforcement from peers drives adolescents to take action more readily than it does adults.[45]

3. Significant Changes in Brain Chemistry Related to Reward Processing Take Place During Adolescence.

In addition to structural changes, the adolescent brain also goes through significant neurochemical changes that affect learning, memory cognition, emotion, and reward processing. "[R]eward-related regions of the brain and their neurocircuitry undergo particularly marked developmental changes during adolescence."[46] Specifically, the human brain relies on chemicals known as neurotransmitters to carry, boost, and modulate signals between neurons and other cells in the body. One such neurotransmitter is dopamine, which has been shown to regulate emotion and motivation processes.[47] "Dopamine is hypothesized to be the primary transmitter that acts within and across limbic, striatal, and frontal circuitry to promote incentive-guided behavior and its regulation."[48]

Animal studies provide strong evidence that dopamine is available in greater concentrations in adolescent brains.[49] Moreover, adolescents undergo "a rapid and dramatic

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[45] Rebecca M. Jones et al., *Adolescent-Specific Patterns of Behavior and Neural Activity During Social Reinforcement Learning*, 14 Cognitive Affective & Behavioral Neuroscience 683 (2014).

[46] Tamara L. Doremus-Fitzwater, Elena I. Varlinskaya & Linda P. Spear, *Motivational Systems in Adolescence: Possible Implications for Age Differences in Substance Abuse and other Risk-Taking Behaviors*, 72 Brain & Cognition 114, 114 (2010).

[47] *Id.*

[48] Dustin Wahlstrom et al., *Developmental Changes in Dopamine Neurotransmission in Adolescence: Behavioral Implications and Issues in Assessment*, 72 Brain & Cognition 146, 148 (2010).

[49] *Id.* at 152 ("[T]he available evidence suggests that both primates and rodents exhibit increases in functionally available dopamine during adolescence, though differences exist with respect to

increase in dopaminergic activity within the socioemotional system."[50] This change is due in part to a remodeling of dopamine receptors in adolescence.[51] In early adolescence, dopamine receptors proliferate in the paralimbic and prefrontal cortical regions of the brain.[52] These dopamine receptors are subsequently reduced and redistributed. "As a result of this remodeling, dopaminergic activity in the prefrontal cortex increases significantly in early adolescence and is higher during this period than at any other point in development."[53]

The increase in dopamine during adolescence "may have important implications for sensation seeking," for example, by causing "potentially rewarding stimuli to be experienced as even more rewarding, thereby heightening the salience of rewards in situations in which both

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the regions and aspects of the dopamine system affected. In primates, cortical and subcortical tissue concentrations of dopamine are increased during adolescence.").

[50] Steinberg, *supra*, 5 Ann. Rev. Clinical Psychol.at 54. *See also* Wahlstrom, *supra*, 72 Brain & Cognition at 152 ("[D]opaminergic innervation of the frontal cortex also peaks during adolescence relative to childhood and adulthood, specifically in cortical layer III, which contains pyramidal cells responsible for cortico-cortical information processing."); Casey, *supra*, 1124 Annals N.Y. Acad. Sci. at 113 ("There are significant peaks in dopamine expression during adolescence. Dopamine projections to the prefrontal cortex continue to develop into early adulthood, with dopamine levels peaking in the prefrontal cortex during adolescence versus earlier or later in life in nonhuman primates (Rosenberg & Lewis 1994, 1995) and in rats (Kalsbeek et al. 1988). Dopamine receptor expression is highest in the accumbens during early adolescence (Tarazi et al. 1998). These findings in rodents suggest that there are specific regions undergoing structural changes, and therefore, connections and communication between subcortical and cortical regions are in transition and in flux during adolescence. Significant evidence suggests that the neuroanatomical changes described above are also occurring during adolescence in humans, but our methods for studying humans only provide an approximate index of such changes.").

[51] Wahlstrom, *supra*, 72 Brain & Cognition at 146.

[52] *Id.* at 152; *see also id.* ("D_1 and D_2 receptor densities appear to be heightened during adolescence compared to adulthood in both cortical and subcortical regions, though peaks in receptor density occur in childhood.").

[53] Laurence Steinberg, *Should the Science of Adolescent Brain Development Inform Public Policy?* 64 Am. Psychol. 739, 743 (2009).

rewards and costs are present."[54] Moreover, this surge in dopamine occurs at a time when other brain structures and functions, including those that involve self-regulation, are not fully mature, potentially further increasing adolescents' susceptibility to risky behaviors.[55] This process has been described as akin to "starting the engines without a skilled driver behind the wheel."[56]

CONCLUSION

Drawing in part on recent advances in MRI-based neuroimaging, scientists have demonstrated that fundamental changes to the human brain occur throughout the adolescent years. Because synaptic pruning and myelination are as yet unfinished, the brain's structural capacity to engage in higher-order executive control of behavior, such as planning and risk avoidance, likewise remains incomplete. In functional terms, the brain's ability to inhibit inappropriate responses, maintain working memory, and process socioemotional signals—including for the purpose of appropriately weighing peer approval or disapproval—remains underdeveloped. And in chemical terms, the adolescent brain appears especially attuned to the prospect of short-term reward.

Scientists continue to consider precisely how these developmental phenomena combine to influence adolescent behavior. *Amici* are of the view that while "adolescents *can* exhibit sophisticated voluntary behavior,"[57] "[t]hese immaturities result in a system that is able to exert

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[54] *Id.*

[55] Laurence Steinberg et al., *Age Difference in Sensation Seeking and Impulsivity as Indexed by Behavior and Self-Report: Evidence for a Dual Systems Model*, 44 Developmental Psychol. 1764, 1764 (2008).

[56] *Id.*

[57] Beatriz Luna, *What Has fMRI Told Us About the Development of Cognitive Control Through Adolescence?*, 72 Brain Cognition 101 (2010).

cognitive control, but in an inconsistent manner with limited flexibility and motivational control."[58] That conclusion is not only consistent with the current state of scientific understanding, but also mirrors what common experience and legal principle have long suggested. Adolescents are advanced in comparison to children, but they are not fully developed behavioral actors. Neuroscience research shows that "[d]espite better cognitive, intellectual, and reasoning abilities than children, adolescents are not simply 'mini-adults' and despite immature emotion regulation, inexperience, and dependence on caregivers, adolescents are not overgrown children."[59] Adolescents are "in a distinct developmental stage that facilitates the adaptive transition from a state of dependence on caregivers to one of relative independence. However, along the road to autonomy, the very same characteristics that catalyze independence may lead adolescents to stumble into harmful behaviors."[60] They tend to chase short-term gain, discount risk, and undervalue long-term consequence. They are not as accomplished as adults when it comes to linking their plans to their goals, or to executing the plans that they do select. They are more prone to react in emotional situations. And, thankfully for all concerned, adolescents tend to mature as they age into adulthood. Importantly, these characteristics render the adolescent amenable to change, positive input, and intervention.[61]

Amici do not formally support either party to this case, and indeed do not believe that science should be the only driver of the legal rules governing juvenile sentencing. *Amici* do

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[58] Luna, *supra*, *The Maturation of Cognitive Control and the Adolescent Brain* at 258.

[59] Adriana Galván, *Insights About Adolescent Behavior, Plasticity and Policy from Neuroscience Research*, 83 Neuron 262, 262 (2014).

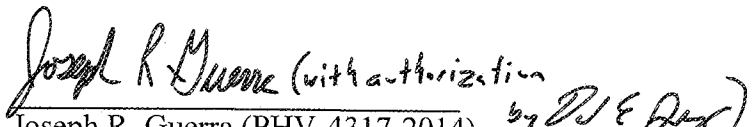
[60] *Id.*

[61] *Id.*; Eveline A. Crone & Ronald E. Dahl, *Understanding Adolescence as a Period of Social-Affective Engagement and Goal Flexibility*, 13 Nature Reviews Neuroscience 636, 636–650 (2012).

respectfully submit, however, that the scientific findings described in this brief can properly help to inform the Court's consideration of whether the sentence imposed in this case is inconsistent with the Eighth and Fourteenth Amendments to the United States Constitution, and with the Supreme Court's decision in *Graham v. Florida.*

Dated: September 2, 2014

Respectfully submitted,



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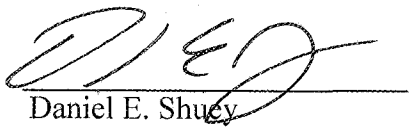

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