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The Poverty of the Neuroscience of Poverty: Policy Payoff or False Promise?

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## I. Introduction

The recent explosion of research linking human conduct to the operation of the brain has attracted attention from the popular media and a spectrum of scholars seeking to better understand a range of human behaviors.<sup>2</sup> The findings of neuroscience have been adduced to explain addiction,<sup>3</sup> criminal offending,<sup>4</sup> teenage risk-taking,<sup>5</sup> compulsive gambling and shopping,<sup>6</sup> academic underachievement,<sup>7</sup> deception<sup>8</sup>, and the prevention of social violence.<sup>9</sup> One expanding area of inquiry is the neuroscience related to the effects of social disadvantage. Social and cognitive scientists have long documented a range of behavioral and cognitive deficits that are found more commonly in people from deprived circumstances. In an effort to better understand and address these deficits, a growing number of brain scientists and behaviorists have turned their attention to studying “brains on poverty” – that is, the structural and functional characteristics of the brain that are linked to various aspects of low socioeconomic status (or SES). Scientists have

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<sup>2</sup> As Sally Satel Scott and Lilienfeld note in their recent book *BRAINWASHED: THE SEDUCTIVE APPEAL OF MINDLESS NEUROSCIENCE*, “[t]he media – and even some neuroscientists it seems – love to invoke the neural foundation of human behavior to explain everything” (2015), at ix.

<sup>3</sup> *Id.* at chapter 3.

<sup>4</sup> See, e.g., *A PRIMER OF CRIMINAL LAW AND NEUROSCIENCE* (Stephen Morse & Adina Roskies eds., 2013); see also Nita Farahany, *Neuroscience and Behavioral Genetics in US Criminal Law: An Empirical Analysis*, 2 *JOURNAL OF LAW AND THE BIOSCIENCES* 485 (2015); Ayal Aharoni, *Can Neurological Evidence Help Courts Assess Criminal Responsibility*, 1128 *ANNALS OF THE NEW YORK ACADEMY OF SCIENCES* 145 (2008).

<sup>5</sup> FRANCES JENSEN, *THE TEENAGE BRAIN* (2015); Laurence Steinberg, *Should the Science of Adolescent Brain Development Inform Public Policy?*, Article Adapted from the Annual Henry and Bryna David Lecture, Delivered at the National Academy of Sciences (Nov. 2011).

<sup>6</sup> See, e.g., *BRAINWASHED*, at 43-47; NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, *FROM NEURONS TO NEIGHBORHOODS* (2000).

<sup>7</sup> See, e.g., MARK KELMAN & GILLIAN LESTER, *JUMPING THE QUEUE* (1998).

<sup>8</sup> Giorgio Ganis & Julian Paul Keenan, *The Cognitive Neuroscience of Deception*, 4 *SOCIAL NEUROSCIENCE* 465 (2009).

<sup>9</sup> Michelle Bosquet Enlow et al., *Battling Blood in the Streets: How Can Neuroscience Promote Public Health and Support Public Policy to Prevent Community Violence?*, Harvard Law School (Sept. 7, 2016).

been especially concerned with deprivation and poverty experienced during childhood, with a corresponding focus on the consequences of low SES for the developing brain and associated childhood and adult behaviors.

The literature on the neuroscience of disadvantage (or “deprivation-neuroscience,” as I call it here) and the claims made on its behalf are the topic of this article. Researchers as well as policy-oriented consumers of this literature frequently suggest (and occasionally assert) that the discoveries of brain science can help generate more effective strategies for addressing poverty and deprivation and thus for reducing or eliminating its harmful effects.<sup>10</sup> This article will assess these claims by evaluating the practical payoff for law and policy of knowledge generated by research in the field. It will consider whether, using the techniques and methods now available and commonly deployed by researchers studying the brains of disadvantaged individuals, neuroscience research can contribute to our ability, over and above what we know or can discover from behavioral science and social observation, to devise and craft interventions to reduce poverty and its adverse consequences. Specifically, it will address whether brain science has made a unique, indispensable contribution to devising methods for preventing the negative behavioral and cognitive effects of social and economic disadvantage or for curing them once they occur.

In addressing these questions, the paper first reviews the literature that seeks to connect socioeconomic status (SES) to structural and functional aspects of the brain and the behaviors linked to those structures and functions. It then considers potential and actual claims regarding implications of the findings for devising effective policies and interventional strategies. As part of its assessment, the article will examine a controversy surrounding the implications of a recent and widely publicized report linking brain morphology, cognitive function, and childhood disadvantage. It will then evaluate a lengthy law review article arguing that neuroscientific findings on poverty’s effects on the developing brain dictate a revision of a federal law, the Individuals with

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<sup>10</sup> See *cites infra*.

Disabilities Education Act, extending special protections and benefits to children with learning disabilities.

Drawing on these discussions, the paper argues that neuroscientific research currently yields no useful information for shaping policy and designing effective interventions to address poverty and inequality and its associated consequences. Nor will it likely alleviate those problems in the foreseeable future. First, neuroscientific studies that examine the brain characteristics associated with deprivation do not, and generally cannot, establish causation. They do not distinguish between innate versus environmental influences on observed brain structure and function nor illuminate the range or extent of genetic-environmental (G x E) interactions. This limitation has important implications for policy. If behavioral deficits associated with poverty are not solely, or even mainly, the result of environmental deprivation (as opposed to innate, genetically programmed propensities that tend to correlate with disadvantaged circumstances, but are not produced by them), then those deficits are less likely to be subject to effective manipulation, at least through the type of preventative interventions that are a major focus of the developmental community to date.<sup>11</sup> Although prospective, randomized trials offer more potential for learning whether poverty's effects on brains (or, for that matter, on corresponding behaviors) can be effectively alleviated -- or whether heritable traits can be mitigated as well -- the ambit for such studies is extremely narrow. In general, because the neuroscience to date cannot, and is not designed to, sort out causal mechanisms, studies in this field offer little help in predicting whether any particular proposal, intervention, program, or policy designed to address poverty will work to prevent or cure the adverse behavioral or brain effects associated with that condition.

Second, and apart from any difficulties with causation, neuroscience offers few if any insights over and above knowledge

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<sup>11</sup> In contrast, there is no a priori reason to predict that interventions geared to *reversing* behavioral or brain deficits are more likely to succeed depending on whether genes or environmental factors play a dominant causal role. However, neuroscience has to date yielded no methods for reversing brain changes or behavioral defects associated with deprivation. Thus such cures have received little attention in the literature drawing on neuroscience.

generated from other fields, including most notably cognitive and behavioral psychology, into how society should address poverty and/or reverse its detrimental effects. The sole insight relevant to the explanatory and pragmatic power of brain science is that behaviors depend on brain states. There can be no observed change in behavior without a corresponding change in the brain. But that understanding, which is non-specific and contingent on no particular research findings, proceeds from a mechanistic, materialistic view of the human organism which has long been widely accepted in the biological and human sciences. This basic insight of biology neither establishes nor predicts the effectiveness of any policy designed to address social adversity and its supposed effects. The paper concludes that, for both theoretical and practical reasons, no legal or policy choices depend on specific observations about the developing brain's activities, shape, size, or connections and on how deprivation alters these. And such observations cannot predict, determine, identify, or establish what works to prevent or reverse documented brain deficits and corresponding behavioral shortcomings. The effectiveness of any interventions must ultimately be shown on a case by case basis through the accumulation of behavioral and social science evidence. And predictions about any policy's effectiveness are only as good as the behavioral evidence on which those predictions rely. Because neuroscience data on poverty's effects must always be correlated with behavioral observations, neuroscience can do no better than the behavioral evidence itself. It thus adds nothing to policy design, over and above what behavioral science can yield.

## II. Research on the neuroscience of deprivation

Numerous studies attempt to examine the link between various social outcomes and low socioeconomic status (SES), variably defined to include poverty, lack of education, low social capital, and other measures.<sup>12</sup> The biological and physiological correlates of these

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<sup>12</sup> See, e.g., Daniel A. Hackman, Martha J. Farah, & Michael J. Meaney, *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 NATURE REVIEWS NEUROSCIENCE 651, 651 (2010), stating that "SES is a complex construct that is based on household income, material resources, education and occupation, as well as related neighborhood and family

outcomes are the subject of research in several disciplines. Children from low SES families have been documented to experience broad consequences for health and well-being, including shorter life-spans and greater susceptibility to later illnesses and physical afflictions. Poverty has been tied to higher levels of stress, exaggerated reactivity, mental illness and emotional disturbance, impulsive and aggressive behaviors, and vulnerability to addiction.<sup>13</sup> These effects are believed to be mediated, at least in part, through the activation of hormonal and stress-reactive pathways, which operate through, and alter, brain circuitry, structure, and function.<sup>14</sup>

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characteristics, such as exposure to violence and toxins, parental care and provision of a cognitively stimulating environment.”

<sup>13</sup> See, e.g., W. Thomas Boyce, *A Biology of Misfortune*, 29 INSTITUTE FOR RESEARCH ON POVERTY FOCUS NEWSLETTER 1 (Spring/Summer 2012); Daniel A. Hackman, Martha J. Farah, and Michael J. Meaney, *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 NATURE REVIEWS NEUROSCIENCE 651, 651 (2010); Stephanie H.M. van Goozen, Graeme Fairchild, & Gordon T. Harold, *The Role of Neurobiological Deficits in Childhood Antisocial Behavior*, 17 CURRENT DIRECTIONS IN PSYCHOLOGICAL SCIENCE 224 (2008); ROBERT M. SAPOLSKY, STRESS AND COGNITION 1031-42 (2004) . See also, e.g., Vincent J Felitti et al., *Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study* , 14 AMERICAN JOURNAL OF PREVENTIVE MEDICINE 245 (1998); Vincent J Felitti . *Adverse Childhood Experiences and Adult Health*, 9 ACADEMIC PEDIATRICS 131 (2009); Vincent J Felitti et al., *Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study*, 14 AMERICAN JOURNAL OF PREVENTATIVE MEDICINE 245 (1998); Paul Tough, *How Kids Learn Resilience*, THE ATLANTIC, June 2016, <http://www.theatlantic.com/magazine/archive/2016/06/how-kids-really-succeed/480744/>.

<sup>14</sup> See, e.g., Daniel A. Hackman, Martha J. Farah, & Michael J. Meaney, *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 NATURE REVIEWS NEUROSCIENCE 651 (2010); Sonia J. Lupien et al., *Effects of Stress Throughout the Lifespan on the Brain*, 10 BEHAVIOUR AND COGNITION NATURE REVIEWS NEUROSCIENCE 434 (2009); Paula S. Nurius et al., *Life Course Pathways of Adverse Childhood Experiences toward Adult Psychological Well-being: A Stress Process Analysis*, 45 CHILD ABUSE & NEGLECT 143 (2015).

A large and growing body of research has focused specifically on the behavioral and cognitive deficits found in children growing up in deprived circumstances.<sup>15</sup> Specifically, it is well-documented that childhood SES is “correlated with intelligence and academic achievement from early childhood and through adolescence.”<sup>16</sup> On average, children from lower SES backgrounds have been observed to fall short on a range of cognitive tasks (including language skills, memory, visualization, and reasoning ability).<sup>17</sup> They also lag in non-cognitive behavioral traits (such as executive function, perseverance, self-control, discount rate and ability to delay gratification) that predict academic and life success. Consistent with this research, children from disadvantaged backgrounds are at enhanced risk to suffer from

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<sup>15</sup> Martha J. Farah, Kimberly Noble, & Hallam Hurt, *Poverty, Privilege, and Brain Development: Empirical Findings and Ethical Implications*, in *NEUROETHICS: DEFINING THE ISSUES IN THEORY, PRACTICE, AND POLICY* 277 (Judy Illes ed., 2006); NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, *FROM NEURONS TO NEIGHBORHOODS* (2000); MARK KELMAN & GILLIAN LESTER, *JUMPING THE QUEUE* (1998); Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 *TRENDS IN NEUROCOGNITIVE SCIENCE* 65 (2009); Daniel A. Hackman, Martha J. Farah, & Michael J. Meaney, *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 *NATURE REVIEWS NEUROSCIENCE* 651 (2010); W. Thomas Boyce, *A Biology of Misfortune*, 29 *INSTITUTE FOR RESEARCH ON POVERTY FOCUS NEWSLETTER* 1 (Spring/Summer 2012). See also Paul Tough, *How Kids Learn Resilience*, *THE ATLANTIC*, June 2016, <http://www.theatlantic.com/magazine/archive/2016/06/how-kids-really-succeed/480744/> (“More recently, researchers using variations on [the ACE] scale have found that an elevated ace score also has a negative effect on the development of a child’s executive functions and on her ability to learn effectively in school.”)

<sup>16</sup> Daniel A. Hackman, Martha J. Farah, & Michael J. Meaney, *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 *NATURE REVIEWS NEUROSCIENCE* 651, 651 (2010).

<sup>17</sup> NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, *FROM NEURONS TO NEIGHBORHOODS* (2000); Daniel A. Hackman et al., *Socioeconomic Status and Executive Function: Developmental Trajectories and Mediation*, 18 *DEVELOPMENTAL SCIENCE* 686 (2015); Gwendolyn M. Lawson et al., *Socioeconomic Status and the Development of Executive Function: Behavioral and Neuroscience Approaches*, in *EXECUTIVE FUNCTION IN PRESCHOOL-AGE CHILDREN: INTEGRATING MEASUREMENT, NEURODEVELOPMENT, AND TRANSLATIONAL RESEARCH* 259 (James A. Griffin, Peggy McCardle, & Lisa S. Freund eds., 2015).

academic difficulties in school, commit disciplinary infractions, and display self-destructive and anti-social behavior.<sup>18</sup>

The neuroscience of deprivation aims to build on these types of behavioral observations to generate a more specific brain-centered body of knowledge that is focused on defined neuroanatomical and neuro-functional systems. The goal is to detail how poverty and low SES might alter particular brain structures, activities, and connections, and to show how particular experiences tend to bring about those changes. Like other fields seeking to elucidate the neural basis for behavior, deprivation neuroscience requires connecting human conduct, traits, capacities, and decisionmaking to brain states, structures, and activities.

Pursuing this project has been made possible by rapid developments in methods for visualizing the brain. Brain scanning techniques that create highly detailed pictures of brain morphology and ongoing brain activity have enabled researchers to link up brain structure and function with various behavioral states or syndromes and to picture the brain in the process of performing a range of activities linked to cognitive abilities.<sup>19</sup> This and other techniques for mapping brain structures, tracing connections, and watching the brain in action have been deployed by neuroscientists and cognitive psychologists interested in the effects of early deprivation.<sup>20</sup>

Martha Farah and her colleagues have been particularly influential in galvanizing and shaping research in the field by developing a theoretical map, based on existing neurofunctional and neuroanatomical studies, that predicts the areas of the brain most likely to be affected by childhood

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<sup>18</sup> See, e.g., Christine Christle, Kristine Jolivette, & C. Michael Nelson, *School Characteristics Related to High School Dropout Rates*, 28 REMEDIAL AND SPECIAL EDUCATION 325 (2007); John M. Wallace et al., *Racial, Ethnic, and Gender Differences in School Discipline among U.S. High School Students: 1991-2005*, 59 NEGRO EDUCATIONAL REVIEW 47 (2008); John Paul Wright et al., *Prior Problem Behavior Accounts for the Racial Gap in School Suspensions*, 42 JOURNAL OF CRIMINAL JUSTICE 257, 257 (2014).

<sup>19</sup> See A PRIMER OF CRIMINAL LAW AND NEUROSCIENCE (Stephen Morse & Adina Roskies eds., 2013), chapters 1 & 2 for a description of fMRI.

<sup>20</sup> A PRIMER OF CRIMINAL LAW AND NEUROSCIENCE (Stephen Morse & Adina Roskies eds., 2013), chapter 3.



adversity. These researchers rely on the observation that “the SES disparity in cognitive development is not uniform across different neurocognitive systems but rather is more pronounced for some neurocognitive systems than for others.”<sup>21</sup> Their strategy is to hone in on particular brain structures known to be associated with the functional deficits observed in children subject to early adversity. For example, behavioral studies show that, compared to the general population, lower income children and adults tend to have poorer working memory,<sup>22</sup> less inhibitory control, and worse executive attention and function.<sup>23</sup> Other areas of documented deficiency are impaired spatial and visual cognition and inferior language skills, as reflected in vocabulary size, syntactic ability, and phonological awareness (that is, ability to distinguish words, variations in word usage, and meaning).<sup>24</sup> Additional deprivation-associated deficits include difficulties with cognitive control and reward processing.<sup>25</sup> Based on an analysis of these impairments, neuroanatomical knowledge, and their own research, Farah and colleagues have identified seven localized regional brain circuits, or “neurocognitive systems” likely to be affected by childhood deprivation, and ripe for more intensive investigation.<sup>26</sup> Much of the work of Farah

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<sup>21</sup> Martha J. Farah et al., *Childhood Poverty: Specific Associations with Neurocognitive Development*, 1110 BRAIN RESEARCH 166, 168 (2006).

<sup>22</sup> Gary W. Evans & Michelle A. Schamberg, *Childhood Poverty, Chronic Stress, and Adult Working Memory*, 106 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES 6545 (2009).

<sup>23</sup> Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 TRENDS IN NEUROCOGNITIVE SCIENCE 65 (2009); Gary W. Evans, *The Environment of Childhood Poverty*, 59 AMERICAN PSYCHOLOGIST 77 (2004); Gary W. Evans & Jennifer Rosenbaum, *Self-regulation and the Income-Achievement Gap*, 23 EARLY CHILDHOOD RESEARCH QUARTERLY 504 (2008).

<sup>24</sup> Martha J. Farah, Kimberly Noble, and Hallam Hurt, *Poverty, Privilege, and Brain Development: Empirical Findings and Ethical Implications*, in NEUROETHICS: DEFINING THE ISSUES IN THEORY, PRACTICE, AND POLICY 277 (Judy Illes ed., 2006); see also Kimberly G. Noble, Martha J. Farah, & Bruce D. Mccandliss, *Socioeconomic Background Modulates Cognition–achievement Relationships in Reading*, 21 COGNITIVE DEVELOPMENT 349 (2006).

<sup>25</sup> Kimberly G. Noble, Bruce D. Mccandliss, & Martha J. Farah, *Socioeconomic Gradients Predict Individual Differences in Neurocognitive Abilities*, 10 DEVELOPMENTAL SCIENCE 464 (2007).

<sup>26</sup> These areas include, inter alia, the prefrontal/executive systems, the lateral cortex/working memory, the anterior cingulate cortex/cognitive control system, and

and her group, and the research linking brain structure and function to low SES generally, relies heavily on neuroscientific techniques of recent vintage. Most important is magnetic resonance imaging (MRI), which can be used to create individual images of brain structures as well as dynamic pictures of localized neural activity. A static, or structural, MRI image is generated when a brain is placed in a magnetic field and the brain molecules are stimulated by radiofrequency pulses. Areas that differ in density and composition create disparate signals, which are captured by the machine to create a contrasting visual image of the brain.<sup>27</sup> Functional MRI (fMRI) depends on measuring three-dimensional hemodynamic (blood flow) patterns associated with by neural activity. Three blood flow patterns are then translated into visual images. For both structural and functional MRI, the techniques of visualization and the presentation of the data vary, depending on research design and available evidence. Some studies relying on single images of individual subjects, whereas others aggregate multiple images from one or more subjects to create a composite pictures of activity levels associated with particular conditions or tasks.

Yet another technique, involving a smaller number of studies, makes use of event-related brain potentials or ERPs, which are stylized electrical signals measured at the brain surface. By picking up on patterned processes taking place throughout the brain, this technique can detect and reflect changes in localized activity. Ordinarily, the electrical recording is timed to coordinate with a stimulus (usually auditory or visual, such as a word or picture or sound flashed to the subject) or with the performance of a functional task (such as listening to a story). The ERPs reported by researchers using this technique are generally averaged

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the ventromedial prefrontal cortex/reward processing system. Martha J. Farah et al., *Childhood Poverty: Specific Associations with Neurocognitive Development*, 1110 *BRAIN RESEARCH* 166 (2006). See also Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 *TRENDS IN COGNITIVE SCIENCES* 65 (2009); Noble, Kimberly G., Bruce D. Mccandliss, and Martha J. Farah. "Socioeconomic Gradients Predict Individual Differences in Neurocognitive Abilities." *Developmental Science* *Developmental Sci* 10.4 (2007): 464-80.

<sup>27</sup> See A PRIMER OF CRIMINAL LAW AND NEUROSCIENCE 40-43 (Stephen Morse & Adina Roskies eds., 2013), describing MRI and fMRI techniques in detail.

over hundreds or thousands of recordings, gleaned from one or more subjects or trials.

Overall, approximately two dozen studies published since 2001 fall into the category of deprivation neuroscience. This work uses either neuro-imaging or ERP techniques to examine the structure or function of the brains of individuals who have suffered some documented form of developmental deprivation, or are known to suffer impairments or deviations from normal functioning linked to lower SES. The great majority of this research relies on some variant of MRI or fMRI methodology. This imaging research, in turn, can be divided into two broad categories. The first (termed here “retrospective”) is designed to mine and analyze data from numerous scans performed and collected in the course of large demographic studies, conducted within the past 15 years or so, that are not specifically directed at examining the effects of low SES. One example is a large multi-site multi-year National Institutes of Health MRI imaging study of normal brain development.<sup>28</sup> Because these data sets contain a large number of brain images of study subjects, as well as information on background, history, and SES, and, in some cases, cognitive attributes, behavior, or performance on specific tasks, they are useful for studying the brain correlates of early disadvantage.

The second type of research (“prospective”) is specifically designed to establish correlations or answer defined questions about the neuro-functional correlates of particular behaviors or performance deficits found in lower SES individuals. These studies usually involve smaller numbers of subjects.

The analyses from the retrospective studies, which number in the single digits in the literature, focus on measurement and morphology, including the size, thickness, and shape of brain centers linked to known cognitive or behavioral functions. The goal is to explore correlations between brain structure and functional impairments by investigating

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<sup>28</sup> See, e.g., Alan C. Evans and the Brain Development Cooperative Group, *The NIH MRI Study of Normal Brain Development*, Volume 30 #1 NEUROIMAGE 184-2-2 (March 2006)(describing the study). See also the project website, found at <https://pediatricmri.nih.gov/nihpd/info/index.html>.

whether particular brain centers found in lower SES individuals, or those who have suffered documented forms of early deprivation, differ measurably from those of less deprived subjects. Some research asks whether brain changes observed on brain scans correspond to functional defects more often found in lower SES individuals. One of the first reports of this type looked at brain scans performed on a group of severely deprived Rumanian orphans using an earlier imaging technique, predating fMRI technology, called Positron Emission Tomography, or PET, which provides a measure of oxygen consumption by brain tissue that reflects relative brain function and integrity. The PET scans of the study subjects showed diminished volume and activity in frontal areas (which are associated with self-control and executive function), as well as in the amygdala and hippocampus (which are seats of emotion and memory).<sup>29</sup> A second retrospective study, which reviewed scans from 445 American subjects of various ages, claimed to document an average decrease in the volume of the hippocampus of children who suffered from deficient maternal care (which tends to be associated with lower SES), which was established by self-report and objective evidence collected in the study.<sup>30</sup> A third report, by Nicole Hair and her colleagues, was based on an analysis of fMRI scans performed on 389 students of various ages. The authors claimed to document a 3-way statistical correlation between a paucity of gray matter (brain tissue) in various parts of the brain, being raised by lower status parents (as defined and documented in the research data), and measured deficits in academic achievement.<sup>31</sup> Yet another study, using 203 scans from 77 infants conducted serially over several months, concluded that “infants from low income families had lower volumes of gray matter” and also that less gray matter was associated with a greater risk of disruptive

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<sup>29</sup> Harry T. Chugani et al., *Local Brain Functional Activity Following Early Deprivation: A Study of Post-institutionalized Romanian Orphans*, 14 *NEUROIMAGE* 1290 (2001).

<sup>30</sup> Claudia Buss et al., *Maternal Care Modulates the Relationship between Prenatal Risk and Hippocampal Volume in Women but Not in Men*, 27 *JOURNAL OF NEUROSCIENCE* 2592 (2007).

<sup>31</sup> Nicole L. Hair et al., *Association of Child Poverty, Brain Development, and Academic Achievement*, 169 *JAMA PEDIATRICS* 822 (2015).

behaviors later in childhood.<sup>32</sup> In a group of 283 scans conducted on children of various ages, Gwendolyn Lawson and her colleagues, working with Martha Farah, observed a statistical relationship between one aspect of SES (parental education), but not others (such as household income) and prefrontal cortical thickness.<sup>33</sup> Finally, just last year, as widely reported in the press, Kim Noble and colleagues, using a large database of more than 1000 scans collected through a government-sponsored pediatric imaging project, reported that low childhood SES, and especially lower levels of income and parental education, are associated with smaller brain surface area in children between 3 and 20 years old, with severely deprived children showing the most pronounced effects. The observed reduction in surface morphology was widely dispersed, affecting centers for language, spatial perception, memory, and executive function. The paper also reported subjects' performance on various tests of cognitive function known to be associated with particular brain areas, and attempted to match these with localized brain size data. A significant three-way correspondence (among measures of disadvantage, smaller brain surface area, and behavioral shortcomings) was found for some functions (self-control and working memory) but not others (verbal skills).<sup>34</sup>

Instead of mining existing datasets, a second set of studies is specifically geared to collecting data designed to link brain structure and function, childhood experiences, and existing behavioral characteristics. These studies generally tend to be smaller, and thus have fewer data points, than retrospective research based on existing data repositories.

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<sup>32</sup> Jamie L. Hanson et al., *Family Poverty Affects the Rate of Human Infant Brain Growth*, 8 PLOS ONE e80954 (2013).

<sup>33</sup> Gwendolyn M. Lawson et al., *Associations between Children's Socioeconomic Status and Prefrontal Cortical Thickness*, 16 DEVELOPMENTAL SCIENCE 641 (2013).

<sup>34</sup> Kimberly G. Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 NATURE NEUROSCIENCE 773 (2015). More recent data from this study, including a more extensive analysis of age-related changes in cortical thickness and brain surface area associated with low SES, has been posted online by Noble and her colleagues. See Luciane R. Piccolo, et al, *Age-Related Differences in Cortical Thickness Vary by Socioeconomic Status*, PLOS ONE, Sept. 19, 2016, online at <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0162511>.

The work also tends to be directed at examining highly specific behavioral functions known to be compromised in disadvantaged children, with targeting of localized brain areas associated with them. The resulting body of work is a congeries of discrete, and often disjointed, observations that focus on distinct brain centers and use a variety of different measures of social disadvantage. For example, Mark Gianaros and colleagues, generating functional FMRI images on 100 adult experimental subjects from a range of backgrounds, claim to establish a correlation between perceived parental social standing and “greater amygdala reactivity to threatening facial expression.”<sup>35</sup> Another research group, recruiting 49 subjects in their 20s, found that individuals with lower family income at age 9 had selectively reduced activity in the prefrontal cortex (an area associated with self-control and executive function) as measured using fMRI techniques. The poorer subjects were also measurably less able than higher SES subjects to suppress amygdala activation during emotional stimulation.<sup>36</sup> Based on fMRI scans performed on 145 children over a six year period, Luby and colleagues found lower volumes in multiple brain centers in lower SES children, and especially those with a history of inadequate maternal care. These observations were also correlated with selective behavioral impairments, such as poor self-control.<sup>37</sup>

Likewise, another scanning study of 58 adolescents reported greater cortical thickness in all lobes of the brain for higher income students in the sample, with thicker morphology predicting better performance on tests of general intellectual ability.<sup>38</sup> Yet another fMRI scanning study,

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<sup>35</sup> Peter Gianaros et al., *Potential Neural Embedding of Parental Social Standing*, 3 SOCIAL COGNITIVE & AFFECTIVE NEUROSCIENCE 91, 91 (2008); *see also* Peter Gianaros et al., *Perigenual Anterior Cingulate Morphology Covaries with Perceived Social Standing*, 2 SOCIAL COGNITIVE AFFECTIVE NEUROSCIENCE 161 (2007).

<sup>36</sup> Pilyoung Kim et al., *Effects of Childhood Poverty and Chronic Stress on Emotion Regulatory Brain Function in Adulthood*, 110 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES 18442 (2013).

<sup>37</sup> Joan Luby et al., *The Effects of Poverty on Childhood Brain Development: The Mediating Effect of Caregiving and Stressful Life Events*, 167 JAMA PEDIATRICS 1135 (2013).

<sup>38</sup> Allyson P. Mackey et al., *Neuroanatomical Correlates of the Income-Achievement Gap*, 26 PSYCHOLOGICAL SCIENCE 925 (2015).

which looked at 150 first grade students, reported that depressed activity in specialized areas for face recognition and language (the left fusiform and perisylvian areas) during active reading was linked to weaker phonological skills, which were in turn more prevalent in the lower SES subjects.<sup>39</sup> A different prospective study using 49 black middle school children (approximately 14-years old) revealed an association between lower levels of early childhood nurturance (ages 4-8) established by questionnaire and a smaller size hippocampus (which is a brain structure involved in memory and emotion) as documented on fMRI. No association was found between scan-documented volumes and other aspects of childhood environmental stimulation, which were also assessed. The authors concluded that “hippocampal volume is especially associated with parental nurturance.”<sup>40</sup> In another dynamic fMRI study of 14 pre-kindergarten children (age 5) that attempted to establish a three-way correlation between brain, behavior, and the environment,<sup>41</sup> researchers observed a strong SES-related gradient in the degree of lateral specialization in the left inferior frontal gyrus, an area known to be involved in reading tasks, with reduced lateralization in lower SES students. However, the association between measured reading-related skills (as assessed through a rhyming task) and degree of lateralization and SES in the sample was weak or non-existent.<sup>42</sup> Finally, another

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<sup>39</sup> Kimberly G. Noble, Martha J. Farah, & Bruce D. Mccandliss, *Socioeconomic Background Modulates Cognition–achievement Relationships in Reading*, 21 COGNITIVE DEVELOPMENT 349 (2006); see also Kimberly G. Noble et al., *Brain-behavior Relationships in Reading Acquisition Are Modulated by Socioeconomic Factors*, 9 DEVELOPMENTAL SCIENCE 642 (2006).

<sup>40</sup> Hengyi Rao et al., *Early Parental Care Is Important for Hippocampal Maturation: Evidence from Brain Morphology in Humans*, 49 NEUROIMAGE 1144, 1144 (2010).

<sup>41</sup> Rajeev D.S. Raizada et al., *Socioeconomic Status Predicts Hemispheric Specialisation of the Left Inferior Frontal Gyrus in Young Children*, 40 NEUROIMAGE 1392, 1393 (2008).

<sup>42</sup> *Id.* at 1396. This pattern led the authors to speculate whether the scans revealed “a relationship between SES and language processing that purely behavioral tests are unable to reveal,” with the connection between SES and the observed laterality operating via “non-linguistic mechanisms.” The authors concluded, somewhat mysteriously, that the weaker language skills of low SES children “are related to reduced underlying neuronal specialization,” which is not necessarily revealed “by behavioural tests alone.”

group used MRI scans to investigate the association between SES and brain anatomy in a group of 23 healthy 10-year-old children with a wide range of parental SES. Their data revealed that language skills differed measurably by SES in the study sample. The authors also reported widespread SES-related differences in volume and surface area over a range of brain structures.<sup>43</sup>

Another cluster of research designed to examine the effects of social disadvantage on the brain makes use of the brain-activity recording, or ERP, technique described above. These studies are largely directed at documenting brain activity patterns associated with verbal abilities, especially oral language comprehension. As reported in one paper, D'Angiulli and colleagues examined ERP patterns in 28 children instructed to attend to auditory stimuli. The data indicated that localized ERP signals recorded in response to auditory stimuli consisting of stories and nonsense sounds played simultaneously in different ears were weaker for low SES than for high SES children. The authors interpreted these results as suggesting that less advantaged children are on average less able to tune out ambient distractions and remain attentive to meaningful verbal material, and speculated that this might help account for their relative deficiencies in reading skills and language comprehension.<sup>44</sup> Stevens and colleagues used a similar design of two-channel auditory stimuli to examine the ERP response pattern in 32 children aged 3 to 8.<sup>45</sup> In a refinement of D'Angiulli's report, their data

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<sup>43</sup> Katarzyna Jednoróg et al., *The Influence of Socioeconomic Status on Children's Brain Structure*, 7 PLOS ONE e42486 (2012). The authors reported an association between lower SES and "smaller volumes of gray matter in bilateral hippocampi, middle temporal gyri, left fusiform and right inferior occipito-temporal gyri" and "in anterior frontal region gyrification," which was "supportive of a potential developmental lag in lower SES children." They saw "no significant association between SES and white matter architecture."

<sup>44</sup> Amedeo D'Angiulli et al., *Towards a Cognitive Science of Social Inequality: Children's Attention-related ERPs and Salivary Cortisol Vary with their Socioeconomic Status*, Proceedings of the 30th Annual Conference of the Cognitive Science Society 211-16 (2008) ; Amedeo D'Angiulli et al., *Children's Event-related Potentials of Auditory Selective Attention Vary with Their Socioeconomic Status*, 22 NEUROPSYCHOLOGY 293 (2008).

<sup>45</sup> Courtney Stevens, Brittni Lauinger, & Helen Neville, *Differences in the Neural Mechanisms of Selective Attention in Children from Different Socioeconomic*



suggests that a weaker ability to filter out irrelevant auditory information was correlated with lower levels of maternal education, which was one aspect of disadvantage among those they measured. In a separate study on 25 mostly low-income children, the same authors were able to elicit a measurable change in the magnitude of lateral ERPs by training low income children to focus their attention. The effectiveness of the training, as evinced by a more normal pattern of ERP signals, correlated with improved language recognition and comprehension. That study made no attempt to measure or control for SES effects. The authors also conceded that their research, which lacked long-term follow up, was not designed to gauge “whether either the attention or receptive language gains persisted after the intervention ended.”<sup>46</sup>

### III. Methodological limitations

As the above summary indicates, the neuroscientific research designed to investigate the effects of early socioeconomic disadvantage on the brain comprises about two dozen actual research papers in the literature, most published within the past 10-15 years. These publications contain either retrospective analysis of evidence generated by others, or reports based on evidence gathered by the researchers themselves. This work consists mainly of imaging studies, supplemented by measures of event-related potentials, or ERPs. As noted, this literature builds on a large, ongoing body of work in behavioral and cognitive psychology, which documents and explores the psychological, emotional, and cognitive patterns associated with various forms of social deprivation, and draws on a broader background of research exploring the physiological, biological, and health-related correlates of adversity.<sup>47</sup> The research is summarized in, and

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*Backgrounds: An Event-related Brain Potential Study*, 12 DEVELOPMENTAL SCIENCE 634 (2009).

<sup>46</sup> Courtney Stevens et al., *Neural Mechanisms of Selective Auditory Attention Are Enhanced by Computerized Training: Electrophysiological Evidence from Language-impaired and Typically Developing Children*, 1205 BRAIN RESEARCH 55 (2008).

<sup>47</sup> See, inter alia, Bradley Brito et al., in NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, *FROM NEURONS TO NEIGHBORHOODS* (2000). According to the April 2016 policy statement released by the American Academy of Pediatrics and the Academic Pediatric Association on the deleterious effects of childhood poverty,

supplemented by, a host of review articles and journalistic reports that, although not containing original data, collate and discuss the findings in the neuroscientific literature and comment on their implications.<sup>48</sup>

The studies, as well as the reviews, reveal the neuroscience of deprivation to be a nascent field. Although heralded with much fanfare, the work to date has not generated a large, unified body of evidence. The anatomical observations are often crude and broad-brush, the sample sizes for original research small, the observations haphazard, and the associations frequently variable, weak, and of marginal statistical

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“[c]hild poverty . . . influences genomic function and brain development by exposure to toxic stress, a condition characterized by ‘excessive or prolonged activation of the physiologic stress response systems in the absence of the buffering protection afforded by stable, responsive relationships.’ Children living in poverty are at increased risk of difficulties with self-regulation and executive function, such as inattention, impulsivity, defiance, and poor peer relationships.” The statement and accompanying report also discusses a host of health-related ills associated with poverty, noting that “poverty has a profound effect on specific circumstances, such as birth weight, infant mortality, language development, chronic illness, environmental exposure, nutrition, and injury.” American Academy of Pediatrics, *Poverty and Child Health in the United States*, 137 PEDIATRICS 1 (2016).

<sup>48</sup> For review, see, e.g., Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 TRENDS IN COGNITIVE SCIENCES 65 (2009); Daniel A. Hackman, Martha J. Farah, & Michael J. Meaney, *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 NATURE REVIEWS NEUROSCIENCE 651 (2010); Martha J. Farah et al., *Childhood Poverty: Specific Associations with Neurocognitive Development*, 1110 BRAIN RESEARCH 166 (2006); Martha J. Farah, Kimberly Noble, & Hallam Hurt, *Poverty, Privilege, and Brain Development: Empirical Findings and Ethical Implications*, in NEUROETHICS: DEFINING THE ISSUES IN THEORY, PRACTICE, AND POLICY 277 (Judy Illes ed., 2006); NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS (2000). See also Sara Burr Johnson, Jenna L. Riis, & Kimberly G. Noble, *State of the Art Review: Poverty and the Developing Brain*, 137 PEDIATRICS E20153075 (2016); American Academy of Pediatrics, *Poverty and Child Health in the United States*, 137 PEDIATRICS 1 (2016); *Deepening Connections between Neuroscience and Public Policy to Understand Poverty*, FAST FOCUS No. 23-2016 (June 2016). For more popular presentations and summaries, see, e.g., Jim Dwyer, *How Poverty Keeps Hurting Young Minds*, NY TIMES, May 4, 2016, at A15; Daniel R. Taylor, *A Doctor’s Call for Action on Childhood Poverty*, PHILADELPHIA INQUIRER, Apr. 24, 2016, at G01 and other articles cited below.

significance. Overall, the impression is of a scientific endeavor that is just getting started and operating on a superficial level while struggling to define its methods and focus.

That state of affairs will likely continue for the indefinite future. Although the pace of the neuroscientific study of deprivation is intensifying, no dramatic leap forward should be expected anytime soon. A number of methodological obstacles stand in the way. First, human neuroscience currently relies heavily on neuro-imaging, and especially the recently refined technique of fMRI, to investigate both functional and morphological aspects of the brain. Although these techniques are work-intensive and require a high degree of subject cooperation, they are considered relatively harmless to targets of study. More intrusive alternatives --- that is, methods that require directly modifying the brain -- remain underdeveloped and are unlikely to emerge anytime soon. Invading and probing the human brain, or altering its function and structure directly through pharmacological, physical, or electrical manipulation, create the potential for considerable harm without any corresponding benefit to the subjects themselves. For this reason, research using invasive techniques is unlikely to attract the participation of volunteer subjects or to win the approval of those assigned to oversee participants' well-being. Research on the effects of poverty on the brain encounters additional limitations from the nature of the inquiry itself. Because the brain is believed to be most vulnerable to deprivation during development, many studies involve children. But generating many fMRI scans on children is an arduous and ethically fraught endeavor. Parents are understandably reluctant to consent to their children's participation, especially in longitudinal research requiring multiple scans over time. Studies must be approved by IRBs, or Institutional Review Boards, which are charged with insuring that the risks of harm are minimized and that benefits for subjects outweigh burdens. IRBs are especially protective of children, who cannot themselves give informed consent. Additional difficulties arise from the contours of study design. Many projects are directed at establishing a three-way correspondence -- among past or present deprivation, brain characteristics, and performance. For the purpose of visualizing

brains and measuring behavior, children do not always make cooperative subjects. When the experiment requires subjects to perform functional operations such as reading, listening, looking, or speaking, children may not act cooperatively, follow directions, or put forth a consistent effort. The measured results for assigned tasks can therefore be unpredictable, erratic, and difficult to standardize. Likewise, getting children to remain immobile in a scanner, or to perform while being scanned, is sometimes difficult.

Finally, an important variable in studies that seek to establish the effects of social deprivation is the past or present environment in which subjects live or are raised. The parameters or proxies for early disadvantage tend to be imprecise and ad hoc. Researchers use the terms “poverty,” “disadvantage,” “deprivation,” and “low SES” somewhat indiscriminately. There is no single standardized metric that holds sway in neuroscience or cognate fields. The potential dimensions of deprivation are variable, and the parameters actually measured in any given study are often driven by available information. These can include household income, home environment, parental education, job status, perceived status, neighborhood characteristics, “life stress,” and aspects of parental behavior, including neglect, abuse, nurturance, emotional support, disciplinary style, enrichment efforts (such as reading to children, or acquiring toys) or quality or quantity of verbal interaction. The methods for assessing these parameters vary, and include retrospective and subjective reports that are often imperfect and incomplete. Finally, children do not necessarily face a consistent or uniform environment while growing up. Childhood deprivation can last for varying periods and conditions of upbringing can change, sometimes drastically, over time. For these reasons, and others, many datasets in deprivation neuroscience are quirky, noisy, unstandardized, or incomplete, and are destined to remain so. These shortcomings reduce the reliability, reproducibility, and statistical significance of the results in the field.<sup>49</sup> The broader point is that the observations of deprivation

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<sup>49</sup> See, e.g., *Deepening Connections between Neuroscience and Public Policy to Understand Poverty*, FAST FOCUS No. 23-2016 (June 2016) (noting a lack of uniformity in measures of SES and disagreement among developmental neuroscientists on the dimensions that should be measured).

neuroscience can be no better than the behavioral measures with which they are correlated. If those measures are flawed, imprecise, or incomplete, then the brain studies will be unreliable and indeterminate as well. Such limitations are endemic to the field, and are not likely to be improved upon anytime soon.

IV. Genes v. Environment – external causation vs. selection effects

One notable feature of the neuroscience studies described above is that they document correlations only, and are not equipped to establish causation. Specifically, the research designs are not geared to sorting out how much of the changes or deficits observed in the brains of less privileged subjects can be attributed to the environmental conditions to which those brains have been exposed, as opposed to an innate or genetically programmed influence. In fact, as discussed more fully below, the problem of establishing causation is not just endemic to neuroscience. Rather, it bedevils much work in the developmental psychology of deprivation, and in behavioral and psychological investigations of all types.

The inability of research on deprivation, including neuroscience, to disentangle mechanisms of causation and attribute observed results to genetic versus environmental influence is central to the question of whether, and how, the science can meaningfully inform public policy. To summarize the main point: the effectiveness of particular strategies, programs, or interventions designed to reduce, or prevent, the effects of childhood deprivation – which are the main focus of policy discussions that draw on deprivation neuroscience -- will almost always depend critically on whether the social conditions these policies seek to alleviate are actually responsible for the adverse brain and behavioral effects attributed to them. If a deprived environment (as opposed to other factors) has a modest or negligible role in producing the deficits observed, then alleviating or removing the cause – by eliminating the deprivation or establishing a more normal or enriched environment -- cannot be expected to alleviate those deficits. To be sure, it can be objected that eliminating early adversity need not be the only way to

cure impairments that result from or are associated with deprived conditions. Traits or behaviors with a strong genetic basis are not perforce impervious to external alleviation or correction. (Often-repeated examples are the ability of eyeglasses – a purely “environmental” intervention – to correct the myopia that results from an inherited eye condition; or the effects of a strict diet, which can relieve many of the symptoms of the genetic condition of phenylketonuria or PKU).<sup>50</sup> However, as discussed and elaborated below, the literature on the practical payoff from deprivation neuroscience is geared almost exclusively to *preventative* strategies. Attempts to reverse the purported effects of poverty on brains and behavior after they have occurred have so far yielded negligible results. We simply have no idea how to reverse the impairments associated with disadvantage, and neither neuroscience nor behavioral science have taught us how to do so.

The discussion in this section so far assumes a simple conceptual dichotomy between external, or environmental, causes of the brain changes associated with deprivation versus innate, or genetic factors. In fact this dichotomy is rarely stark in practice: human behavior is known to be influenced by both mechanisms, with experience modifying the expression (or phenotype) of genetic traits (or genotype) to a variable extent through so-called genetic-environmental, or G x E interactions.<sup>51</sup> The phenotypical

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<sup>50</sup> See, e.g., Steven Pinker, *Why Nature & Nurture Won't Go Away*, DAEDALUS 5, 7 (Fall 2004) 7 (noting that a strict diet can relieve the symptoms and damage from PKU, but only partially).

<sup>51</sup> For lucid reviews of research on gene-environment interactions, and the influence of innate endowment versus environmental influence on human behavior, personality, and cognitive ability, see Stephen B. Manuck & Jeanne M. McCaffery, *Gene-Environment Interaction*, 65 ANNUAL REVIEW OF PSYCHOLOGY 41 (2014); Steven Pinker, *Why Nature & Nurture Won't Go Away*, DAEDALUS 5 (Fall 2004); STEVEN PINKER, THE BLANK SLATE (2002); Steven Pinker, *The Blank Slate* (draft Afterword to the 2016 Edition) (on file with author). For a summary of the pervasive role of genetic factors in human behavior, see, e.g., Robert Plomin et al., *Top Ten Replicated Findings from Behavioral Genetics*, 11 PERSPECTIVES ON PSYCHOLOGICAL SCIENCE 3 (2016). For specific examples, see, e.g., Avshalom Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 SCIENCE 851 (2002); J. Kim-Cohen et al., *MAOA, Maltreatment, and Gene-*

modifications of gene expression, which can range from transient to durable, affect human morphology, physiology, function, and behavior, and are responsible for functional and structural specialization between and within different body organs, including distinct brain centers, both during and after development. In any instance, the interaction is always one of degree: to what extent and under what conditions can environmental factors alter the expression of genes, and what are the limits on that expression? Answering those questions requires establishing the so-called “norm of reaction” – which is the full range of phenotypic (physical or behavioral) manifestations observed across the full spectrum of ambient conditions that an organism can encounter.

As applied to the study of social disadvantage, as with any other arena of human behavior, the interaction of environmental factors with genetic endowment is dauntingly complex. In fact, the contribution of genetic endowment to phenotypic variation is neither fixed nor necessarily linear. Rather, it is contingent on the environment, or range of environments, the organism encounters. Likewise, the influence of ambient factors can run the gamut from “quite a bit” to “hardly at all,” depending on the trait at issue, or the condition under which the trait is observed. Some environments suppress the expression of genetic variation, whereas others enhance genes’ influence and cause genetic distinctions to dominate. This does not mean that genes impose no limit on physical traits or behavior. Under some conditions, even if not all, genes can cause phenotypes to dramatically diverge. A simple example of this point is the height of plants, which is under strong genetic control. Plants with different alleles (or genetic sequences) that control height will grow to similar height in a dry environment. But genetically tall plants will tower over plants with “short” gene variants when provided with water and nutrients.<sup>52</sup> In the same vein, some

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*Environment Interaction Predicting Children’s Mental Health: New Evidence and a Meta-Analysis*, 11 MOLECULAR PSYCHIATRY 903 (2006).

<sup>52</sup> See, e.g., Steven Pinker, *Why Nature & Nurture Won’t Go Away*, DAEDALUS 5, 6 (Fall 2004) (Observing that “different strains of corn may grow to different heights when equally irrigated, but a plant from a taller strain might end up shorter if it is

researchers have claimed that deprivation enhances the environmental contribution to measured intelligence (thereby causing many poor children to fall short of their intellectual potential) because it suppresses the expression of genes for high intelligence. In contrast, environmental enrichment increases the contribution of genetic endowment to individual differences in intellectual performance by enabling genes for high intelligence to be fully expressed (thus allowing the privileged to achieve their “natural” limits). According to a recent study, a similar pattern appears to prevail with body weight.<sup>53</sup> The analysis of demographic data suggests that genetic propensities to obesity dominate individual outcomes in the current era, in which people have wide access to abundant, cheap, high calorie food, whereas in past decades, when food was less available and access more tightly controlled by conventional restraints, body weight was more uniform (and almost people thinner) because genetic differences were less often expressed.<sup>54</sup> Once again, as with intelligence, so with obesity: “enrichment” facilitates the expression, hence the dominance, of genetic differences, which produces a pattern of greater individual variation in observed traits.

The factors that determine intelligence and obesity are hotly contested, with no clear consensus on how genes and environment

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deprived of water.”) He notes from this and other examples that although “some genetic effects may be nullified in certain environments, not all of them are.” Specifically, he asserts that “studies that measure both genetic and environmental similarity (such as adoption designs where correlations with adoptive and biological parents can be compared) show numerous main effects of personality, intelligence, and behavior across a range of environmental variation.” Pinker also warns against inferring from exceptional extremes that “heredity imposes no constraints on behavior.” *Id.* at 6. Rather, “just because extreme environments can disrupt a trait does not mean that the ordinary range of environments will modulate that trait, nor does it mean that environment can explain the nature of the trait.” *Id.* at 7.

<sup>53</sup> Guang Guo et al., *The Genome-Wide Influence on Human BMI Depends on Physical Activity, Life Course, and Historical Period*, 52 *DEMOGRAPHY* 1651 (2015).

<sup>54</sup> The study also suggests that there are class and educational dimensions to the gene-environment interaction for body weight. Better educated individuals appear more able to control their weight regardless of genetic propensity. Thus, even in the current climate their body weight is more influenced by environmental factors, including eating and exercise habits. *Id.*



combine to produce observed patterns. This is not the place for a sophisticated review of the array of conceptual and methodological puzzles presented by these phenomena. Rather, these examples are provided to show that the relationship of genes to environment, and genotype to phenotype, is complex and unpredictable. That complexity applies as well to the potential role of environmental and genetic factors in producing the brain and behavioral disparities associated with SES.

As already noted, scientists have identified many behavioral traits that are more frequently observed in the lower SES population, including poor social skills, impulsivity, lack of foresight, low IQ, learning difficulties, distractibility, neuroticism, hyper-reactivity to stress, lack of resilience, and impersistence. It is possible to posit a causal story for each trait, attributing deficits to an array of external forces—ranging from material conditions to interactions with parents and the broader world -- that are brought to bear on deprived individuals as part of the environment to which they are exposed. Most straightforwardly, lack of material resources can operate directly on the child and its brain by interfering with the satisfaction of basic needs, such as for food, adequate shelter, or medical care. Or poverty can operate indirectly by influencing parental behavior, which may contribute in myriad ways to detrimental conditions of upbringing. Stressed parents are less likely to create positive or rich home environments, engage in stimulating activities, or otherwise generate opportunities that enhance children's well-being. Parental stress from lack of resources and material insecurity can compromise parenting skills, leading to indifference, neglect, and even abuse. Bad parenting and poor parent-child interactions in turn decrease children's chances of developing adequate verbal skills, high cognitive ability, and self-control. Deficits in these areas lead children to make bad choices that in turn increase their own risk of being poor. Variations on these insights are routinely hypothesized and discussed in the vast literature on early disadvantage and what to do about it.<sup>55</sup> But there is also a

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<sup>55</sup> See, e.g., NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS (2000) for a comprehensive review. See also, e.g., Council on Community Pediatrics, *Poverty and Child Health in the United States*,

large scientific literature that recognizes that innate, genetic factors can shape human behavior.<sup>56</sup> Many of the traits that show deficits among lower SES individuals have been shown to be heritable to varying degrees, and some to a significant extent.<sup>57</sup> These traits frequently influence social skills and interactions, including parenting practices, and tendencies that put people at risk for poverty may also compromise their ability to function as parents. This raises the possibility that the behavioral deficits observed in poor children may be generated through some combination of genetic and environmental mechanisms. Parents afflicted with traits associated with poverty may be at greater risk to engage in poor parenting, and to compromise the quality of the family environment in which children grow up. Likewise, poor children may inherit an enhanced risk of behavioral impairments directly from their parents through genetic transmission, or can develop those deficits from inadequate parenting influenced by similar parental genetic endowments, or can be shaped by some combination of both influences. But the important point is that external forces, including material deprivation, may not be the sole or even the dominant mechanisms by which poor parents produce poor children. Rather, adverse genetic traits may be transmitted to offspring directly, independently of inadequate parenting. These can influence the behaviors and deficits that impede children's ability to function and escape poverty.

A. James Thompson's critique of the Noble brain size study

The literature to date looking at the neuroscience of poverty does little or nothing to disentangle potential genetic and

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137 PEDIATRICS 1 (2016) (comprehensive statement of the American Academic of Pediatrics, containing an overview of presumed social and environmental causes of the detrimental effects of child poverty).

<sup>56</sup> See, e.g., Steven Pinker, *Why Nature & Nurture Won't Go Away*, DAEDALUS 5 (Fall 2004); Steven Pinker, *The Blank Slate* (draft Afterword to the 2016 Edition) (on file with author).

<sup>57</sup> See, e.g., Robert Plomin et al., *The Top Ten Replicated Findings from Behavioral Genetics*, 11 PERSPECTIVES IN PSYCHOLOGICAL SCIENCES 3 (2015) (providing a comprehensive list of personality and behavioral traits that have been shown to have a large heritable component, and reviewing the literature on behavioral genetic evidence).

environmental explanations for the observed brain and behavioral profiles of disadvantaged children. This signature shortcoming is laid bare by an illuminating online conversation between James Thompson, a noted British psychometrician, and Professor Kim Noble, a Columbia University developmental neuroscientist, on the topic of her co-authored 2015 paper in *Nature Neuroscience*, described above.<sup>58</sup> The Noble study found that family income and parental education were associated with brain size and surface area, with children deprived on some SES measures observed to have smaller brains. The thrust of James Thompson's critique of the Noble study, laid out in a widely read blog called "Psychological Comments," was directed at remarks by the authors of the study, in both the results and discussion sections of their paper, elaborating on the broader social significance of the reported findings. Although noting the authors' disclaimer that "it is unclear what is driving the links between SES and brain structure," Thompson objects to the papers' uncritical tone and lopsided emphasis on environmental explanatory factors, including the invocation of "ongoing disparities in postnatal experience or exposures, such as family stress, cognitive stimulation, environmental toxins or nutrition," and "corresponding differences in the prenatal environment."<sup>59</sup> The core of Thompson's

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<sup>58</sup> See Kimberly Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 *NATURE NEUROSCIENCE* 773 (2015); James Thompson, *Admixture in the Americas: European Intelligence*, *PSYCHOLOGICAL COMMENTS* (March 15, 2016), <http://www.unz.com/jthompson/admixture-in-americas-european/>.

<sup>59</sup> *Id.*, quoting Kimberly Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 *NATURE NEUROSCIENCE* 773 (2015). In support of his critique, Dr. Thompson quotes this paragraph from the paper's discussion section: "We found that parental education was linearly associated with children's total brain surface area, implying that any increase in parental education, whether an extra year of high school or college, was associated with a similar increase in surface area over the course of childhood and adolescence. Family income was logarithmically associated with surface area, implying that, for every dollar in increased income, the increase in children's brain surface area was proportionally greater at the lower end of the family income spectrum. Furthermore, surface area mediated links between income and children's performance on certain executive function tasks." Thompson reads these comments as implying "that an

objection is that the authors neglect the possible role of genetic variation as an explanation for the SES-related differences in brain size and cognitive and behavioral capacities they observe. States Thompson (in commenting on the discussion section of the paper): “You will note that inherited characteristics are not mentioned in this important section. Not a single word. It seems to have escaped notice that the apparent SES/brain link might both be driven by a common factor of inherited intelligence.”<sup>60</sup> Thompson’s complaint, in effect, is that the paper should have at least considered the possibility that the environmental factors measured in the study (education and family income) might not be the sole, or even the most important, cause of the observed SES-related morphological gradients. Rather, a genetic mechanism could be an important source of the observed differences. Adults with innate tendencies to smaller brains might be less smart, hence increasing their risk of poverty. Those adults would tend to pass on genes for reduced brain size (and the resulting reduced cognitive capacity) to their children, thus generating the observed correlation between parental poverty and smaller brains in children.

In fact, as Thompson concedes, the paper’s authors do not ignore genes altogether. Genetic factors figure in the authors’ decision to control for genetic group ancestry (as a proxy for race) in their analysis of the data. Although Noble and her co-authors are somewhat coy on the reasons for this choice, they do acknowledge that “brain morphology differs, at least subtly, among different ancestry groups.”<sup>61</sup> Based on this statement and the background literature on which it is based, Thompson infers that the paper introduced controls to deal with average racial group differences, observed in previous research and presumably also in Noble’s sample,

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extra year of education might increase the surface area of the brain,” and likewise that providing more material resources would be expected to have the same effect.

<sup>60</sup> James Thompson, *Admixture in the Americas: European Intelligence*, PSYCHOLOGICAL COMMENTS (March 15, 2016), <http://www.unz.com/jthompson/admixture-in-americas-european/>.

<sup>61</sup> Kimberly G. Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 NATURE NEUROSCIENCE 773 (2015), at 3, online version.

in brain surface area, size, and morphology. Even with group ancestry differences factored out, however, the authors still find that brain surface area varied systematically with some aspects SES. In commenting on this result, Noble et al states:

“[A]lthough the inclusion of genetic ancestry does not preclude the possibility that these findings [i.e., the results reported in their paper] may reflect, in part, an unmeasured heritable component, it reduces as far as possible the likelihood that apparent SES effects were mediated by genetic ancestry factors associated with SES in the population.”<sup>62</sup>

Thompson faults this assertion as misleading, because, although somewhat ambiguous, it strongly suggests that genes associated with ancestry (as a rough proxy for race) are the only ones that count in producing brain surface area differences in the study subjects. Ergo, any remaining surface area differences (that is, those correlating with socioeconomic status, independent of ancestry) must be traceable to environmental factors. As he explains, although “the paper has done well to include a genomic version of race,” that inclusion “does not cover the major factor of intelligence being heritable in all genetic groups.”<sup>63</sup> In other words, it “does not correct for the overall heritability of intelligence and the heritability of other characteristics like brain size in [the] study children.”<sup>64</sup> As Thompson summarizes, “the paper and the comments will lead readers to believe that lack of money is stunting the brains of poorer children. This is

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<sup>62</sup> *Id.* at 777. The authors add at another point that “brain morphology differs, at least subtly, among different ancestry groups. Thus it is often difficult to rule out the possibility that genetic ancestry mediates associations between SES and brain morphological differences.”

<sup>63</sup> See James Thompson, *Howitzer or Katyusha: Reply to Prof. Noble*, PSYCHOLOGICAL COMMENTS (April 19, 2015), <http://www.unz.com/jthompson/howitzer-or-katyusha-reply-to-prof-noble/>. Also, James Thompson, *Admixture in the Americas: European Intelligence*, PSYCHOLOGICAL COMMENTS (March 15, 2016), <http://www.unz.com/jthompson/admixture-in-americas-european/>.

<sup>64</sup> See James Thompson, *Howitzer or Katyusha: Reply to Prof. Noble*, PSYCHOLOGICAL COMMENTS (April 19, 2015), <http://www.unz.com/jthompson/howitzer-or-katyusha-reply-to-prof-noble/>.

possible, but not proved by this study because of obvious genetic confounders. The authors should have made [this] clearer.”<sup>65</sup>

Thompson concludes that the failure to give equal time to alternative genetic mechanisms of causation, net of specific markers of ancestry, represents a significant flaw in the authors’ presentation of their research. He is especially skeptical of any attempt to draw conclusions, or even to speculate, about broader implications for interventions or policies designed to address the problems associated with poverty. In this vein, he criticizes as unfounded the authors’ statement that, “by elucidating the structural brain differences associated with socioeconomic disparities, we may be better able to identify more precise . . . targets for intervention, with the ultimate goal of reducing socioeconomic disparities in development and achievement.”<sup>66</sup> Likewise, he questions the authors’ observation that “policies targeting families at the low end of the income distribution may be most likely to lead to observable differences in children’s brain and cognitive development”<sup>67</sup> and disparages the assertion that “many leading social scientists and neuroscientists believe that policies reducing family poverty may have meaningful effects on children’s brain functioning and cognitive development.”<sup>68</sup>

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<sup>65</sup> *Id.* Steven Pinker makes a similar point in his succinct critique of the same Noble paper: “Another study found that the surface area of children’s brains correlates with family income, and concluded that ‘wider access to resources likely afforded by the more affluent may lead to differences in a child’s brain structure’ – never entertaining the possibility (in fact well-supported by behavioral genetics) that children might inherit genes that made their parents bigger-brained, hence smarter, hence richer.” Steven Pinker, *The Blank Slate* (draft Afterword to the 2016 Edition) (on file with author). Pinker likened Noble’s inferences to the dubious conclusion, from the fact that “detached and neglectful parents had more maladjusted children” that “of course . . . ‘parenting matters.’” Steven Pinker, *The Blank Slate* (draft Afterword to the 2016 Edition) (on file with author).

<sup>66</sup> Kimberly G. Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 *NATURE NEUROSCIENCE* 773, 778 (2015).

<sup>67</sup> *Id.*

<sup>68</sup> *Id.* at 775. In a similar vein, the authors of the paper state in the results section that “[i]t is possible that, in these regions, associations between parent education and children’s brain surface area may be mediated by the ability of more highly educated parents to earn higher incomes, thereby having the ability to purchase

Thompson argument, it seems, is that nothing in Noble's research establishes that observed SES differences in brain morphology are actually caused by environmental factors. Therefore, no conclusions can be drawn about the effectiveness of manipulating the environment to reduce poverty.<sup>69</sup> His reasoning appears to be that, if actual deprivation, neglect, and lack of resources are the only or main causes of smaller brains, then relieving those conditions should result in larger brains and the superior capacities associated with them. But if those conditions are not important – if size is pre-programmed in the brain – then preventing adverse environmental conditions cannot be assumed to improve outcomes. Speculation about whether particular policies or interventions can reduce the effects of poverty is thus unwarranted.

#### B. Genes and environment in deprivation neuroscience

The Thompson blog commentary on the Noble study is instructive in laying bare the ambiguities inherent in the deprivation neuroscience literature, the formidable difficulties of disentangling internal from external mechanisms, and the choice by the papers' authors, unjustified in Thompson's view, to spotlight environmental causes to the detriment of the possibility of genetic transmission. However, Thompson's comment considers only one paper. A key question is whether the Noble study's handling of the issue of genetic vs. environmental interactions is emblematic of the approach adopted in this area of neuroscience generally. A survey of the field so far suggests that, although researchers in neuroscience and other cognate areas have not entirely ignored the existence of innate influences or so-called "selection effects," the dominance of environmental factors and ambient conditions in producing observed brain and behavioral

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more nutritious foods, provide more cognitively stimulating home learning environments, and afford higher quality child care settings or safer neighborhoods, with more opportunities for physical activity and less exposure to environmental pollutants and toxic stress . . . . It will be important in the future to disambiguate these proximal processes by measuring home, family and other environmental mediators."

<sup>69</sup> See more *supra* and *infra* on the relationship of causation to intervention.

problems is generally assumed.<sup>70</sup> Some researchers neglect even to acknowledge a possible role for innate influences<sup>71</sup> whereas others mention them only in passing or with brief, boilerplate disclaimers. So, for example, in a paper on the association between SES and performance on memory tasks, the authors state, without further elaboration or citation, that the effects observed “may be due to the heritability of acquired memory ability across SES.”<sup>72</sup> Likewise, in reviewing the anatomical correlates of reading ability disparities across social class, another group of authors notes, without further comment or documentation, that “[t]he possibility exists that the SES variable in this study was [partly] a measure of interactive genetic and environmental factors . . . including parents’ genetically limited linguistic capacity.”<sup>73</sup> Notwithstanding these brief acknowledgments, genetic transmission is rarely emphasized or discussed in any detail. The extensive literature on behavioral genetics and the heritability of particular traits and skills is given cursory treatment or, most often, simply ignored. James Thompson sizes up the situation:<sup>74</sup>

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<sup>70</sup> See, e.g., NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS 286 (2000); American Academy of Pediatrics, *Poverty and Child Health in the United States*, 137 PEDIATRICS 1 (2016).

<sup>71</sup> See, e.g., Courtney Stevens et al., *Neural Mechanisms of Selective Auditory Attention Are Enhanced by Computerized Training: Electrophysiological Evidence from Language-impaired and Typically Developing Children*, 1205 BRAIN RESEARCH 55 (2008); Courtney Stevens, Brittni Lauinger, & Helen Neville, *Differences in the Neural Mechanisms of Selective Attention in Children from Different Socioeconomic Backgrounds: An Event-related Brain Potential Study*, 12 DEVELOPMENTAL SCIENCE 634 (2009); Rajeev D.S. Raizada et al., *Socioeconomic Status Predicts Hemispheric Specialisation of the Left Inferior Frontal Gyrus in Young Children*, 40 NEUROIMAGE 1392 (2008).

<sup>72</sup> Douglas Herrman & Marry Ann Guadagno, *Memory Performance and Socio-Economic Status*, 11 APPLIED COGNITIVE PSYCHOLOGY 113 (1997).

<sup>73</sup> Mark A. Eckert, Linda J. Lombardino, & Christiana M. Leonard, *Planar Asymmetry Tips the Phonological Playground and Environment Raises the Bar*, 72 CHILD DEVELOPMENT 988 (2001).

<sup>74</sup> James Thompson, *Howitzer or Katyusha: Reply to Prof. Noble*, PSYCHOLOGICAL COMMENTS (April 19, 2015), <http://www.unz.com/jthompson/howitzer-or-katyusha-reply-to-prof-noble/>.



“As opposed to the genetics plus environment position, the dominant position in much of contemporary psychology seems to be the sociological argument, which gives precedence to social class, income, wealth and power. The argument goes thus: class strongly influences living circumstances; those living circumstances determine most social outcomes; class casts some people into poverty, poverty stunts intellectual development, lower intelligence is a downstream effect of class-based poverty, so the best way of dealing with low ability is to increase income.”

The secondary literature that seeks to synthesize and draw out the implications of the neuroscience shows a similar tendency to focus on social circumstances. Although the methodological limitations of the studies warrant equipoise on causation, that neutrality is rarely evident. Rather, the tilt towards environment, and the downplaying of genetic explanations, is unmistakable. So, for example, a review of the deprivation neuroscience findings to date, although acknowledging that genetic predispositions vary for the traits under consideration, nonetheless chooses to emphasize that “cognitive performance is modified by epigenetic mechanisms indicating that experience has a strong influence on gene expression and resultant phenotypical cognitive traits.”<sup>75</sup> The authors provide no

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<sup>75</sup> Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 *TRENDS IN COGNITIVE SCIENCES* 65, 69 (2009). The use of the term “epigenetics” here is potentially misleading, as it promises new insights but in fact adds nothing to the old understanding that ambient conditions modify gene expression. According to Steven Pinker, in explaining the recent popularity of that term, “the yearning for some biological phenomenon that promises liberation from the seemingly fatalistic constraints of evolution and genetics is perennial” making it “inevitable that some new research topic would be seized upon as the longed for release. That new topic is ‘epigenetics.’” . As Pinker points out, that release is not forthcoming, because epigenetics simply refers to the garden variety environmental contributions to phenotype (observed traits or behaviors) that are produced by various environmental influences brought to bear on genetic variants. In other words, epigenetics is the study of how environment influences the somatic expression of genes. According to Pinker, “for as long as we’ve known that every cell in the body contains a complete copy of the genome, we’ve known that genes must be turned on and off in response to signals from outside the cell.” Recent

citations or support for their statement that the influence of “experience” – including, presumably, SES -- on the traits in question is generally “strong,” – as opposed to modest or weak.<sup>76</sup> Nor do they analyze particular attributes, or the evidence on genetic versus environmental contributions to the expression of those attributes, on a case by case basis.

Similarly, in reviewing deprivation neuroscience research, James Ryan acknowledges that genes and environment likely contribute to learning disabilities and intellectual ability generally, and notes that there “may never be” an answer to the question of “whether and how poverty impacts cognitive development.”<sup>77</sup> He nonetheless proceeds to make extensive recommendations for

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research has added considerable detail to our knowledge of how these influences actually operate, including the specific modifications in DNA structure and expression that control whether particular genes are activated. “Yet may people react to the uncovering of mechanisms of gene regulations as if it were a revolutionary discovery that calls for a rethinking of nature and nurture.” Steven Pinker, *The Blank Slate* (draft Afterword to the 2016 Edition) (on file with author). (page number ---) According to Pinker, no such rethinking is in order. Specifically, epigenetic mechanisms are not equivalent to the inheritance of acquired characteristics, or so called “Lamarckian” inheritance. With only very rare exceptions, environment cannot alter the genome of either somatic or germ cells directly. Rather, it influences somatic phenotype, which is the individual organism’s *expression* of a fixed genomic endowment. *Id.* But genes and environment have long been known to interact in this way.

<sup>76</sup> See also Hengyi Rao et al., *Early Parental Care Is Important for Hippocampal Maturation: Evidence from Brain Morphology in Humans*, 49 *NEUROIMAGE* 1144 (2010). (Mentioning genes as a factor in the observed correlations between maternal nurturance and hippocampal maturation, but suggesting that animal cross-fostering studies support relatively weak genetic influences compared to environmental factors).

<sup>77</sup> See James E. Ryan, *Poverty as Disability and the Future of Special Education Law*, 101 *THE GEORGETOWN LAW JOURNAL* 1455, 1489 (2013). See also, e.g., Jennifer H. Suor et al., *Tracing Differential Pathways of Risk: Associations Among Family Adversity, Cortisol, and Cognitive Functioning in Childhood*, 86 *CHILD DEVELOPMENT* 1142 (2015); M. M. Sweitzer et al., *Polymorphic Variation in the Dopamine D4 Receptor Predicts Delay Discounting as a Function of Childhood Socioeconomic Status: Evidence for Differential Susceptibility*, 8 *SOCIAL COGNITIVE AND AFFECTIVE NEUROSCIENCE* 499 (2012) (citing literature suggesting a heritable component to behavioral discount rates).

reforming the legal treatment of learning-disabled children, on the assumption that relieving poverty will alleviate the cognitive and learning deficits associated with poverty. But that assumption may not be warranted if the difficulties low income children experience in the classroom are not chiefly the result of their poverty and its accompanying disadvantages.<sup>78</sup> Yet another comprehensive review, although acknowledging a range of opinions and estimates on the relative contribution of genetic endowment versus environment to “the variability of ability and personality found in the population,”<sup>79</sup> and noting that some psychometricians believe “that the developmental consequences of persistent family environmental influences, such as socioeconomic status and parenting, are remarkably small,”<sup>80</sup> nonetheless puts forth recommendations for a host of new policies and comprehensive, expensive interventions designed to alleviate the detrimental effects of low SES. The relevance of genetic causes or selection effects to the potential efficacy of this list of recommendations is not addressed.<sup>81</sup>

This section has thus far emphasized what researchers and commentators say about the implications of their work. The tendency to stress environmental factors, often to the exclusion of others, is understandable. Broad public interest in the studies, and especially the willingness to provide public funding to support expensive research, often depends on identifying concrete payoffs in the form of interventions, policies, and practical programs.

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<sup>78</sup> For more extensive discussion of the Ryan paper, *see infra*.

<sup>79</sup> *Id.*

<sup>80</sup> NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS 286 (2000), discussing the “parents don’t matter” school of behavioral genetics.

<sup>81</sup> *Id.* at chapter 13 (“Promoting Healthy Development Through Intervention,” and chapter 14 “Conclusions and Recommendations”). *See also, e.g.*, American Academy of Pediatrics, *Poverty and Child Health in the United States*, 137 PEDIATRICS 1 (2016) statement and recommendations, discussed in more detail *infra*.

As the analysis above of the Kim Noble brain size study reveals, however, what deprivation neuroscience researchers say about their work is often seriously out of sync what their studies actually show. The methods and results, not the discussion section, are the heart of any scientific work. Although sometimes acknowledging causal uncertainty, the studies considered here do little or nothing to resolve that uncertainty. They generally make no effort to parse out environmental versus genetic factor or to assign a precise role to each. In fact, they are not designed to do that. The main reason, as the researchers themselves sometimes acknowledge, is that sorting out these variables presents formidable methodological challenges that the field is simply not equipped to meet. To the extent that researchers are working on separating environmental and genetic causes of human behavior, the most promising routes do not lie within the ambit of deprivation neuroscience as currently practiced, which is focused on establishing simple correlations among brain morphology, brain function, social background, and behavioral traits. Rather, as elaborated below, the techniques of behavioral genetics offer the most promise for disentangling the influence of genes and environment, and exploring their interaction. The cumbersome behavioral methods of comparing twins and siblings, which have been in use for decades, have recently been supplemented by sophisticated techniques of genome-wide DNA analysis. These new methods have been made possible by dramatic advances in DNA sequencing and the accumulation of genomic data from large human populations.

The limited potential of deprivation studies to disentangle the role of nature and nurture can be demonstrated by considering how the experimental design of this research could be altered or supplemented to better illuminate causation. Emblematic of the difficulties of using standard neuroscience techniques for this purpose is Kim Noble's response to James Thompson's suggestion, as part of his critique of her 2015 study as detailed above, that measuring the intelligence and/or brain morphology of the mothers of children in the study might help to gauge the possible genetic contribution to the SES-related brain surface gradient her group observed in the children.

(No data on mothers, or parents, was collected). Noble observes that even though the genetic endowments of parents and children may be correlated, “so are parent-child environments.” In other words, the mothers’ measured IQ does not simply reflect her genetic endowment, but also her upbringing. And the mothers of poor children were more likely to grow up poor themselves. Thus, even if the researchers saw an association between maternal IQ and children’s brain size and/or cognitive functioning, that would still not enable them to make a definitive determination as to whether the children’s brain size and corresponding brain functioning were mostly controlled by genes or environment. Likewise, Noble notes, “scanning the parents would not solve the problem either, as parental brain morphometry would be both genetically and environmentally influenced as well.”<sup>82</sup>

Noble is correct that the additional data on mothers, although potentially helpful, is at best an imperfect method for disentangling the genetic versus environmental determinants of the brain size and morphology disparities that are the focus of her study. But that limitation applies to the specific study at hand, which uses techniques of brain scanning and behavioral testing on a relatively small number of subjects. In fact, behavioral geneticists have used a variety of behavioral methods, devised and refined over decades, for teasing out genetic and environmental components in various contexts and for a wide variety of traits.<sup>83</sup> The classic “gold standard” approach is to collect data on monozygotic twins who are separated early in life and adopted into different families.<sup>84</sup> Valuable evidence can also be

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<sup>82</sup> Kimberly Noble, *Income, Brain, Race: Prof Kimberly Noble Replies*, PSYCHOLOGICAL COMMENTS (Apr. 17, 2015), <http://www.unz.com/jthompson/income-brain-race-prof-kimberly-noble/>.

<sup>83</sup> See, e.g., references cited in note –supra.

<sup>84</sup> For twin studies and anti-social behavior, see, e.g., Soo Hyun Rhee & Irwin D. Waldman, *Genetic and Environmental Influences on Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies*, 128 PSYCHOLOGICAL BULLETIN 490 (2002). See also MICHAEL C. NEALE & LON R. CARDON, *METHODOLOGIES FOR GENETIC STUDIES OF TWINS AND FAMILIES* (1992); JOHN C. LOEHLIN & ROBERT C. NICHOLS, *HEREDITY, ENVIRONMENT, AND PERSONALITY: A STUDY OF 850 SETS OF TWINS* (1976). See also Steven Pinker, *Why Nature & Nurture Won't Go Away*, DAEDALUS 5, 10 (Fall 2004), and note 24 (noting that, through studies of twins and

obtained by tracking siblings separated at birth, and comparing them statistically to separated identical twins and unrelated individuals.<sup>85</sup> But natural adoption experiments and analyses of sibling data have always offered only limited promise due to pitfalls inherent in their retrospective design. These studies are plagued by erratic record keeping, imperfect datasets, omitted variables, small numbers of sample points (now reduced even further by the decline of of single mothers choosing to give up their children for adoption), and the restrictions in the SES range of adoptive parents (few of whom are poor).

Lately, an explosion of interest in behavioral genetics has been driven by rapid developments in genome-wide DNA sequencing. By generating large data bases and libraries of human DNA profiles, scientists have been able to accumulate information on human variation over numerous individuals from disparate populations. This evidence has formed the basis for genome-wide frequency analysis of genes and alleles, which are in turn statistically linked to anatomical and behavioral characteristics observed in these populations. So called “genome-wide association studies” enable scientists to pinpoint genetic sequences associated with particular traits, and to infer the profile of genetic influences on those traits.<sup>86</sup>

As already noted, the interaction of genetics and environment has implications for the practical payoff of policies and interventions

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adoptees, behavioral geneticists have “discovered that in fact virtually all behavioral traits are partly (although never completely) heritable,” and citing references.)

<sup>85</sup> For a description, *see, e.g.*, Robert Plomin, *Behavioral Genetic Methods*, 54 JOURNAL OF PERSONALITY 226 (2006).

<sup>86</sup> For a description of genome-wide association (GWA) studies, *see, e.g.*, Austin L. Hughes, *Me, My Genome, and 23andMe*, 40 THE NEW ATLANTIS 6 (2013); Dana B Hancock et al., *Meta-analyses of Genome-wide Association Studies Identify Multiple Loci Associated with Pulmonary Function*, 42 NATURE GENETICS 45 (2010); Teri A. Manolio, *Genomewide Association Studies and Assessment of the Risk of Disease*, 363 NEW ENGLAND JOURNAL OF MEDICINE 166 (2010); Luciano, M., Wright, M., Duffy, D. et al., *Genome Wide Scan of IQ Finds Significant Linkage to a Quantitative Trait Locus on 2Q*, *Behav Genet* (2006) 36: 45; [Cornelius A. Rietveld](#) et al., *Common genetic variants associated with cognitive performance identified using the proxy-phenotype method*, PNAS, September 23, 2014, vol. 111 no. 38.

designed to prevent or alleviate the purported effects of deprivation, which are the focus of the social implications of deprivation neuroscience.<sup>87</sup> And the efficacy of any particular measure will be a function of whether the deficits sought to be prevented are due mostly to poverty or represent the expression of genetic propensities more common among the poor. Apart from the genomic analytic techniques already detailed, another way to bypass the nature-nurture conundrum is to go directly to the practical question of whether specific proposed external interventions can alleviate observed deficits. The gold standard approach to establishing the causal efficacy of interventions (thereby separating external versus genetic causation) is to conduct controlled prospective clinical trials. In fact, in her dialogue with James Thompson, Kim Noble suggests as much by stating that “the bottom line is that, to truly establish the direction of causality, we need a random experiment.”<sup>88</sup> She announces plans to conduct a study in which “a sample of low-income mothers will be randomized upon the birth of their child to receive a large or small monthly income supplement.”<sup>89</sup> The families would then be followed longitudinally “to estimate the causal impact of an unconditional cash transfer on children’s cognitive, emotional and brain development.” This design “will provide definitive evidence on the extent to which young children’s cognitive and brain development is affected by poverty reduction.”<sup>90</sup> If the children of poor families receiving cash transfers show significantly larger brains and better cognitive functioning than SES-matched controls who don’t receive similar benefits, that result would strongly suggest that lack of money is responsible for the adverse effects.

This plan is promising as far as it goes. The problem is that it does not go very far. Prospective efficacy trials are not an all-purpose solution to the problem of selection effects because the potential for randomized controlled trials to illuminate the causes and cures for

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<sup>87</sup> See *supra* and *infra*.

<sup>88</sup> Kimberly Noble, *Income, Brain, Race: Prof Kimberly Noble Replies*, PSYCHOLOGICAL COMMENTS (Apr. 17, 2015), <http://www.unz.com/jthompson/income-brain-race-prof-kimberly-noble/>.

<sup>89</sup> *Id.*

<sup>90</sup> *Id.*

SES-related deficits is strictly limited. Trials are a feasible approach when interventions are straightforward, easily manipulated, and fully within researchers' control (such as handing families extra money). At most, simple interventions (such as the income supplement proposed by Kim Noble) may offer the potential to answer some discrete policy questions.

Unfortunately, many aspects of low SES are not amenable to simple interventions. The contributors to disadvantage are complex, poorly understood, and difficult, unethical, or impossible to manipulate. Children can't be assigned to different families, home environments, cultures, neighborhoods, or parents. Given these realities, researchers will never be able to answer most of the crucial questions, including whether and to what extent parenting style directly causes, or is merely associated with, traits found more frequently in lower SES populations. And if parental behaviors and parenting styles that actually affect children's outcomes have a strong heritable component, they will prove at least partly, and possibly very, resistant to researchers' efforts to modify them.

For all these reasons, prospective controlled trials have limited potential to illuminate effective interventions. In the absence of such trials, researchers are thrown back on classic twin/sibling studies or statistical techniques applied to existing datasets on brains, SES, and behavior. As noted, statistical analyses of descriptive data for the purpose of teasing out causal factors are always subject to imperfections, including undetected or omitted variables in the scientific and behavioral evidence, yielding results that are ambiguous or simply unrevealing. Although genome-wide analysis, as facilitated by advances in DNA sequencing technology and the accumulation of databases on multiple populations, offers great promise for identifying the genetic basis of common human traits and variations, that promise lies largely in the future. The main point, though, is that the studies that will end up illuminating the sources of behavioral and biological variation correlated with SES represent an extension of the large and growing body of work generated by behavioral geneticists, which stands outside the ambit of deprivation neuroscience as currently practiced.



## V. Practical Implications

As already noted, the relative contribution of innate versus environmental factors in generating any behavioral trait or deficit associated with a deprived upbringing has important implications for public policies and interventions designed to help the disadvantaged. Kim Noble and her colleagues, in the 2015 study discussed above, imply at several points that poverty is responsible for reducing brain size, which in turn results in some types of cognitive and emotional underperformance in lower SES children. But an alternative mechanism is that poor parents are, on average, genetically endowed with smaller brains; those genes are passed on to offspring (poor children), whose brains also tend to be reduced in size. As the Thompson critique suggests, a significant role for genetic transmission could place measurable limitations on the influence of external factors like poverty. But this in turn may have consequences for the success of efforts to enhance children's brain size and cognitive function by improving children's circumstances.

To be sure, the existence of a heritable component does not rule out alleviation through improved environmental conditions. Because genetic tendencies and environment interact, improvement or enrichment might help. But until the degree of genetic influence, and indeed the full range of reactivity to environmental inputs over the relevant population, is actually elucidated, the effects of any measures designed to alleviate disadvantage cannot be predicted. If it turns out that poverty and its attendant conditions are not a major or important cause of particular deficits – if the deficits are simply correlated with those social circumstances – then preventing poverty and reducing disadvantage will not do much to alleviate the associated behavioral difficulties. That is, policies that are designed to prevent or improve on developmental conditions may have a modest or negligible effect. In fact, as noted, the vast majority of suggestions and recommendations that make reference to deprivation neuroscience are geared towards early intervention, prevention, and alleviation of the circumstances that supposedly cause harm in the first place. On that score, the state of the science, and its inability to identify causal

mechanisms, counsels a non-committal, or at least extremely cautious, stance towards any practical recommendations that might be gleaned from research on the brains of disadvantaged individuals.

A similar caution is in order with respect to measures to *cure* the effects of poverty once they occur. *Regardless* of the causal mechanism, neuroscience as now conducted is virtually useless in identifying effective interventions after the fact. That is because, even if the research could establish that poverty compromises the brain, and even if the specific neurochemical mechanisms of that compromise were elucidated, those insights do not necessarily point the way to a cure – that is, to the specific measures that will *reverse* the cognitive and behavioral effects of a deprived upbringing once they occur.<sup>91</sup> To be sure, fixing damaged brains appears to implicate neuroscientific knowledge. For example, it would be at least theoretically possible to develop a pill to rebuild lost synapses, enlarge the number of functioning brain cells, or multiply or re-establish connections damaged by early deprivation. But the deprivation neuroscience now being conducted, and neuroscience in general, offers no realistic promise for such developments. Undoing the damage to deprived brains is not even remotely on the horizon, and the possibility of discovering how to do this is pure speculation. Additionally, even if the actual neurophysiological or neurochemical mechanisms could eventually be worked out in sufficient detail, behavioral studies are nonetheless an indispensable step towards discovering and describing those mechanisms, because only behavioral studies can reveal the real-world effects of any particular intervention.. Whether some kind of direct manipulation of the brain reverses the damage done by environmental factors must be established empirically on a case by case basis, using trial and error. There is no substitute for this case by case approach to connecting external interventions to the kinds of behavioral improvements that policy-makers are seeking.

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<sup>91</sup> See AMY L. WAX, RACE, WRONGS, AND REMEDIES (2009) (discussing the myth of reverse causation).

In the absence of techniques to manipulate brains directly, we are thrown back on trying to influence the ambient social circumstances that produce or perpetuate disadvantage, or to bring about the conditions that can cure its negative effects. Unfortunately, even if brain science can help identify the social conditions that are harmful or beneficial to the brain, it can tell us nothing about how to produce those circumstances. Thus, even apart from the difficulties of teasing out genetic vs. environmental causation, it is doubtful that neuroscience can help inform policies towards the disadvantaged and shape the interventions undertaken to assist them. When it comes to identifying measures to alleviate poverty and its effects, the study of the brain's structure, function, and chemistry offers no insights over and above those gleaned from behavioral studies and the generic understanding, long predating recent neuroscientific work, that distinct behaviors correspond to distinct brain states.<sup>92</sup>

It is not surprising, then, that the reviews and articles that claim to offer recommendations that come out of, and are informed by, neuroscientific research (and deprivation neuroscience in particular) suffer from a signal defect. They fail to explain how knowledge of the brain's structure and function points uniquely, over and above cognitive and behavioral observations, to particular programs or interventions. And they are vague on how specific programmatic recommendations follow from the content of neuroscientific discoveries. All told, the literature shows that neuroscience is of no help in formulating effective policies to address poverty and its effects. The same recommendations could be – and have been -- made without the benefit of neuroscience. And the policy agenda that commentators purport to derive from work in deprivation neuroscience is entirely non-specific. The most favored policies, which usually involve governmentally funded and coordinated services and programs, are no more consistent with, or compelled by, existing studies than an alternative, more traditional, approach to the social problem of poverty.

A. Addressing poverty and its effects –

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<sup>92</sup> See *supra* (intro); also *infra*.

Illustrating this point requires a review of claims made on behalf of deprivation neuroscience. What do papers reporting and commenting on the research say about the pragmatic implications of this work? As documented above, while not denying genetic inputs altogether, the neuroscience reports themselves tend to emphasize the environmental over the innate. And the secondary literature that seeks to glean recommendations from the science tilts towards assuming the dominance of environmental factors and, thus, finds a broad ambit for programmatic interventions.

The basic neuroscience literature itself, in contrast with commentaries from related fields or journalistic reports, tends to be somewhat circumspect about the implications of the research for policy. Most primary research papers refrain from identifying specific payoffs, and stick to open-ended predictions, isolated observations, and vaguely expressed expectations. Not atypical is the statement in one article that “[g]reater knowledge about the developmental timing of risk exposures and brain development would be extremely valuable for informing interventions.”<sup>93</sup> One research study expresses the hope (as yet unrealized) of discovering an effective “pharmacological intervention,”<sup>94</sup> while another offers a generic reassurance that none of the findings reported imply immutability, or resistance to effective correctives.<sup>95</sup> Yet another

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<sup>93</sup> Pilyoung Kim et al., *Effects of Childhood Poverty and Chronic Stress on Emotion Regulatory Brain Function in Adulthood*, 110 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES 18442 (2013).

<sup>94</sup> See Stephanie H.M. van Goozen, Graeme Fairchild, & Gordon T. Harold, *The Role of Neurobiological Deficits in Childhood Antisocial Behavior*, 17 CURRENT DIRECTIONS IN PSYCHOLOGICAL SCIENCE 224 (2008).

<sup>95</sup> Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 TRENDS IN COGNITIVE SCIENCES 65 (2009); see also Gwendolyn M. Lawson et al., *Associations between Children's Socioeconomic Status and Prefrontal Cortical Thickness*, 16 DEVELOPMENTAL SCIENCE 641, 649 (2013) (noting that the prefrontal cortical thickness disparities by SES observed in the study “[n]eed not be innate and unchangeable.”); see also Laurence Steinberg, *Should the Science of Adolescent Brain Development Inform Public Policy?*, Article Adapted from the Annual Henry and Bryna David Lecture, Delivered at the National Academy of Sciences (Nov. 2011) (Seeking to dispel the notion “that

report touts programmatic efforts designed to “enhance cognitive stimulation,”<sup>96</sup> whereas two others advance the general assertion that developmental neuroscience holds promise for redesigning public and educational programs for the disadvantaged.<sup>97</sup> Farah and colleagues, in a review of the ethical and policy implications of the data, acknowledge the difficulties of “disentangling cause and effect,” but state that “[t]he issue of whether and to what extent SES differences cause neurocognitive differences or vice versa should not be confused with the issue of whether we have an obligation to help children of any background become educated productive citizens.”<sup>98</sup> Another paper reviewing research and future directions suggests that neuroscientific findings so far have “opened new avenues for innovation in the design of interventions” to address the brain effects of childhood poverty.<sup>99</sup> In sum, a general endorsement of active intervention to help disadvantaged children is a commonplace in these articles. What is notably lacking are specifics on which interventions would be helpful, and how neuroscience, *qua* neuroscience, proves their effectiveness

The papers that get into more detail point to a familiar litany of proposals that have long been popular staples of the broader policy literature on child development, and that almost always predate the

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differences in brain structure/function means behaviors are ‘hard wired’ or cannot be changed”).

<sup>96</sup> Daniel A. Hackman, Martha J. Farah, & Michael J. Meaney, *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 NATURE REVIEWS NEUROSCIENCE 651 (2010); Helen Neville et al., *Neurocognitive Consequences of Socioeconomic Disparities*, 16 DEVELOPMENTAL SCIENCE 708 (2013) .

<sup>97</sup> Bruce S. McEwen & Peter J. Gianaros, *Central Role of the Brain in Stress and Adaptation: Links to Socioeconomic Status, Health, and Disease*, 1186 ANNALS OF THE NEW YORK ACADEMY OF SCIENCES 190 (2010); Allyson P. Mackey et al., *Neuroanatomical Correlates of the Income-Achievement Gap*, 26 PSYCHOLOGICAL SCIENCE 925 (2015).

<sup>98</sup> Martha J. Farah, Kimberly Noble, & Hallam Hurt, *Poverty, Privilege, and Brain Development: Empirical Findings and Ethical Implications*, in NEUROETHICS: DEFINING THE ISSUES IN THEORY, PRACTICE, AND POLICY 277 (Judy Illes ed., 2006).

<sup>99</sup> Sebastião J. Lipina & M. Soledad Segretin, *Strengths and Weakness of Neuroscientific Investigations of Childhood Poverty: Future Directions*, 9 FRONTIERS IN HUMAN NEUROSCIENCE 9 (2015) at 3.

neuroscience research in question.. One study looking at the effects of early deprivation on the structure of the hippocampus asserts that “[t]he finding that the effects of poverty on hippocampal development are mediated through caregiving and stressful life events further underscores the importance of high quality early childhood caregiving.” The authors go on to recommend parenting education and preschool programs “that provide high quality supplementary caregiving and safe haven to vulnerable young children.”<sup>100</sup> Another group touts a cluster of small, intensive, much studied early childhood programs, initiated in the 1970s (the Perry preschool program, the Chicago child-parent study, and the Abecedarian project)<sup>101</sup> that, in long term follow-up, have produced positive, albeit modest, improvements in rates of employment, criminal offending, and marriage, but not cognitive ability.<sup>102</sup> Other proposals include visiting nurse programs, Moving to Opportunity housing vouchers, intensive cognitive training,<sup>103</sup> and cash transfers to single parents.<sup>104</sup> Perhaps the most specific recommendation comes from the observation by Stevens and her colleagues that underprivileged children are less able to suppress distracting sounds when listening to stories and spoken language.<sup>105</sup> The authors have developed a training program to

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<sup>100</sup> Joan Luby et al., *The Effects of Poverty on Childhood Brain Development: The Mediating Effect of Caregiving and Stressful Life Events*, 167 JAMA PEDIATRICS 1135 (2013).

<sup>101</sup> See Pamela Hines et al., *Laying the Foundation for Lifetime Learning*, 333 SCIENCE 951 (2011). See also Joan Luby et al., *The Effects of Poverty on Childhood Brain Development: The Mediating Effect of Caregiving and Stressful Life Events*, 167 JAMA Pediatrics 1135 (2013).

<sup>102</sup> Lawrence J. Schweinhart, Benefits, Costs, and Explanation of the High Scope Perry Preschool Program, Paper Presented at the Meeting of the Society for Research in Child Development, Tampa, Florida (April 26, 2003).

<sup>103</sup> Helen Neville et al., *Neurocognitive Consequences of Socioeconomic Disparities*, 16 DEVELOPMENTAL SCIENCE 708 (2013).

<sup>104</sup> See Kimberly G. Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 NATURE NEUROSCIENCE 773 (2015); Greg J. Duncan & Katherine Magnuson, *The Long Reach of Early Childhood Poverty*, PATHWAYS 22 (Winter, 2011). See also NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS (2000).

<sup>105</sup> Courtney Stevens et al., *Neural Mechanisms of Selective Auditory Attention Are Enhanced by Computerized Training: Electrophysiological Evidence from*

counteract that effect, for which there is evidence of limited, short-term success.<sup>106</sup>

The discussions of policy in scientific reviews and the secondary literature tend to make bolder claims and set out more detailed proposals.<sup>107</sup> One book length treatment of early child development contains two chapters devoted to a lengthy list of recommendations, ranging from specific to vague, that purport to grow out of the evidence accumulated across fields, including developmental and deprivation neuroscience. These are, inter alia, creating “school readiness initiatives,” making greater investments in “young children’s mental health needs,” creating programs to help parents because “children’s early development depends on the health and well-being of their parents,” finding ways to reduce chronic stress “stemming from abuse and neglect throughout the early childhood years and beyond,” intervening to reduce exposures to poor nutrition, infections, environmental toxins, and drugs,<sup>108</sup> and providing more generous funding to “improve the qualifications and increase the

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*Language-impaired and Typically Developing Children*, 1205 BRAIN RESEARCH 55 (2008).

<sup>106</sup> *Id.*

<sup>107</sup> See Helen Neville et al., *Neurocognitive Consequences of Socioeconomic Disparities*, 16 DEVELOPMENTAL SCIENCE 708 (2013); Helen Neville & Courtney Stevens, Experience Shapes Human Brain Development and Function: A Framework for Planning Interventions for Children At-risk for School Failure, Summary of AAAS presentation, to be given February 15, 2008; Pamela Hines et al., *Laying the Foundation for Lifetime Learning*, 333 SCIENCE 951 (2011); NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS (2000); Ross A. Thompson, *Bridging Developmental Neuroscience and the Law: Child-Caregiver Relationships*, 63 HASTINGS L.J. 1443 (2011-2012); Clare Huntington, *Neuroscience and the Child Welfare System*, 21 JOURNAL OF LAW AND POLICY 37 (2012); Laurence Steinberg, Should the Science of Adolescent Brain Development Inform Public Policy?, Article Adapted from the Annual Henry and Bryna David Lecture, Delivered at the National Academy of Sciences (Nov. 2011); FRANCES JENSEN, THE TEENAGE BRAIN (2015); Paul Tough, *The Poverty Clinic*, THE NEW YORKER, March 21, 2011, at 31.

<sup>108</sup> NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS 391-92 (2000).

compensation” for “children’s nonparental caregivers.”<sup>109</sup> The authors do not explain how this lengthy wish list is tied to particular neuroscience findings, as opposed to behavioral observations or evidence from sociological studies generally. Nor is data presented on the results anticipated from these proposed programs, or how those results can be predicted from, or relate to, the findings on brain changes observed in deprived children.

In the same vein, Helen Neville and colleagues, in reviewing potential lessons from deprivation neuroscience, advise an intensive focus on “developing methods for ameliorating” observed SES disparities. Alluding to “mechanisms whereby SES can influence brain and cognitive development,” the authors conclude that “there is ample evidence that this [amelioration] can be done.” Their proposals include the familiar list of early childhood programs (Perry Preschool, Abecedarian, etc.), parental enrichment, training of school age children to improve attention (as developed by Stevens, et al, discussed above), and family-based coaching for parents.<sup>110</sup> The authors do not offer concrete evidence on the effectiveness of these interventions in reducing the SES-related brain differences documented in the neuroscience literature.

Similarly, in a 2011 issue of *Science* that includes a comprehensive review of the neuroscience to date, the editors discuss programs designed to enhance language comprehension, reading ability, executive function, memory, and learning generally.<sup>111</sup> Once again, the handful of small, intensive, initial early childhood programs (Perry preschool, Abecedarian, Chicago parent-child initiative) is highlighted. Other proposals including investing in teacher enrichment and quality, programs aimed at improving parenting and caregiving, and efforts to train children to improve self-control, executive function, memory, basic reasoning, mindfulness, selective

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<sup>109</sup> NATIONAL RESEARCH COUNCIL, INSTITUTE OF MEDICINE, FROM NEURONS TO NEIGHBORHOODS 387-403 (2000).

<sup>110</sup> Helen Neville et al., *Neurocognitive Consequences of Socioeconomic Disparities*, 16 DEVELOPMENTAL SCIENCE 708 (2013).

<sup>111</sup> Pamela Hines et al., *Laying the Foundation for Lifetime Learning*, 333 SCIENCE 951 (2011) .



attention, and language skills. The authors cite a few promising outcome studies, but acknowledge that most programmatic interventions have shown equivocal results. Once again, the connection to particular neuroscientific data, as opposed to behavioral observations or other evidence, is not explored.<sup>112</sup>

In a report on a recent interdisciplinary conference on the policy implications of developmental and deprivation neuroscience, The Institute for Research on Poverty issued a short published report of the proceedings, which were oriented towards “identifying ways that neuroscience research could be used to provide improved insights about the effects of poverty and to develop more effective antipoverty policies in response to these insights.”<sup>113</sup> The document contains many caveats and bemoans the lack of standardization in the “norms regarding what types of social, economic, and demographic data are collected in brain studies and how such factors should be measured.” Beyond that, the report contains few specifics on how neuroscience can be put to work to identify interventions that will actually prove effective in reducing the detriments of poverty, other than expressing the vague hope that “neuroscience studies focused on socioeconomic questions could offer the advantage of helping policymakers design interventions that are better targeted and more cost effective,” and also that “neuroscience may lead to better insights about whether an intervention leads to the intended result.”<sup>114</sup>

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<sup>112</sup> See Pamela Hines et al., *Laying the Foundation for Lifetime Learning*, 333 SCIENCE 951 (2011) and articles contained therein.

<sup>113</sup> *Deepening Connections between Neuroscience and Public Policy to Understand Poverty*, FAST FOCUS No. 23-2016 (June 2016).

<sup>114</sup> *Deepening Connections between Neuroscience and Public Policy to Understand Poverty*, FAST FOCUS No. 23-2016 2, 3 (June 2016). The report does assert that brain images might be able to offer “more real time feedback than behavioral or achievement-based measures” because behaviors “are not fully visible or measurable until years after the interventions, whereas brain scans may be able to show results more quickly after implementation of the intervention.” The report speculates that the “effects of . . . interventions might be captured far earlier using brain scans or other biological measures.” What the reports fails to note is that, unless behavioral effects are actually linked to the brain results, there is no guarantee that observed brain changes have any behavioral significance.

Finally, in a report published this past spring in the journal *Pediatrics*, the Council on Community Pediatrics issued a major policy statement from the American Academy of Pediatrics which set forth “Principles to Guide and Define the Child Health Care System and/or Improve the Health of all Children.” While not undertaking a detailed review, the report briefly alluded to behavioral and basic science research related to child poverty, and especially to the behavioral effects of toxic stress and adversity. The report contained a comprehensive set of recommendations for pediatric practice based on the research findings, including enhanced nutrition support, home visiting initiatives, parental training and support programs, and the expansion and funding for other “essential benefits programs that assist low-income and poor children,” including “Early Head Start and Head Start, Medicaid, CHIP, WIC, home visiting, SNAP, school meal programs and other programs that increase access to healthy food, and Child Care Development Block Grant–funded programs.”<sup>115</sup>

The report is accompanied by a separate article that focuses more specifically on the effects of poverty on the developing brain. After surveying the deprivation neuroscience in extensive detail, this article offers a host of programmatic and policy suggestions that are claimed to grow out of its findings, including implications for pediatric practice.<sup>116</sup> These include having primary care providers “evaluate and address social needs such as housing, employment, education and food,”<sup>117</sup> as well as “environmental mediators of neurodevelopment” such as “parenting stress, [and] cognitive stimulation.” The article goes on to propose that pediatricians participate in projects with child development specialists to provide “parent child interaction coaching” and to “support play and shared reading.” The report also urges pediatricians to advocate for “expanding high quality community resources for families, as well as

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<sup>115</sup> See American Academy of Pediatrics, *Poverty and Child Health in the United States*, 137 *PEDIATRICS* 1 (2016); Sara B. Johnson, Jenna L. Riis, and Kimberly G. Noble, *State of the Art Review: Poverty and the Developing Brain*, 137 *PEDIATRICS* 1 (2016).

<sup>116</sup> See Sara B. Johnson, Jenna L. Riis, and Kimberly G. Noble, *State of the Art Review: Poverty and the Developing Brain*, 137 *PEDIATRICS* 1 (2016).

<sup>117</sup> *Id.* At 12.

coordinated systems to implement them.” It suggests that “pediatricians may serve as ideal advocates for programs and supports that provide financial benefits to poor families,” which, it predicts, will produce improvements “in long-term cognitive and health outcomes.”

The article gives few details on how and whether pediatricians’ advocacy will actually result in measurable improvements in poor children’s behavior, brain morphology, or brain functioning. Nor does it provide data on the creation, enactment, and implementation of proposed interventions, or the concrete results expected from them. Also lacking is any explanation of how the neuroscientific insights, or any of the studies that link scan results to SES and measures of performance, add to the already existing body of developmental and behavioral psychology that purports to establish the importance of socioeconomic status to future outcomes for disadvantaged children. In sum, the value added by neuroscience, over and above other knowledge and informational sources, is neither explored nor explained, but simply taken for granted.

Law professors have also gotten into the business of relying on deprivation neuroscience to justify legal and policy recommendations. In touting a pro-active approach to child welfare and arguing against “minimalism” or less aggressive intervention, Professor Claire Huntington maintains that there is a “deeper and less recognized reason to question . . . minimalism,” which is the “growing body of research by neuroscientists.” She asserts that this research demonstrates “that a child’s early life experiences and environment literally shape the child’s brain architecture, with lifelong consequences that are very difficult to reverse[,]” and also that “children’s relationships with their primary caregivers” are “at the core of brain development.” When these relationships are deficient, she states, “the developing brain is deeply affected.”<sup>118</sup> The author concludes that the science teaches that “prevention is essential,” because the brain changes occasioned by poor caregiving

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<sup>118</sup> Claire Huntington, *Neuroscience and the Child Welfare System*, 21 JOURNAL OF LAW AND POLICY 37, 39-40 (2012).

can be hard to reverse. While acknowledging that the field of neuroscience may well be too nascent to form the basis of specific proposals,<sup>119</sup> she nonetheless puts forward a series of recommendations. She endorses the Nurse-Family Partnership initiative, which arranges public health nurse visits to low income mothers, as an “example of a program that helps foster child brain development.”<sup>120</sup> She adds that the research “cautions against the removal [from their parents] of young children in all but the most serious circumstances” because “the loss of the primary attachment figure for a very young child can be devastating. Instead the child welfare system should seek to treat the whole family.”<sup>121</sup>

Once again, missing from this exposition is a specific explanation of how neuroscience evidence adds to the already existing body of developmental and behavioral psychology that establishes the importance of caregiver behavior and child-maternal attachment. Nor does Huntington further support her proposition that family preservation, as opposed to a range of other potential responses to inadequate caregiving, is better for children. In short, she does not make clear how neuroscience contributes, or contributes uniquely, to her conclusions and legal recommendations.

Ross Thompson is another law professor who tries his hand at exploring the practical payoffs from the emerging body of developmental neuroscience.<sup>122</sup> In a lengthy article on how “understanding the biological foundations of human development” has “implications for legal analysis,” Thompson proceeds to recommend specific initiatives that he claims grow out of the scientific data. After reviewing some of the studies, Thompson concludes that the science reveals “complex interactions between brain maturation and experience over time” as well as the “importance of early experience and the significance of caregiving quality for buffering stress and enduring the consequences of early

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<sup>119</sup> *Id.* at 53.

<sup>120</sup> *Id.* At 50.

<sup>121</sup> *Id.* At 56.

<sup>122</sup> Ross A. Thompson, *Bridging Developmental Neuroscience and the Law: Child-Caregiver Relationships*, 63 HASTINGS L.J. 1443 (2011-2012).

adversity.” According to Thompson, the evidence of “brain plasticity” carries “the most important legal implications,”<sup>123</sup> because it “accounts for the efficacy of preventative and intervention efforts targeted to children in adversity,” and especially for early intervention.<sup>124</sup> It follow that measures should be adopted that “contribute to the prevention and remediation of conditions of early adversity,” including programs that “focus on the early identification of families at risk . . . and the provision of supportive services to strengthen the quality of care.” These include “home visitation programs, nutritional assistance, parental support, access to high quality child care, . . . other forms of ‘preventative family preservation,’ . . . well-designed early childhood education programs that are supplemented by social emotional support, and target programs to address the specific causes of parental inadequacy.”<sup>125</sup>

In light of Professor Thompson’s sweeping recommendations, two points are worth noting. First, observational studies and behavioral evidence, wholly apart from research on brain function, have long tried to establish the importance of early conditions of upbringing and yielded similar programmatic suggestions. Indeed, Thompson’s proposals echo those advanced by many developmental and behavioral psychologists and their political allies, and some have been or are being adopted. In short, there is nothing new or surprising here, and no special information or observations that bring unique insights to policy. Second, as with the other commentaries already discussed, the author does not tie particular neuroscientific findings to proposed interventional strategies. Nor does he link neuroscientific

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<sup>123</sup> *Id.* at 1457.

<sup>124</sup> *Id.* at 1443 (summary).

<sup>125</sup> *Id.* at 1456-57. Thompson cites the American Academy of Pediatrics policy statement “urging the involvement of pediatric[ians] in reducing childhood exposure to adversity and young children’s exposure to toxic stress through . . . parental education, developmental screening, and community advocacy,” as well as other measures designed to “reduc[e] the consequences of exposure for children.” *Id.* at 1453-54.

results to actual studies or predictions about the effectiveness of particular programs.<sup>126</sup>

As a general matter, the secondary literature that purports to find unprecedented programmatic insights in developmental neuroscience offers nothing new. Rather, it reiterates

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<sup>126</sup> In a similar vein, Paul Tough has written a popular article in *The New Yorker* on how physicians can benefit from the evidence generated by developmental neuroscience. The article reports on a study undertaken at Kaiser Permanente in the 1990s, called the Adverse Childhood Experience study and profiles a young pediatrician, Nadine Burke. It describes how Burke familiarized herself with the research on the effects of adverse experiences on health, including the creation of “lasting changes in the brain and the body.” While asserting that knowledge of how adversity “disrupts brain circuits” provides “a very exciting opportunity to bring biology into early childhood policy,” the article is hazy on how such insights might redirect actual medical practice. Rather, it echoes the general observation, set out by Huntington and Thompson, that “intervening early can improve later outcomes” in health, education, and behavior. The article adds the caveats that experts “are still struggling to figure out how to put this new theory into clinical practice” and that “there’s not yet a lot of good data to tell us which kinds of interventions are most effective.” Paul Tough, *The Poverty Clinic*, *THE NEW YORKER*, March 21, 2011, at 31.

Another example of claiming policy payoffs from neuroscience is to be found in a *British Guardian* article reporting on the 2015 Noble study shortly after it was published. As reported in that article, Michael Thomas, director of the Birkbeck-UCL Centre for Educational Neuroscience, suggests that this study and others like it could help researchers tease out more precisely how low income might affect children’s brains. “If we find that all these factors are equally responsible, that is prenatal health, stress levels, nutrition and cognitive stimulation, the only way to fix the issue is to get rid of poverty, and that’s a hard thing to do. But if we can narrow it down to some factors that are particularly influential in causing problems for the kids, that makes it more possible to intervene,” he states. He does not elaborate further on how neuroscience, as opposed to other behavioral scientific methods, can best perform this “narrowing down” function.

Finally, a short article appearing in May 2016 in the *New York Times* (Jim Dwyer, *Studying How Poverty Keeps Hurting Young Minds, and What to Do about It*, *N.Y. TIMES* (May 6, 2016), <https://www.nytimes.com/2016/05/04/nyregion/studying-how-poverty-keeps-hurting-young-minds-and-what-to-do-about-it.html>) draws on neuroscience studies, data on stress reactions, and the Adverse Childhood Experience study, to recommend “city policies like universal prekindergarten,” as well as forming “caring, consistent relationships with adults.” Once again, these proposals are not tied to specific research results.

recommendations that have long been popular among developmental experts from a range of social science fields in light of behavioral observations, accumulated evidence, or just plain common sense. For example, the eminent economist James Heckman, who studies the economics of human capital and skill formation, has long stressed the importance of early intervention to enhance the life-chances and trajectory of children from deprived backgrounds. Marshalling the evidence on the efficacy of interventions at different stages of childhood and adolescence, he has concluded that, because “skill builds on skill” and “early inputs strongly affect the productivity of later inputs,” interventions designed to alleviate early deprivation provide the most effective results.<sup>127</sup> Although Heckman’s work on child development does not rely on neuroscience, the policies he advocates match those recommended by commentators who purport to find startling insights in brain research. Clearly, brain science offers no such insights, and adds nothing to the social science and behavioral data already available.

In addition to reiterating proposals from that predate the research in deprivation neuroscience, the policy work that purports to rely on that science is largely oblivious to the peculiar obstacles and pragmatic difficulties of efforts to influence private behavior, especially as between parents and children and within families. Moreover, the vast majority of proposals are geared towards progressive, government-sponsored, collectively coordinated efforts. They emphasize top-down programmatic solutions and publically funded policies, without any attention to whether these actually work, or work best, to prevent the ill effects attributed to deprivation. Nor do they consider whether social problems such as “parental inadequacy” might better be rectified through informal, private measures that assign a much smaller role to government. In neglecting alternatives to conventional progressive initiatives, the commentators are oblivious to important strands of political and social science thought, including critiques of some governmental aid

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<sup>127</sup> For a summary of James Heckman’s work on skill development, see e.g., James Heckman, *Skill Formation and the Economics of Investing in Disadvantaged Children*, 312 *SCIENCE* 1900 (2006).

to the poor as promoting bad habits and discouraging avenues of self-help that might have more positive effects in the long run.<sup>128</sup> For example, there is research suggesting that irregular family forms and arrangements, such as multi partner fertility, step-parent and mixed parentage families, households where children cohabit with non-biologically-related males, and families experiencing serial adult partnerships, pose enhanced risks of child abuse and neglect.<sup>129</sup> One approach to that risk is to eschew financial benefits for irregular living arrangements, which may be encouraged by overly generous government subsidies, in favor of shoring up, advocating for, and supporting more traditional family forms.<sup>130</sup>

In choosing between these different alternatives to helping children avoid the ill effects of social disadvantage, deprivation neuroscience has nothing to offer. The information gleaned from the science is completely unenlightening as to the strategy that would best alleviate the effects of poverty and attendant adversities and behaviors, whether over the short or long term. Nor does it tell us whether support for traditional families and established structures is

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<sup>128</sup> See, e.g., CHARLES MURRAY, *LOSING GROUND* (1984); MYRON MAGNET, *THE DREAM AND THE NIGHTMARE* (2000); WILLIAM VOEGELI, *NEVER ENOUGH: AMERICA'S LIMITLESS WELFARE STATE* (2012). See also, e.g., *2016 Index of Culture and Opportunity: Poverty and Dependence*, MEDIUM, <https://medium.com/2016-index-of-culture-and-opportunity/poverty-and-dependence-7d5dd7d02b95> (last visited July 20, 2016) (detailing dependence-promoting aspects of welfare and transfer programs).

<sup>129</sup> MARTIN DALY & MARGO WILSON, *THE TRUTH ABOUT CINDERELLA: A DARWINIAN VIEW OF PARENTAL LOVE* (1999); Martin Daly & Margo Wilson, *Child Abuse and Other Risks of not Living with Both Parents*, 6 *ETHOLOGY AND SOCIOBIOLOGY* 197 (1985); Margo I. Wilson, Martin Daly, & Suzanne J. Weghorst, *Household Composition and the Risk of Child Abuse and Neglect*, 12 *JOURNAL OF BIOSOCIAL SCIENCE* 333 (1980).

<sup>130</sup> See, e.g., W. Bradford Wilcox, Ph.D., Paul Taylor, & Chuck Donovan, *When Marriage Disappears: The Retreat from Marriage in Middle America*, Heritage Foundation Lectures, Lecture No. 1179 on Family and Marriage, February 22, 2011, <http://www.heritage.org/research/lecture/2011/02/when-marriage-disappears-the-retreat-from-marriage-in-middle-america>; Kay S. Hymowitz, *Marriage and Caste in America: Separate and Unequal Families in a Post-Marital Age*, Heritage Foundation Lectures, Lecture No. 1005, January 30, 2007, <http://nhcornerstone.org/wp-content/uploads/downloads/2010/10/hl1005.pdf>.



more or less effective than the standard progressive approach, which relies on government-sponsored services and financial handouts for dysfunctional parents and at-risk families. In short, whether a given intervention or policy will help or hurt, alleviate the cognitive and behavioral problems associated with childhood disadvantage or backfire, do nothing or make things worse, is not information that neuroscience can provide. Neuroscience is completely non-committal and indeterminate on these questions. Yet the implicit assumption behind the articles that prescribe policies based on neuroscience seem to be that brain science can tell us what is needed, point the way to appropriate interventions, and assure their efficacy. These assumptions are dubious at every point. Deprivation neuroscience, in and of itself, cannot underwrite any policy proposal nor establish the effectiveness of any government-engineered intervention. Only evidence as to the concrete effects of actual programs, gathered in real time, can do that.

#### B. Neuroscience and disabilities law

Advocating for programs to address the social problem of childhood deprivation is not the only use to which deprivation neuroscience has been put by the legal and policy community. In a lengthy law review article, James Ryan takes the position that neuroscientific evidence dictates a fundamental restructuring of federal statutes regulating the education of learning disabled children.<sup>131</sup> The argument, which is well-crafted and thoughtful, merits serious consideration. Ultimately, it fails.

Although benefits and protections for the disabled are provided under several statutory schemes, Ryan is principally concerned with the federal Individuals with Disabilities Education Act (or IDEA), which mandates individually tailored special education programs for covered students, and provides protection from disciplinary sanctions.<sup>132</sup> Ryan finds fault with the IDEA

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<sup>131</sup> James E. Ryan, *Poverty as Disability and the Future of Special Education Law*, 101 GEORGETOWN LAW JOURNAL 1455 (2013).

<sup>132</sup> Individuals with Disabilities Education Act (IDEA), 20 U.S.C. A. §§ 1400-1444 (2004). See 20 U.S.C.A. § 1401 (10) (F) (14).

definition of covered learning disabilities and the manner in which the definition has been interpreted and applied. His central contention is that recent findings in deprivation neuroscience reveal that the scope of the statute's coverage should be expanded or significantly revised.

The IDEA protects children with a “disorder in one or more basic psychological processes” that interfere with the ability to “listen, think, speak, read, write, spell, or do mathematic calculations.” Specified disorders -- such as “brain injury, dyslexia, developmental aphasia”-- are deemed covered, but the statute contains discrete exclusions. Individuals are not considered learning disabled if their deficits are “primarily the result . . . of environmental, cultural, or economic disadvantage.”<sup>133</sup> Based on the statutory definition and exclusions, and in reliance on the consensus of disability experts, the IDEA's protections have been conferred almost exclusively on children who meet the so-called “discrepancy” model of disabled learning. Children conforming to this model characteristically combine an overall normal range intelligence with deficits in selective learning-related abilities (such as difficulties with reading or receptive language).

Ryan's main target is the exclusion from statutory protection of learning deficits associated with (and presumably caused by) social disadvantage. Ryan's argument centers on a key categorical distinction that, in his view, underwrites the express statutory exclusion of deficits traceable to low SES. Although acknowledging that the statute itself does not by its terms distinguish between “innate” versus “environmental” causes, Ryan construes the scope of the statute's coverage, at least as it has come to be applied, as turning on this distinction. Deficits deemed eligible are generally thought to stem from “innate” or purely “internal” influences, whereas the excluded categories -- such as problems with learning caused by “economic disadvantage” -- are those thought to be produced primarily by external or environmental forces. This divide, he argues, can be gleaned from the included and excluded categories, in

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<sup>133</sup> James E. Ryan, *Poverty as Disability and the Future of Special Education Law*, 101 THE GEORGETOWN LAW JOURNAL 1455, 1457 (2013).

conjunction with “neighboring language in the statute.” As Ryan sees it, “[t]here appears to be universal agreement among commentators that ‘real’ learning disabilities are “an internal disorder, innate to the students.”<sup>134</sup> The operating assumption is that these types of deficits are rooted in purely biological or neurological defects that are the result of defective genes or genetic accidents. In other words, disabled students are “born that way.” They do not “become that way” through the agency of external influences such as poverty and disadvantage.

Ryan draws on the findings of deprivation neuroscience to argue that the exclusion for learning deficits traceable to poverty and disadvantage is unjustified and incoherent, and that these difficulties should be treated the same as learning problems stemming from other (purely “innate”) causes. The neuroscience, he claims, shows that “a child’s environment,” including low SES, alters brain development, structure, and function in “physical mechanistic ways.” Those effects, argues Ryan, are “no less concrete and real,” and of no lesser concern for the educational fate and functioning of the developing children, than brain changes due to genetics, disease, or exposure to neurotoxins – including the genetically programmed, “innate,” structural deficits that are thought to lie behind the stylized learning disabilities recognized as within the statute’s scope.<sup>135</sup>

Central to Ryan’s thesis is his “challenge [to the] the notion that there is a sharp divide between internal and external causes of

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<sup>134</sup> *Id.* at 1458 & note 14.

<sup>135</sup> James E. Ryan, *Poverty as Disability and the Future of Special Education Law*, 101 THE GEORGETOWN LAW JOURNAL 1455, 1491 (2013). In attacking the “discrepancy” model of learning disability, Ryan also asserts that “researchers consistently [have] found little, if any, difference between those identified as disabled [on this behavioral criterion] and those considered slow learners.” According to Ryan, the many studies revealing similarities between the two groups across a “host of dimensions, including achievement levels, growth, behavior, response to instruction” or even “a broad range of neurobiological factors” provide additional support for abandoning the categorical distinctions, including the exclusion for poverty-related learning problems, that are inscribed in the law. *Id.* at 1472.

learning disabilities.”<sup>136</sup> The most important aspect of his argument for our purposes, however, relates to how he justifies his position. He states that “the research regarding the effects of poverty on brain development” not only calls into question the “entire concept of innate or inherent learning disabilities,” but also any categorical distinction between deficits that are “innate” and those that are not. Because there is now “scientific” proof that poverty alters the brain, and evidence on how that happens, the IDEA should be rewritten to broaden the scope of its protections.

Ryan’s argument is marred by some confusion surrounding the word “innate.” One possible interpretation of that word is that it connotes a purely internal cause for the brain state and/or accompanying behavior at issue. On this meaning, an “innate” brain state is one attributed to purely genetic or inherited factors (and thus independent of external and environmental influence). But the word “innate” can also carry a different (and broader) meaning, as simply equivalent to “internal,” in the sense of being accompanied by, and determined by, a particular brain state, regardless of causation or how that brain state came about. In this sense, all behaviors are ultimately “innate” in that they all are grounded in a corresponding, specific brain state.

Ryan’s use of the term appears to adopt the latter meaning. His principal point appears to be that, although the causal path to learning deficits may implicate a spectrum of genetic versus environmental causes, all learning difficulties are, in the end “innate,” in that they correspond to an alteration or abnormality in the “internal” state of the brain. What should matter is this end point of the brain, and how it functions, not how that end point was achieved. There appears to be no principled reason to distinguish between different causal pathways (environmental, genetic, or some mix) leading to the “internal deficiencies or modifications” in brains that compromise the ability to learn. What matters is the state of the brain that has trouble learning, and not how the brain state came about. Provenance is irrelevant to anything we should care about. The

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<sup>136</sup> *Id.* at 1479.

distinction between “born” and “made” learning deficits, argues Ryan, is arbitrary and unjustified. Therefore, the law should treat them equally.

Ryan acknowledges that his reasoning entails a *reductio*. All learning difficulties due to environmental influence, or innate factors, or some mix of these, appear to be candidates for designation as “disabilities,” because all shortcomings would appear to have a distinct basis in the brain, whatever the source.<sup>137</sup> As Ryan puts it “any difference in cognitive function whether genetic or environmental in origin, reflects a difference in brain function.”<sup>138</sup> So every learning impairment, however defined, can potentially be included under the rubric of “brain dysfunction.” Ryan acknowledges that this line of reasoning can lead to potentially awkward practical consequences. If all behavioral deficits, including all diminutions in learning ability regardless of cause, are “written on the brain,” then it follows that all would satisfy the criterion of being “internal” or “innate.”

But if that is the case, what is the scope of the category of “learning disabled?” Indeed, because everything that alters the ability to learn “changes the brain,” compensable disabilities could result from virtually any factor that influences learning ability or actual learning. Ryan notes, for example, that inadequate instruction throughout the school years can “influence brain development and function.”<sup>139</sup> In the same vein, the failure to enroll in a quality preschool program can produce a lifelong lag in subsequent academic achievement. So can growing up in a culture that de-emphasizes learning, or reading, or verbal communication, or analytic thought. At the very least, Ryan’s critique threatens to demolish the distinction

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<sup>137</sup> Ryan downplays or just ignores the possibility that poverty-related defects may stem partly or largely from innate or genetic factors, but that failure makes no real difference for his argument. Even if poverty-related difficulties are not, as he seems to assume, wholly “caused” by deprivation, that does not undermine his basic point that the current legal treatment based on internal vs. external causation is dubious.

<sup>138</sup> *Id.* at 1497.

<sup>139</sup> James E. Ryan, *Poverty as Disability and the Future of Special Education Law*, 101 THE GEORGETOWN LAW JOURNAL 1455, 1496 (2013).

between the learning disabled and ‘garden variety slow learners’ who were never meant to be covered by the statute. Indeed, if the disabled category rightly includes anyone whose performance falls below a certain threshold, regardless of the reason, the critical question then becomes who is sufficiently deficient. How far short of normal would qualify for preferential treatment? It’s hard to know whether a dramatic expansion in the category of children entitled to the special protections and extra resources fits in with the overall goal of IDEA, because that goal is not well-articulated. Ryan gestures towards these conundrums without exploring or solving them.

Ryan’s critique of the IDEA has much to recommend it. There are many good reasons to question the “discrepant learning” model that the IDEA appears to adopt. Unfortunately, the lessons of neuroscience are not among them. No discoveries from that field justify a critique of this statute’s scope, which stands or falls regardless of how poverty alters the brain or deprivation compromises the ability to learn. Understanding these mechanisms merely provides particularized evidence for a core materialist axiom: that distinct behaviors correspond to different brain states. But that generic insight was already uniformly accepted and widely understood before any of the neuroscience research was conducted. The specific findings of neuroscience add nothing to it. In particular, they provide no independent rationale for impugning the IDEA’s definitional distinctions or for revising the law.

This point is supported by a critique of the IDEA written nearly fifteen years before Ryan’s article appeared. In *Jumping the Queue*, Mark Kelman and Gillian Lester advance an argument similar to Ryan’s: the scope of the IDEA’s protections are arbitrary and unjustified. Their reasoning, however, relies only peripherally on empirical evidence, and then almost entirely on behavioral studies. The main justifications for their position are normative and pragmatic.<sup>140</sup> According to Kelman and Lester, children the IDEA defines as “learning disabled” should not be singled out for special

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<sup>140</sup> See, e.g., MARK KELMAN & GILLIAN LESTER, *JUMPING THE QUEUE* 156-57 (1998).

benefits, entitlements, and protections. Rather, a wide variety of pupils, including the “socioeconomically disadvantaged” and “poor achievers” generally,<sup>141</sup> should receive extra help, protection, and resources. Kelman and Lester attack the existing statutory categories mainly on grounds of justice.<sup>142</sup> Based on “competing principles” of redistribution that “demand that we compensate for environmental or both environmental and genetic disadvantages,”<sup>143</sup> the authors deny that LD students are more deserving of special help and protection than students with other types of learning impairments, including those traceable to poverty or social deprivation.<sup>144</sup> In none of these cases are the students at fault or causally responsible for their difficulties. Rather, because they are the victims of bad luck (either from an unfortunate biological endowment or from being born into poverty and disadvantage), it is society’s obligation to offer them aid and hold them harmless to the extent feasible. Kelman and Lester also question the evidence that classic learning disabled, or LD,

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<sup>141</sup> MARK KELMAN & GILLIAN LESTER, *JUMPING THE QUEUE* 157 (1998).

<sup>142</sup> Kelman and Lester, like Ryan, also argue that the categorical distinction between children with a “bona-fide” learning disability (LD) and underachievement from other causes rests on shaky scientific grounds. But the science they rely on is not the new neuroscience of disadvantage, which postdates the publication of their book. Rather, they question the behavioral diagnostic techniques long used to label children as having LDs on the traditional “deficit” model, finding “rampant” misclassification and “considerable similarities” in patterns displayed by students across a broad spectrum of learning deficits. MARK KELMAN & GILLIAN LESTER, *JUMPING THE QUEUE* 29 (1998).

<sup>143</sup> *Id.* at 156.

<sup>144</sup> *Id.* at 195 et seq. The authors also speculate that favoring learning disabled (LD) children with a “performance-potential gap” fits in with a “left multicultural” identity politics consistent with an anti-discrimination model that views these pupils as in greater danger of suffering “discrimination” than children who are garden-variety poor learners, or merely “slow” for a range of “ordinary” reasons, including cultural deprivation or poverty. That is, “[p]upils with [these] learning disabilities alone are entitled because they alone have been constructed as a politically plausible oppressed group, a subclass . . . of a larger oppressed group, people with disabilities,” who are known to be subject to “aversive prejudice and stereotyping.” Children who are merely poor or culturally deprived, in contrast, do not belong to a distinct group of social victims, subject to special forms of mistreatment and entitled to extra solicitude.

students benefit more from special help than other “slow learners” and “poor readers” without such a diagnosis.<sup>145</sup> On consequentialist or efficiency grounds, they contend, there is also no basis for any distinction among these categories of children.

Like Ryan, Kelman and Lester conclude that the definition of “learning disabled” within the IDEA is both arbitrary and too narrow. But their route to this conclusion is very different from Ryan’s. Ryan argues that findings of neuroscience dictate a revision of the category of students the law protects. In contrast, Kelman and Lester formulate an argument from principles of fairness and desert, relying on luck egalitarian ideals that recognize society’s obligation to aid people who are disadvantaged through no choice or fault of their own.<sup>146</sup> A variety of children have reading and learning difficulties that hold them back in school, compromise their academic achievement, and impede their life prospects. Slow learners are slow learners. Since these shortcomings are not their fault, why does it matter how they got to be that way? Moral and normative considerations, and not any

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<sup>145</sup> MARK KELMAN & GILLIAN LESTER, *JUMPING THE QUEUE* 219 (1998). *See also id.* at 33, 118, 138, 152. Ryan, for his part, does not completely ignore the question of interventional efficacy. He points out that, in 2004, the IDEA was modified to allocate some money to struggling students based solely on their projected “response to intervention (RTI),” or potential for improvement, regardless of the source of their learning difficulties. As Ryan notes, the RTI approach sidesteps the strict disability definition and encourages schools “to extend extra assistance to ALL struggling students” – including poor students. Ryan further observes that whether interventions improve learning does not map onto the traditional learning disability or LD discrepancy category – “a failure to respond to interventions has not been confirmed as a reliable method of identifying students with learning disabilities.” Ryan at 1476.

<sup>146</sup> For a recent exposition of the luck egalitarian approach to societal compensation and redistribution see Peter Vallentyne, *Brute Luck and Responsibility*, August 3, 2016 draft, online and on file with author. *See also* JUSTICE AND BAD LUCK, Stanford Encyclopedia of Philosophy, April 11, 2014, <https://plato.stanford.edu/entries/justice-bad-luck/> (visited December 19, 2016).



information revealed by neuroscience, render the IDEA's entitlements and benefits unjust and incoherent.<sup>147</sup>

Kelman and Lester's book underscores that Ryan's reliance on deprivation neuroscience to attack the IDEA is misguided. To be sure, all the authors accept basic understandings that are grounded in the bedrock facts of biological materialism. Their arguments assume a fundamental relationship between the brain and behavior, and between mental states and physical states. People are organic, biological beings, and all behavior has a biological foundation. It follows that every distinct behavior corresponds to a distinct brain state and there can be no change in behavior without a change in the brain. Difficulties with learning, whatever their source or profile, can be traced to brains structures or functions that are not present in the brains of those lacking such difficulties.

In addition, these authors' arguments are consistent with the fundamental tenets and causal predicates of behavioral genetics. Brain states and human behavior are the product of a range of internal and external forces, including genetic or "innate" predispositions, environmental factors, and some interactive mixture of the two. These are categorical understandings that predate, and do not depend on, any particular body of neuroscientific research or any specific findings at all. They have been repeatedly validated by our understanding of the biological nature of organisms, and require no

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<sup>147</sup> MARK KELMAN & GILLIAN LESTER, *JUMPING THE QUEUE* 218-226; 194-226 (1998). Kelman and Lester, although resting on normative arguments, do not fully engage the question of the incentive effects created by a significant expansion in the IDEA entitlement scheme. For example, if parental behavior, for which parents can arguably be held responsible, contributes to the learning deficits of their children, society may want to be less generous towards those deficits – both from the point of view of desert and moral hazard – than towards those due to the misfortune of a poor genetic endowment. On the other hand, drawing these distinctions requires visiting the sins of the parents on the children, which is widely considered unjust. And, indeed, all the authors here seem to accept the fundamental tenet that, whatever the provenance of their learning difficulties, children are essentially blameless and equal in their deservingness. Even conceding that is the case, however, moral hazard for social actors may differ depending on category and circumstances.

specific observation about particular brains. Scans and measurements do not deepen our understanding of these fundamental truths. Neuroscience only fills in the details. None of these details matter to the justice of the IDEA.

VI. The policy payoff from deprivation neuroscience

Deprivation and disadvantage, and their effects on human behavior, are social problems of vexing urgency with no obvious, easy solutions. Neuroscience has little or nothing to contribute to addressing these problem, and is unlikely to add anything of significance in the future. Specifically, developmental neuroscience yields no distinct information on how to design interventions, programs, and policies to alleviate social and economic disadvantage and its effects, over and above contributions from cognate fields and disciplines that are focused on behavioral measures and outcomes. These include behavioral neuroscience, cognitive psychology, child development, sociology, behavioral psychology, developmental economics, and demographics.

The payoff from neuroscience is seriously circumscribed by a signal shortcoming that it shares with behavioral and social science: the limited ability to distinguish the effects of external influences from “selection” – that is innate or genetic factors that are associated with, and causally productive of, the behaviors associated with deprivation. And to the extent that “brain science” has any relevance to legal and policy questions, its contribution comprises the generic axiom of scientific materialism that long predates any specific findings in the field.

As already discussed, many commentators rely on neuroscience for the proposition that childhood SES exerts a significant effect on brain structure and function. But, even if this strong causal statement is correct, it was already understood from studies of behavior. At most, recent neuroscience reinforces this message and fills in some details. It identifies distinctive patterns of brain morphology and/or activity associated with particular behavioral deficits found more commonly among lower SES individuals. But that association offers little help in formulating effective proposals for

alleviating disadvantage and its consequences. Designing policies and interventions requires, first, that the goals of such efforts be identified. In the case of disadvantage, the objective is either to diminish or eliminate the disadvantage itself, or to reduce, reverse, or attenuate its effects. Second, establishing the efficacy of any interventions depends on an arduous process of evaluating outcomes. On the simplest level, and assuming causation, prevention obviously works. If no one were poor, the developmental impairments that poverty produces would be avoided. Eliminating a cause – assuming it is a cause -- eliminates its effects.

But eliminating disadvantage, in all its aspects, is devilishly difficult. The project founders on formidable practical impediments as well as a host of unknowns. The term “poverty” draws our attention to a lack of material resources. But too little money may not be responsible for the deficits associated with poverty, and providing what money can buy won’t necessarily work improvements. Can neuroscience help sort out whether lack of money, or something else correlated with it, stands at the head of the chain of adverse influences on the developing brain? At best, neuroscience is an intermediate step, and a strictly optional one, towards illuminating the question of whether the particular behavioral traits associated with poverty – including problems with memory, learning, self-control, impulsivity, or verbal ability – are the product of inadequate parenting, family environment, material deprivation, direct genetic transmission, or some combination of these.

Because behavioral patterns, including inputs and outputs, are mandatory components of any fruitful investigation of poverty and its consequences, all neuroscience must establish its claims by linking up physical findings with behavior. The study of behavioral patterns is thus an essential component of any meaningful research that identifies brain states and neuro-functional mechanisms associated with producing or alleviating deprivation. Likewise, in figuring out what works to address the effects of deprivation, it is behavior, and not brain states, that must be the focus of the inquiry. Behavioral outcomes are the ultimate test of whether a set of external interventions will prevent, attenuate, or cure the detrimental effects of

deprivation. Neuroscience can do no better than the behavioral evidence itself. It thus adds nothing to policy design, over and above what behavioral science can yield.

It follows that good behavioral measures are essential to crafting effective policies. And predictions about any policy's effectiveness are only as reliable as the behavioral evidence on which those predictions rely. Moreover, because neuroscience data on poverty's effects must always be correlated with behavioral observations, showing that associated brain states are altered or improved by specific interventions is a purely optional step. Certainly, knowledge of mechanisms and morphology cannot teach us how to address poverty and its effects, whereas behavioral observations are essential, and indispensable, to the discovery of pragmatic payoffs. Only behavioral studies can establish the association between disadvantage and effective methods for alleviating its detrimental effects. The information generated by neuroanatomical or functional imaging is neither necessary nor sufficient for identifying effective interventions.

Might neuroscience nonetheless offer the promise of a streamlined methodology? It has been suggested that, if neuroscience could eventually establish an airtight connection between brain states and designated behavioral effects, neuro-morphological or physiological markers might then serve as proxies that can supplant or stand in for behavioral measures.<sup>148</sup> That suggestion is dubious. Such a substitution is not only superfluous, but also burdensome and impractical. Scanning a brain or measuring brain activity is almost always harder and more intrusive than directly documenting the behavioral changes that are the ultimate target of any policy intervention and the final gauge of its effectiveness.<sup>149</sup> And the link

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<sup>148</sup> See, e.g., suggestion discussed in *Deepening Connections between Neuroscience and Public Policy to Understand Poverty*, FAST FOCUS No. 23-2016 (June 2016).

<sup>149</sup> Kent A. Kiehl et al., *Temporal Lobe Abnormalities in Semantic Processing by Criminal Psychopaths as Revealed by Functional Magnetic Resonance Imaging*, 130 PSYCHIATRY RESEARCH: NEUROIMAGING 27 (2004); Robert D. Hare & Craig S. Neumann, *Psychopathy as a Clinical and Empirical Construct*, 4 ANNUAL REVIEW CLINICAL PSYCHOLOGY 217 (2008). See, e.g., Adelle

between the proxy and the behavior is difficult and cumbersome to establish. The complexity of behavior and its circumstances suggests that myriad trials, in a range of situations, would be necessary to establish any neural signature, and would always leave open the possibility of unanticipated circumstances and imperfect prediction. In sum, evaluating interventions by looking at brain states is unlikely to yield simpler and more reliable information than is available through behavioral avenues of empirical investigation and analysis.

The conclusion that brain science offers no independent programmatic payoff is further demonstrated by the paucity of novel or unprecedented recommendations to be found in the neuroscience literature and the commentary that draws out its implications. As discussed above, commentators repeatedly highlight the Perry preschool, Abecedarian, and the Chicago parent-child projects, initiated in the 1960s and 1970s, that offered intensive services to poor children and their families, with modest long-term results. The other suggested measures comprise a familiar litany of projects that have been touted by progressive specialists for decades. At best, neuroscience supports (although does not definitively establish) developmental experts' longstanding position that help for poor children should come earlier rather than later. Unfortunately, the rate limiting step in implementing that insight is not a lack of scientific knowledge. The initial success of the few most promising early childhood programs have never been replicated on a large scale, and other initiatives have had at most limited value. The obstacles are behavioral, pragmatic, ethical, cultural, material, and political. Knowing more about the brain won't help remove them.

The policy payoff of neuroscience is further undercut by the failure, and indeed the inability, of the research to disentangle the role of environmental and innate factors in generating the detriments of disadvantage. As already discussed, reducing poverty is most likely to make a difference if the experience of poverty, as opposed to innate characteristics, is the main or sole engine of the ill effects associated

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E. Forth et al., *The Assessment of Psychopathy in Male and Female Noncriminals: Reliability and Validity*, 20 PERSONALITY AND INDIVIDUAL DIFFERENCE 531 (1996).

with low SES. Although traits with a significant genetic component are not necessarily fixed and impervious to interventional improvements, they might be. In any event, because the nature of gene/environmental interaction is often poorly understood, the payoff from any proposal must be assessed on a case by case basis. But this constraint, even when acknowledged, is often minimized by scientists working in the neuro-deprivation field and scholars commenting on their work. Implicitly or by implication, environmental factors are assumed to dominate, especially when it comes to neuroscience's potential to shape effective interventions.

Is science close to sorting out the importance of genes and environment in accounting for the patterns associated with lower SES? The answer is no, and further research faces practical and ethical impediments. As noted, prospective randomized trials, with well-defined treatment and control groups, provide the most promising avenue for disentangling causal mechanisms and identifying effective interventions. But the practical ambit of such trials is limited.<sup>150</sup> At the end of the day, there is no substitute for seeing what works. What matters is whether specific forms of assistance improve target behavior. Visualizing or measuring something about the brain is not an essential component of the inquiry. Brain science, although intrinsically interesting, is inessential clutter.

## VII. Conclusion:

Neuroscientific studies are worth doing, as they add to our general knowledge of the brain. But they do not help us with the social problems of deprivation, poverty, and disadvantage. Neuroscience yields no independent policy payoff in these areas, and scholars, scientists, and journalists should stop claiming otherwise. Because causation is so difficult to disentangle, the effects of particular interventions on children growing up in deprived circumstances cannot be predicted through any neuroscientific investigations, but only by practical ones, deploying the methods of

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behavioral and social science. Designing policies depends on real-world constraints and behaviors, and the feasibility – ethical, pragmatic, and political -- of their manipulation. Neuroscience adds nothing to this inquiry.