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

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THE POVERTY OF THE NEUROSCIENCE OF POVERTY: POLICY PAYOFF OR FALSE PROMISE?

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ABSTRACT: A recent body of work in neuroscience examines the brains of people suffering from social and economic disadvantage. This article assesses claims that this research can help generate more effective strategies for addressing these social conditions and their effects. It concludes that the so-called neuroscience of deprivation has no unique practical payoff, and that scientists, journalists, and policy-makers should stop claiming otherwise. Because this research does not, and generally cannot, distinguish between innate versus environmental causes of brain characteristics, it cannot predict whether neurological and behavioral deficits can be addressed by reducing social deprivation. Also, knowledge of brain mechanisms yields no special insights, over and above behavioral science and social observation, into how to alleviate harms attributed to deprivation. That project depends on changing real-world circumstances and behaviors, which is limited by ethical, practical, and political constraints.

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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.[1] The findings of neuroscience have been adduced to explain addiction,[2] criminal offending,[3] teenage risk-taking,[4] compulsive gambling and shopping,[5] academic underachievement,[6] deception,[7] and the prevention of social violence.[8] One expanding

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1. In their recent book, Sally Satel and Scott O. Lilienfeld note that "[t]he media—and even some neuroscientists, it seems—love to invoke the neural foundations of human behavior to explain everything . . . ." SALLY SATEL & SCOTT O. LILIENFELD, BRAINWASHED: THE SEDUCTIVE APPEAL OF MINDLESS NEUROSCIENCE, at ix (2015).

2. *Id.* at 51.

3. *See, e.g.*, A PRIMER OF CRIMINAL LAW AND NEUROSCIENCE at xv–xvi (Stephen Morse & Adina Roskies eds., 2013); *see also* Eyal Aharoni et al., *Can Neurological Evidence Help Courts Assess Criminal Responsibility? Lessons from Law and Neuroscience*, 1128 ANNALS N.Y. ACAD. SCI. 145 (2008); Nita Farahany, *Neuroscience and Behavioral Genetics in US Criminal Law: An Empirical Analysis*, 2 J. L. & BIOSCIENCES 485, 487 (2015).

4. FRANCES JENSEN & AMY ELLIS NUTT, THE TEENAGE BRAIN 105–06 (2015); Laurence Steinberg, *Should the Science of Adolescent Brain Development Inform Public Policy?*, ISSUES SCI. & TECH., Spring 2012, at 67.

5. *See, e.g.*, SATEL & LILIENFELD, *supra* note 1, at 43–47. *See generally* COMM. ON INTEGRATING THE SCIENCE OF EARLY CHILDHOOD DEVELOPMENT, NAT'L RESEARCH COUNCIL & INST. OF MED., FROM NEURONS TO NEIGHBORHOODS: THE SCIENCE OF EARLY CHILDHOOD DEVELOPMENT (Jack P. Shonkoff & Deborah A. Phillips eds., 2000) [hereinafter FROM NEURONS TO NEIGHBORHOODS].

6. *See, e.g.*, MARK KELMAN & GILLIAN LESTER, JUMPING THE QUEUE (1998).

7. *See* Giorgio Ganis & Julian Paul Keenan, *The Cognitive Neuroscience of Deception*, 4 SOC. NEUROSCIENCE 465, 466 (2009).

8. *See Lectures and Panels: Battling Blood in the Streets: How Can Neuroscience Promote Public Health and Support Public Policy to Prevent Community Violence?*, PETRIE-FLOM CTR.

area of inquiry is the neuroscience related to the effects of social disadvantage.[9] Social and cognitive scientists have long documented a range of behavioral and cognitive deficits that are found more commonly in people from deprived circumstances.[10] 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 (SES).[11] Scientists have 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.[12]

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. 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 finding methods for preventing the negative behavioral and cognitive effects of social and economic disadvantage or for curing them once they occur.

(Sept. 7, 2016), http://petrieflom.law.harvard.edu/events/details/battling-blood-in-the-streets. Panelists included Michelle Bosquet Enlow, Shannon Cosgrove, Fatimah Loren Muhammad, and Charles Homer. *Id.*

9. *See, e.g.*, Daniel A. Hackman et al., *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 NATURE REVS. NEUROSCIENCE 651, 651 (2010).

10. *See, e.g.*, Hackman et al., *supra* note 9; Robert M. Sapolsky, *Stress and Cognition*, *in* THE COGNITIVE NEUROSCIENCES 1031 (Michael S. Gazzaniga ed., 2004); Stephanie H.M. van Goozen et al., *The Role of Neurobiological Deficits in Childhood Antisocial Behavior*, 17 CURRENT DIRECTIONS PSYCHOL. SCI. 224 (2008); *see also* Vincent J. Felitti, *Adverse Childhood Experiences and Adult Health*, 9 ACAD. 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 AM. J. PREVENTIVE MED. 245 (1998); W. Thomas Boyce, *A Biology of Misfortune*, 29 FOCUS (Inst. for Research on Poverty, Madison, Wisc.), Spring/Summer 2012, at 1; Paul Tough, *How Kids Learn Resilience*, ATLANTIC (June 2016), http://www.theatlantic.com/magazine/archive/2016/06/how-kids-really-succeed/480744/.

11. Hackman et al., *supra* note 9.

12. *See* FROM NEURONS TO NEIGHBORHOODS, *supra* note 5; KELMAN & LESTER, *supra* note 6; Martha J. Farah et al., *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); Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 TRENDS NEUROCOGNITIVE SCI. 65 (2009); Hackman et al., *supra* note 9; Boyce, *supra* note 10.

In addressing these questions, the article first reviews the literature that seeks to connect SES to structural and functional aspects of the brain and the behaviors linked to them. 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 contending that neuroscientific findings on poverty's effects on the developing brain dictate a revision of a federal law, the Individuals with Disabilities Education Act, which extends special protections and benefits to children with learning disabilities.

Drawing on these discussions, the article 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.[13] 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 generated from other fields, including most notably cognitive and behavioral psychology, into how society should address poverty, reverse its detrimental effects, or both. The sole insight

13. In contrast, there is not 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. Hackman et al., *supra* note 9, at 651, 657. However, neuroscience has yet 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. *See id.*

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 nonspecific 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 article 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 or on how deprivation alters these. Additionally, such observations cannot predict, determine, identify, or establish what works to prevent or reverse documented brain deficits and corresponding behavioral shortcomings.[14] The effectiveness of any interventions must ultimately be demonstrated 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 they 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.

I. RESEARCH ON THE NEUROSCIENCE OF DEPRIVATION

Numerous studies attempt to examine the link between various social outcomes and low SES, variably defined to include poverty, lack of education, low social capital, and other measures.[15] The biological and physiological correlates of these outcomes are the subject of research in several disciplines.[16] 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 illness and physical afflictions.[17] Poverty has been tied to higher levels of stress, exaggerated reactivity, mental disorders and emotional disturbance, impulsive and aggressive behaviors, and vulnerability to addiction.[18] These effects are believed to be mediated, at least in part, through the

14. *See infra* Part II.

15. *See, e.g.*, Hackman et al., *supra* note 9 ("SES is a complex construct that is based on household income, material resources, education and occupation, as well as related neighborhood and family characteristics, such as exposure to violence and toxins, parental care and provision of a cognitively stimulating environment.").

16. *See, e.g.*, FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 385 ("State-of-the-art knowledge about early childhood development is multidimensional and cross-disciplinary. It extends from painstaking efforts to understand the evolving circuitry and biochemistry of the immature brain to large-scale investigations of how family characteristics, neighborhood influences, and cultural values affect the well-being of children as they grow up."); Martha J. Farah et al., *Childhood Poverty: Specific Associations with Neurocognitive Development*, 1110 BRAIN RES. 166 (2006) (describing research focused on a neuroanatomical approach).

17. Felitti et al., *supra* note 10; Boyce, *supra* note 10.

18. *See, e.g.*, Emalee G. Flaherty et al., *Adverse Child Exposures and Reported Child Health at Age 12*, 9 ACAD. PEDIATRICS 150 (2009); Hackman et al., *supra* note 9, at 651–52; Boyce, *supra*

activation of hormonal and stress-reactive pathways, which operate through, and alter, brain circuitry, structure, and function.[19]

A large and growing body of research has focused specifically on the behavioral and cognitive deficits found in children growing up in deprived circumstances.[20] Specifically, it is well documented that childhood SES is "correlated with intelligence and academic achievement from early childhood and through adolescence."[21] 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).[22] They also lag in noncognitive behavioral traits (such as executive function, perseverance, self-control, discount rate and ability to delay gratification) that predict academic and life success.[23] Consistent with this research, children from disadvantaged backgrounds are at enhanced risk to suffer from academic difficulties in school, commit disciplinary infractions, and display self-destructive and antisocial behavior.[24]

The neuroscience of deprivation aims to build on these types of behavioral observations to generate a brain-centered body of knowledge that is focused on defined neuroanatomical and neurofunctional systems. The goal is to detail how poverty and low SES might alter particular brain structures, activities, and connections, and to show how specific adverse 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 decision-making 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

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note 10; *see also* Felitti, *supra* note 10; Felitti et al., *supra* note 10; Sapolsky, *supra* note 10; Van Goozen et al., *supra* note 10; Tough, *supra* note 10.

19. *See, e.g.*, Hackman et al., *supra* note 9, at 653–55; Sonia J. Lupien et al., *Effects of Stress Throughout the Lifespan on the Brain*, 10 NATURE REVS. 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).

20. *See, e.g.*, FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 5; KELMAN & LESTER, *supra* note 6, at 71–82; Farah et al., *supra* note 12; Daniel A. Hackman & Martha J. Farah, *Socioeconomic Status and the Developing Brain*, 13 TRENDS NEUROCOGNITIVE SCI. 65 (2009); Hackman et al., *supra* note 9; Boyce, *supra* note 10; *see also* Tough, *supra* note 10 ("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.").

21. Hackman et al., *supra* note 9, at 651.

22. *See* FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 287; Daniel A. Hackman et al., *Socioeconomic Status and Executive Function: Developmental Trajectories and Mediation*, 18 DEVELOPMENTAL SCI. 686, 696–98 (2015). *See generally* 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 et al. eds., 2016).

23. Hackman et al., *supra* note 9, at 696.

24. *See, e.g.*, Christine Christle et al., *School Characteristics Related to High School Dropout Rates*, 28 REMEDIAL & SPECIAL EDUC. 325, 333 (2007); John M. Wallace, Jr. et al., *Racial, Ethnic, and Gender Differences in School Discipline Among U.S. High School Students: 1991–2005*, 59 NEGRO EDUC. REV. 47, 57–59 (2008); John Paul Wright et al., *Prior Problem Behavior Accounts for the Racial Gap in School Suspensions*, 42 J. CRIM. JUST. 257, 257 (2014).

detailed pictures of brain morphology and ongoing brain activity have enabled researchers to link up brain structure and function with various behavioral states and syndromes and to picture the brain while it performs a range of activities linked to cognitive abilities.[25] 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.[26]

Martha Farah and her colleagues have been particularly influential in galvanizing and shaping research in the field by developing a theoretical map, based on preexisting neurofunctional and neuroanatomical studies, which predicts the areas of the brain most likely to be affected by childhood adversity.[27] 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."[28] 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.[29] For example, behavioral studies show that, compared to the general population, lower income children and adults tend to have poorer working memory,[30] less inhibitory control, and worse executive attention and function.[31] Other areas of documented deficiency are impairments in spatial and visual cognition, 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)[32] and difficulties with cognitive control.[33] Based on an analysis of these deficits, 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.[34]

Much of the work of Farah and her group, and the research linking brain structure and function to low SES generally, relies heavily on neuroscientific

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25. For a description of fMRI, see A PRIMER OF CRIMINAL LAW AND NEUROSCIENCE, *supra* note 3, at 1, 37–38.

26. *See id.* at 75–76, 78.

27. Farah et al., *supra* note 16, at 166–67.

28. *Id.* at 168.

29. *Id.* at 167.

30. *See generally* Gary W. Evans & Michelle A. Schamberg, *Childhood Poverty, Chronic Stress, and Adult Working Memory*, 106 PROC. NAT'L ACAD. SCI. 6545 (2009).

31. Hackman & Farah, *supra* note 20, at 65; *see* Gary W. Evans, *The Environment of Childhood Poverty*, 59 AM. PSYCHOLOGIST 77 (2004); Gary W. Evans & Jennifer Rosenbaum, *Self-Regulation and the Income-Achievement Gap*, 23 EARLY CHILDHOOD RES. Q. 504 (2008).

32. Farah et al., *supra* note 12, at 279–80; *see also* Kimberly G. Noble et al., *Socioeconomic Background Modulates Cognition–Achievement Relationships in Reading*, 21 COGNITIVE DEV. 349 (2006).

33. Kimberly G. Noble et al., *Socioeconomic Gradients Predict Individual Differences in Neurocognitive Abilities*, 10 DEVELOPMENTAL SCI. 464, 471 (2007).

34. These areas include, inter alia, the prefrontal/executive systems, the lateral cortex/working memory, the anterior cingulate cortex/cognitive control system, and the ventromedial prefrontal cortex/reward processing system. *See* Farah et al., *supra* note 16, at 167; *see also* Hackman & Farah, *supra* note 20, 279–81; Noble et al., *supra* note 33, at 465.

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.[35] 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.[36] 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.[37] Functional MRI (fMRI) depends on measuring three-dimensional hemodynamic (blood flow) patterns associated with variations in brain activity.[38] The 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.[39] Some studies rely on single images of individual subjects whereas others aggregate multiple images from one or more subjects to create a composite picture of activity levels associated with particular conditions or tasks.[40]

Yet another technique, involving a smaller number of studies, makes use of event-related brain potentials (ERPs), which are stylized electrical signals measured at the brain surface.[41] 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, 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 over hundreds or thousands of recordings, gleaned from one or more subjects or trials.[42]

Overall, approximately two dozen studies published since 2001 fall into the category of deprivation neuroscience. This work uses either neuroimaging or ERP techniques to examine the structure or function of the brains of individuals who have experienced some documented form of developmental deprivation, or who suffer impairments or functional abnormalities that are known to be correlated with 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. The second type of research ("prospective") is specifically designed to establish the

35. A PRIMER OF CRIMINAL LAW AND NEUROSCIENCE, *supra* note 3, at 40.

36. *Id.*

37. *See id.* at 40–43 (describing MRI and fMRI techniques in detail).

38. *Id.* at 41.

39. *Id.* at 40–42.

40. *Id.*

41. *See* Shravani Sur & V.K. Sinha, *Event-Related Potential: An Overview*, 18 INDUS. PSYCHIATRY J. 70, 70 (2009).

42. *See* Sandra K. Loo & Scott Makeig, *Clinical Utility of EEG in Attention-Deficit/Hyperactivity Disorder: A Research Update*, 9 NEUROTHERAPEUTICS 569, 570 (2012).

neurofunctional patterns associated with particular behaviors or performance deficits found in lower SES individuals.[43]

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. An example of research providing data for such analyses is a large multisite, multiyear National Institutes of Health MRI imaging study of normal brain development.[44] 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, the studies are useful for investigating the brain correlates of early disadvantage.[45] The goal is to explore correlations between brain structure and functional impairments by investigating 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, and whether those brain changes correspond to functional defects more commonly 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. These reports used an earlier imaging technique, predating fMRI technology, called Positron Emission Tomography (PET). PET provides a measure of oxygen consumption by brain tissue that reflects relative brain function and integrity.[46] 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).[47]

A second retrospective study reviewed scans from 445 American subjects of various ages. It 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). Documentation was established by self-report and objective evidence collected in the study.[48] A third report, by Nicole Hair and her colleagues, was based on an analysis of fMRI scans performed on 389 students of various ages.[49] The authors claimed to establish a three-way statistical correlation between a paucity of gray matter (brain tissue) in various

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43. *See* Hackman et al., *supra* note 9, at 654.

44. *See, e.g.*, Brain Dev. Coop. Grp., *The NIH MRI Study of Normal Brain Development*, 30 NEUROIMAGE 184 (2006) (describing the study); *see also* PEDIATRIC MRI DATA REPOSITORY WEBSITE, https://pediatricmri.nih.gov/nihpd/info/index.html (last visited Mar. 18, 2017) (the project website).

45. *See* Hackman et al., *supra* note 9, at 651–53.

46. *See generally* Harry T. Chugani et al., *Local Brain Functional Activity Following Early Deprivation: A Study of Post-Institutionalized Romanian Orphans*, 14 NEUROIMAGE 1290 (2001).

47. *Id.* at 1299.

48. *See generally* Claudia Buss et al., *Maternal Care Modulates the Relationship Between Prenatal Risk and Hippocampal Volume in Women but Not in Men*, 27 J. NEUROSCIENCE 2592 (2007).

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

parts of the brain, being raised by lower status parents (as defined and documented in the research data), and measured deficits in academic achievement.[50] 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 behaviors later in childhood.[51] Also, 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.[52] Finally, just last year, as widely reported in the press, Kimberly 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 a smaller brain surface area in children between 3 and 20 years old, with severely deprived children showing the most pronounced effects.[53] The observed reductions in surface area were 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.[54] 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).[55]

Instead of mining existing datasets, the prospective studies in the field are specifically geared to collecting new 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. 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 those deficits. 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

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50. *Id.*

51. Jamie L. Hanson et al., *Family Poverty Affects the Rate of Human Infant Brain Growth*, PLOS ONE, Dec. 2013, at 1.

52. *See generally* Gwendolyn M. Lawson et al., *Associations Between Children's Socioeconomic Status and Prefrontal Cortical Thickness*, 16 DEVELOPMENTAL SCI. 641 (2013).

53. Kimberly G. Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 NATURE NEUROSCIENCE 773, 777 (2015).

54. *Id.*

55. *Id.* 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), http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0162511.

to have established a correlation between perceived parental social standing and "greater amygdala reactivity to threatening facial expression."[56] 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.[57] The poorer subjects were also measurably less able than higher SES subjects to suppress amygdala activation during emotional stimulation.[58] 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, especially those with a history of inadequate maternal care. These observations were also correlated with selective behavioral impairments, such as poor self-control.[59]

Likewise, another scanning study of 58 adolescents reported greater cortical bulk 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.[60] Yet another fMRI scanning study, 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.[61] A different prospective study using 49 black middle school children (approximately 14 years old) revealed an association between less early childhood nurturance (ages 4–8) established by questionnaire and a smaller size hippocampus (which is a brain structure involved in memory and emotion) on fMRI.[62] No association was found between scan-documented volumes and other measured aspects of childhood environment.[63] The authors concluded that "hippocampal volume is specifically associated with early parental nurturance."[64] In another dynamic fMRI study of 14 prekindergarten children (age 5) looking at the correlation between brain, behavior, and the environment, 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

56. Peter Gianaros et al., *Potential Neural Embedding of Parental Social Standing*, 3 SOC. COGNITIVE & AFFECTIVE NEUROSCIENCE 91, 91 (2008); *see also* Peter Gianaros et al., *Perigenual Anterior Cingulate Morphology Covaries with Perceived Social Standing*, 2 SOC. COGNITIVE AFFECTIVE NEUROSCIENCE 161, 168–169 (2007).

57. Pilyoung Kim et al., *Effects of Childhood Poverty and Chronic Stress on Emotion Regulatory Brain Function in Adulthood*, 110 PROC. NAT'L ACAD. SCI. 18442, 18442 (2013).

58. *Id.*

59. *See* 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, 1140–41 (2013).

60. Allyson P. Mackey et al., *Neuroanatomical Correlates of the Income-Achievement Gap*, 26 PSYCHOL. SCI. 925, 925 (2015).

61. *See* Noble et al., *supra* note 32; *see also* Kimberly G. Noble et al., *Brain-Behavior Relationships in Reading Acquisition Are Modulated by Socioeconomic Factors*, 9 DEVELOPMENTAL SCI. 642 (2006).

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

63. *Id.* at 1147.

64. *Id.* at 1144.

reading tasks, with reduced lateralization in lower SES students.[65] However, the association between measured reading-related skills (as assessed through a rhyming task), degree of lateralization, and SES in the sample was weak or non-existent.[66] Finally, a research group used MRI scans to investigate the association between SES and brain anatomy in 23 healthy, 10-year-old children with a wide range of parental SES.[67] 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.[68]

Another cluster of studies designed to examine the effects of social disadvantage on the brain makes use of the brain-activity recording, or ERP, technique described above. This approach is largely directed at documenting brain activity patterns associated with verbal abilities, especially oral language comprehension. For instance, D'Angiulli and colleagues examined ERP patterns in 28 children who were instructed to attend to auditory stimuli.[69] 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 disadvantaged children are on average less able to tune out ambient distractions and remain attentive to meaningful verbal material. They speculated that this might help account for their relative deficiencies in reading skills and language comprehension.

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.[70] In a refinement of D'Angiulli's report, their data suggests that a weaker ability to filter out irrelevant auditory information was correlated with lower levels of ma-

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

66. *Id.* at 1396. This pattern led the authors to speculate whether the scans showed "a relationship between SES and language processing that purely behavioural tests are unable to reveal," with the connection between SES and the observed laterality operating via "non-linguistic mechanisms." *Id.* 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." *Id.* at 1392.

67. Katarzyna Jednoróg et al., *The Influence of Socioeconomic Status on Children's Brain Structure*, PLOS ONE, Aug. 2012, at 1, 9.

68. *Id.* 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" as well as "gyrification effects in anterior frontal regions," which was "supportive of a potential developmental lag in lower SES children." *Id.* at 1. They saw "no significant association between SES and white matter architecture." *Id.*

69. *See* Amedeo D'Angiulli et al., *Children's Event-Related Potentials of Auditory Selective Attention Vary with Their Socioeconomic Status*, 22 NEUROPSYCHOLOGY 293, 299 (2008); 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*, 30 PROC. 30TH ANN. CONF. COGNITIVE SCI. SOC'Y 211, 215 (2008);

70. Courtney Stevens et al., *Differences in the Neural Mechanisms of Selective Attention in Children from Different Socioeconomic Backgrounds: An Event-Related Brain Potential Study*, 12 DEVELOPMENTAL SCI. 634, 634 (2009).

ternal education, which was one aspect of disadvantage among those measured.[71] 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.[72] 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.[73] 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."[74]

II. METHODOLOGICAL LIMITATIONS

As the above summary indicates, neuroscientific work designed to investigate the effects of early socioeconomic disadvantage on the brain has yielded about two dozen actual research papers, most published or posted online within the past 10–15 years. These publications contain either a 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.[75] The research is summarized in, and 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.[76]

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71. *Id.* at 640.

72. 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 RES. 55, 64 (2008).

73. *Id.*

74. *Id.*

75. *See generally* Natalie H. Brito & Kimberly G. Noble, *Socioeconomic Status and Structural Brain Development*, FRONTIERS NEUROSCIENCE, Sept. 2014, at 1, https://doi.org/10.3389/fnins.2014.00276. 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,

> "[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."

Council on Cmty. Pediatrics, *Poverty and Child Health in the United States*, PEDIATRICS, Apr. 2016, at 1, 1–2. The statement and accompanying report also discuss a host of health-related ills associated with poverty, noting that "[p]overty has a profound effect on specific circumstances, such as birth weight, infant mortality, language development, chronic illness, environmental exposure, nutrition, and injury." *Id.* at 1.

76. For further review, see, e.g., FROM NEURONS TO NEIGHBORHOODS, *supra* note 5; Farah et al., *supra* note 16; Farah et al., *supra* note 12; Hackman & Farah, *supra* note 20; Hackman et al., *supra* note 9, at 651; *see also* Sara Burr Johnson et al., *State of the Art Review: Poverty and the*

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 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 neuroimaging, and especially the recently refined technique of fMRI, to investigate both functional and morphological aspects of the brain. Although imaging 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 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 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 fMRI scans on children is an arduous and ethically fraught endeavor.[77] 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 Institutional Review Boards (IRBs), which are charged with insuring that the risks of harm are minimized and that benefits for subjects outweigh burdens. IRBs are especially protective of minors, 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,

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Developing Brain, PEDIATRICS, Apr. 2016, at 1; Council on Cmty. Pediatrics, *supra* note 75; *Deepening Connections Between Neuroscience and Public Policy to Understand Poverty*, FAST FOCUS, June 2016, at 1, 1–3, http://www.irp.wisc.edu/publications/fastfocus/pdfs/FF23-2016.pdf. For more popular presentations and summaries, see generally Jim Dwyer, *How Poverty Keeps Hurting Young Minds*, N.Y. TIMES, May 4, 2016, at A15; Daniel R. Taylor, *A Doctor's Call for Action on Childhood Poverty*, PHILA. INQUIRER Apr. 24, 2016, at G01.

77. *See* Andrew Fenton et al., *Ethical Challenges and Interpretive Difficulties with Non-Clinical Applications of Pediatric fMRI*, 9 AM. J. BIOETHICS 3 (2009).

children do not always make cooperative subjects.[78] Getting children to remain immobile in a scanner, or to perform while being scanned, is fraught with difficulties. 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 can therefore be unpredictable, erratic, and difficult to standardize.

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, and researchers use the terms *poverty*, *disadvantage*, *deprivation*, and *low SES* somewhat indiscriminately. As a result, there is no single standardized metric that holds sway in neuroscience or cognate fields, and the dimensions of deprivation actually measured in any given study are often selective and 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 number of 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.[79] The broader point is that the observations of deprivation neuroscience can be no better than the behavioral measures with which they are correlated. If those measures are flawed, imprecise, or irregular, then the brain studies will be unreliable and variable as well. Such limitations are endemic to the field, and are not likely to be corrected anytime soon.

III. GENES VS. 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 sort 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 innate or genetically programmed factors. In fact, as discussed more

78. *See generally* Nora Raschle et al., *Pediatric Neuroimaging in Early Childhood and Infancy: Challenges and Practical Guidelines*, 1252 ANNALS N.Y. ACAD. SCI. 43 (2012).

79. *See, e.g.*, *Deepening Connections Between Neuroscience and Public Policy to Understand Poverty*, *supra* note 76, at 2–3 (noting a lack of uniformity in measures of SES and disagreement among developmental neuroscientists on the dimensions that should be measured).

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 trace observed results to genetic versus environmental influences 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, one potential objection to this line of reasoning is that eliminating early adversity need not be the only way to cure impairments, however caused, that are associated with deprived conditions. Traits or behaviors with a strong genetic basis can sometimes be externally alleviated or corrected. Oft-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.[80] 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 cure the impairments associated with disadvantage, and neither neuroscience nor behavioral science has taught us how to do so.

The discussion in this Part so far assumes a simple conceptual divide between external, or environmental, causes of the brain changes associated with deprivation versus innate, or genetic factors. In fact, this dichotomy is not all-or-none in practice: virtually all 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.[81] The phenotypical modifications of gene

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80. *See, e.g.*, Steven Pinker, *Why Nature & Nurture Won't Go Away*, DAEDALUS, Fall 2004, at 5, 10 (noting that a strict diet can relieve the symptoms and damage from PKU, but only partially).

81. 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 STEVEN PINKER, THE BLANK SLATE (2002); Stephen B. Manuck & Jeanne M. McCaffery, *Gene-Environment Interaction*, 65 ANN. REV. PSYCHOL. 41 (2014); Pinker, *supra* note 80; *Afterward* to STEVEN PINKER, THE BLANK SLATE (forthcoming 2016) (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 10 Replicated Findings from Behavioral Genetics*, 11 PERSP. ON PSYCHOL. SCI. 3 (2016). For specific examples, see, e.g., Avshalom Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated*

expression, which can range from transient to durable, affect human morphology, physiology, and functioning, and are responsible for functional and structural specialization between and within different body organs, including distinct brain centers, both during and after development.[82] 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 expressive possibilities? Answering those questions requires establishing the so-called "norm of reaction," defined as the complete range of phenotypic (physical or behavioral) manifestations observed across the full spectrum of ambient conditions that an organism can encounter.[83]

As applied to the study of social disadvantage, as with any other arena of human behavior, the interaction of genetic with environmental factors is dauntingly complex, with the contribution of genetic endowment to phenotypic variation neither fixed nor necessarily linear. Rather, the interaction is contingent on the environment, or range of environments, the organism encounters. The contribution of ambient factors can run the gamut from "quite a bit" to "hardly at all," depending on the trait at issue and the circumstances in which it 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.[84]

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 influence height will exhibit similar growth in a dry environment.[85] But genetically tall plants will tower over plants with "short" gene variants when provided with water and nutrients.[86] In the same vein, some researchers have claimed that deprivation enhances the environmental contribution to measured intelligence (thereby causing many poor children to fall short of their intellectual potential)

Children, 297 SCIENCE 851, 851–52 (2002); J. Kim-Cohen et al., *MAOA, Maltreatment, and Gene-Environment Interaction Predicting Children's Mental Health: New Evidence and a Meta-Analysis*, 11 MOLECULAR PSYCHIATRY 903, 910 (2006).

82. *See generally* Manuck & McCaffery, *supra* note 81.

83. *Id.* at 58.

84. *See* Pinker, *supra* note 80, at 10.

85. *Id.*

86. *Id.* Pinker observes that "different strains of corn may grow to different heights when equally irrigated, but a plant from the taller strain might end up shorter if it is deprived of water." *Id.* He notes from this and other examples that although "some genetic effects may be nullified in certain environments, not all of them are." *Id.* at 11. 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." *Id.* Pinker also warns against inferring from exceptional extremes that "heredity imposes no constraints on behavior." *Id.* at 10. 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 11.

because it suppresses the expression of genes for high intelligence.[87] 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 gifted to achieve their full "natural" potential).[88] According to a recent study, a similar pattern appears to prevail with body weight.[89] 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. In past decades, in contrast, when food was less available and access more tightly controlled by conventional restraints, body weight was more uniform (and most people thinner) because genetic differences were less often expressed.[90] Once again, as with intelligence, so with obesity: "enrichment" facilitates the expression, hence the dominance, of genetic variations, which produce a pattern of greater individual differences in observed traits.

The factors that determine intelligence and obesity are hotly contested, with no clear consensus on how genes and environment 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 relationships of genes to environment, and genotype to phenotype, are complex and unpredictable. That complexity applies as well to the contribution of environmental and genetic factors to the brain and behavioral disparities associated with SES.

As already noted, scientists have identified behavioral traits that are more frequently observed in the lower SES population, including poor social skills and learning difficulties. It is possible to posit a causal story that attributes each impairment to an array of external forces—ranging from material conditions to interactions with parents to constraints imposed by the broader world. 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. 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, strong cognitive ability, and self-control. Deficits in these areas lead children to make bad choices that add to their own risk of remaining poor.

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87. *See* Erik Turkheimer et al., *Socioeconomic Status Modifies Heritability of IQ in Young Children*, 14 PSYCHOL. SCI. 623, 623 (2003).

88. *Id.*

89. *See* Guang Guo et al., *The Genome-Wide Influence on Human BMI Depends on Physical Activity, Life Course, and Historical Period*, 52 DEMOGRAPHY 1651, 1668 (2015).

90. 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. *See id.*

Variations on these insights are routinely hypothesized and discussed in the vast literature on early disadvantage and what to do about it.[91] But there is also a large body of work that recognizes that innate, genetic factors can shape human behavior.[92] Many of the traits that show deficits among lower SES individuals have been shown to be heritable in varying degrees, and some to a significant extent.[93] These traits frequently influence social skills and interactions, including parenting practices. Indeed, the same 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 complicated combinations 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.[94] 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 these.[95] But the important point is that external forces, including material deprivation, may not be the sole or even the dominant mechanism by which poor parents produce poor children. Rather, adverse genetic traits may be transmitted to offspring directly, independently of inadequate parenting. These can contribute to the behaviors and deficits that impede children's ability to function effectively in society and avoid poverty in adulthood.

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 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 Kimberly Noble, a Columbia University developmental neuroscientist, on the topic of her coauthored 2015 paper in Nature Neuroscience, described above.[96] 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.[97] The thrust of James Thompson's critique of the Noble

91. For a comprehensive review, see, e.g., FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 250–66. *See also* Council on Cmty. Pediatrics, *supra* note 75 (comprehensive statement of the American Academy of Pediatrics, containing an overview of presumed social and environmental causes of the detrimental effects of child poverty).

92. *See, e.g.*, Pinker, *supra* note 80; *Afterward* to THE BLANK SLATE, *supra* note 81.

93. *See, e.g.*, Plomin et al., *supra* note 81 (providing a list of personality and behavioral traits that have been shown to have a large heritable component, and reviewing the literature on behavioral genetic evidence).

94. *See id.* at 11; *see also* FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 268.

95. FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 286.

96. *See* Noble et al., *supra* note 53, at 773; *see also* James Thompson, *Income, Brain, Race, and a Big Gap*, UNZ REV. [PSYCHOL. COMMENTS] (Mar. 31, 2015), http://www.unz.com/jthompson/income-brain-race-and-big-gap/.

97. Noble et al., *supra* note 53, at 777.

study, laid out in a widely read blog called *Psychological Comments*,[98] was directed at remarks made by the authors in both the results and discussion sections of their paper elaborating on the broader social significance of the reported findings.[99]

Although noting the authors' disclaimer that "it is unclear what is driving the links between SES and brain structure," Thompson highlights the papers' uncritical tone and lopsided emphasis on environmental explanatory factors, including "ongoing disparities in postnatal experience or exposures, such as family stress, cognitive stimulation, environmental toxins or nutrition," and "corresponding differences in the prenatal environment."[100] The core of Thompson's objection is that the authors failed to consider the possible role of genetic variation as an explanation for the SES-related differences in brain size and cognitive and behavioral capacities they observed. Thompson states (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."[101] Thompson's complaint, in effect, is that the paper should have at least discussed 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.[102] Rather, a genetic mechanism could be contributing to 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 diminished cognitive capacity) to their children, thus generating the observed correlation between parental poverty and smaller brains in children.[103]

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98. The *Psychological Comments* blog has now migrated to and been absorbed into the Unz Review website. For an archive of James Thompson's blog entries, see *James Thompson Archive*, UNZ REV., http://www.unz.com/author/james-thompson/ (last visited Apr. 24, 2017).

99. Thompson, *supra* note 96.

100. *Id.* (quoting Noble et al., *supra* note 53, at 777). In support of his critique, Dr. Thompson quotes this paragraph from the Noble 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.

Id. Thompson reads these comments as implying "that an 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. *Id.* In other words, the passage strongly implies that the brain size reduction observed among low SES children could be *eliminated* by improving social conditions, which in turn points to an exclusively external cause for the observed deficits.

101. *Id.*

102. *Id.*

103. *Id.*

In fact, as Thompson concedes, Noble and her coauthors do not ignore genes altogether.[104] 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 the paper is somewhat evasive on the reasons for this choice, it does acknowledge that "brain morphology differs, at least subtly, among different ancestry groups."[105] Based on this statement and the background literature, Thompson infers that the authors introduced controls to deal with average racial group differences, observed in previous research and presumably also in Noble's sample, 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 of SES.[106] In commenting on this result, Noble and her coauthors state:

> [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.[107]

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 SES, 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."[108] 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."[109] As Thompson summarizes, "[T]he paper and the comments will lead readers to believe that lack of money is stunting the brains of poorer children. This is possible, but not proved by this study because of obvious genetic confounders. The authors should have made [this] clearer."[110]

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104. *Id.*

105. Noble et al., *supra* note 53, at 774.

106. *Id.* at 776–77.

107. *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." *Id.* at 774.

108. Thompson, *supra* note 96; *see also* James Thompson, *Howitzer or Katyusha: Reply to Prof Noble*, UNZ REV. [PSYCHOL. COMMENTS] (Apr. 19, 2015), http://www.unz.com/jthompson/howitzer-or-katyusha-reply-to-prof-noble/ [hereinafter Thompson, *Howitzer or Katyusha*] (reiterating that "heritability of behaviour and abilities" is a central idea missing from Noble's paper).

109. Thompson, *Howitzer or Katyusha*, *supra* note 108.

110. Thompson, *supra* note 96. 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

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 behavioral problems associated with poverty.[111] 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."[112] 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"[113] 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."[114]

Thompson's argument 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. His point comports with the reasoning, already noted, that establishing causation has important implications for policy. 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.[115] But if those conditions are not important—if size is largely preprogrammed in the brain—

> genetics) that children might inherit genes that made their parents bigger-brained, hence smarter, hence richer.

Afterward to THE BLANK SLATE, *supra* note 81 (manuscript at 3) (quoting Noble's coauthor, Elizabeth Sowell, Dir., Developmental Cognitive Neuroimaging Lab. (News Release, Family Income, Parental Education Related to Brain Structure in Children and Adolescents, CHILD. HOSPITAL L.A. (Mar. 30, 2015), https://secure.chla.org/site/apps/nlnet/content2.aspx?c=ipINKTOAJsG&b=7632571&ct=14553967&notoc=1#.WOVqe2_yuUl)). 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.'" *Id.*

111. Thompson, *supra* note 96.

112. *Id.* (quoting Noble et al., *supra* note 53, at 778).

113. *Id.* (quoting Noble et al., *supra* note 53, at 777).

114. *Id.* (quoting Noble et al., *supra* note 53, at 778). In a similar vein, Noble and her coauthors state in the results section of the paper 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 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.

Noble et al., *supra* note 53, at 775.

115. Thompson, *supra* note 96; *see also supra* pp. 15–16 and *infra* p. 28.

then preventing adverse environmental conditions cannot be assumed to improve outcomes. Until these possibilities are sorted out, 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 potential genetic influences. 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 problems is generally assumed.[116] Some researchers neglect even to acknowledge a possible role for innate influences[117] whereas others mention them only in passing or with brief, boilerplate disclaimers.[118] So, for example, in a paper on the association between SES and performance on memory tasks, the authors state, without further elaboration, that the effects observed "may be due to the heritability of acquired memory ability across SES."[119] 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."[120] 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:

> 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

116. *See, e.g.,* FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 286; Council on Cmty. Pediatrics, *supra* note 75, at 2.

117. *See, e.g.*, Raizada et al., *supra* note 65, at 1392; Stevens et al., *supra* note 70; Stevens et al., *supra* note 72.

118. *See, e.g.*, Mark A. Eckert et al., *Planar Asymmetry Tips the Phonological Playground and Environment Raises the Bar*, 72 CHILD DEV. 988, 997 (2001); Douglas Herrman & Mary Ann Guadagno, *Memory Performance and Socio-Economic Status*, 11 APPLIED COGNITIVE PSYCHOL. 113, 116 (1997).

119. Herrman & Guadagno, *supra* note 118.

120. Eckert et al., *supra* note 118.

> 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.[121]

The secondary literature analyzing 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."[122] The authors provide no citations or support for their assertion that the influence of "experience"—including, presumably, SES—on the traits in question is generally "strong,"—as opposed to modest or weak.[123] Nor do they analyze particular attributes, or the evidence on genetic versus environmental contributions to 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

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121. Thompson, *Howitzer or Katyusha*, *supra* note 108.

122. Hackman & Farah, *supra* note 20, at 69. 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*." *Afterward* to THE BLANK SLATE, *supra* note 81 (manuscript at 9). 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, which is nothing new. 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." *Id.*

Recent 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," notes Pinker, "many people react to the uncovering of mechanisms of gene regulations as it if were a revolutionary discovery that calls for a rethinking of nature and nurture." *Id.* 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. *Id.* at 10. 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. *See id.* But genes and environment have long been known to interact in this way.

123. *See also* Rao et al., *supra* note 62. Rao and his coauthors mention genes as a factor in the observed correlations between maternal nurturance and hippocampal maturation, but suggest that animal cross-fostering studies support relatively weak genetic influences compared to environmental factors. *Id.* at 1149.

answer to the question of "whether and how poverty impacts cognitive development."[124] He nonetheless proceeds to make extensive recommendations for reforming the legal treatment of learning-disabled children, on the assumption that relieving poverty will address the cognitive and learning deficits associated with poverty. But that assumption is dubious if the difficulties low income children experience in the classroom are not chiefly the result of their poverty and its accompanying disadvantages.[125] Yet another comprehensive review acknowledges a range of opinions and estimates on the relative contribution of genes versus environment to "the variability of ability and personality found in the population"[126] and notes that some psychometricians believe "that the developmental consequences of persistent family environmental influences, such as socioeconomic status and parenting, are remarkably small."[127] The review 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.[128]

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This Part has thus far emphasized what researchers 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. But as the analysis above of the Kimberly Noble brain size study reveals, what deprivation neuroscience researchers say about their work is often seriously out of sync with 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 factors or to assign a precise role to each.

In fact, these studies are not designed to accomplish that task. The main reason, as the researchers themselves sometimes acknowledge, is that sorting

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124. James E. Ryan, *Poverty as Disability and the Future of Special Education Law*, 101 GEORGETOWN L.J. 1455, 1480 (2013); *see also* Jennifer H. Suor et al., *Tracing Differential Pathways of Risk: Associations Among Family Adversity, Cortisol, and Cognitive Functioning in Childhood*, 86 CHILD DEV. 1142 (2015); Maggie 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 SOC. COGNITIVE & AFFECTIVE NEUROSCIENCE 499, 499 (2013) (citing literature suggesting a heritable component to behavioral discount rates).

125. For more extensive discussion of the Ryan paper, see *infra* Section IV.B.

126. FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 287.

127. *Id.* at 287–88; *see id.* at 286 (discussing the "parents don't matter" school of behavioral genetics).

128. *Id.* ch. 13 ("Promoting Healthy Development through Intervention") and ch. 14 "Conclusions and Recommendations"); *see also* Council on Cmty. Pediatrics, *supra* note 75.

out these variables presents formidable methodological challenges that the techniques used in the field are simply not equipped to meet.[129] 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 field of behavioral genetics offers 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 are 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.[130] Emblematic of the difficulties of using standard neuroscience techniques for this purpose is James Thompson's suggestion, as part of his critique of Noble's 2015 study as detailed above, that measuring the intelligence or brain morphology, or both, 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 response, Noble defended the failure to collect the suggested data by observing that even though the genetic endowments of parents and children may be correlated, "so are parent-child environments."[131] 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 cognitive functioning, that would still not enable them to make a definitive determination about whether the children's characteristics were mostly controlled by genes or environment. Likewise, Noble noted, "scanning the parents would not solve the problem either, as parental brain morphometry would be both genetically and environmentally influenced as well."[132]

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 were 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

129. Kimberly Noble, *Income, Brain, Race: Prof Kimberly Noble Replies*, UNZ REV. (Apr. 17, 2015), http://www.unz.com/jthompson/income-brain-race-prof-kimberly-noble/ [https://perma.cc/BF5S-K7FZ].

130. *See generally* Hackman, *supra* note 9 (discussing the roles of nature and nurture in the experimental design of deprivation research).

131. Noble, *supra* note 129.

132. *Id.*

small number of subjects. In fact, behavioral geneticists have employed a variety of empirical methods, devised and refined over decades, for teasing out genetic and environmental components for a wide range of traits. The classic "gold standard" approach is to collect data on monozygotic twins who are separated early in life and adopted into different families.[133] Valuable evidence can also be obtained by tracking siblings separated at birth, and comparing them statistically to separated identical twins and unrelated individuals.[134] But natural adoption experiments and analyses of sibling data have always offered only limited promise because of pitfalls inherent in their retrospective design.[135] 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 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).[136]

Lately, rapid developments in genome-wide DNA sequencing have revitalized research in behavioral genetics. 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 can be statistically linked to anatomical and behavioral characteristics observed in specific 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.[137]

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

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133. For studies on twins and antisocial behavior, see generally JOHN C. LOEHLIN & ROBERT C. NICHOLS, HEREDITY, ENVIRONMENT, & PERSONALITY: A STUDY OF 850 SETS OF TWINS (1976); MICHAEL C. NEALE & LON R. CARDON, METHODOLOGIES FOR GENETIC STUDIES OF TWINS AND FAMILIES (1992); Soo Hyun Rhee & Irwin D. Waldman, *Genetic and Environmental Influences on Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies*, 128 PSYCHOL. BULL. 490 (2002); Pinker, *supra* note 80, at 14. Pinker notes that through studies of twins and adoptees, behavioral geneticists have "discovered that in fact virtually all behavioral traits are partly (although never completely) heritable" and cites references. *Id.* at 14, 14 n.24.

134. For a description, see generally Robert Plomin, *Behavioral Genetic Methods*, 54 J. PERSONALITY 226, 239–40 (1986).

135. *See, e.g.*, Jeffrey J. Haugaard & Cindy Hazan, *Adoption as a Natural Experiment*, 15 DEV. & PSYCHOPATHOLOGY 909, 912 (2003) ("Behavior genetics has been criticized on several grounds. . . . [including that] estimates of genetic influence differ, depending on how heterogeneous or homogeneous a sample is. Also, for twins reared apart, it is assumed that their environments are uncorrelated; however, criteria for screening potential adoptive families may result in the twins being adopted by families with similar characteristics, thus making their environments similar along many dimensions.").

136. For a brief discussion on a few of these challenges, see *id.* at 910–13.

137. For a description of genome-wide association (GWA) studies, see generally Dana B Hancock et al., *Meta-Analyses of Genome-Wide Association Studies Identify Multiple Loci Associated with Pulmonary Function*, 42 NATURE GENETICS 45 (2010); M. Luciano et al., *Genome-Wide Scan of IQ Finds Significant Linkage to a Quantitative Trait Locus on 2Q*, 36 BEHAV. GENETICS 45, 45 (2006); Teri A. Manolio, *Genomewide Association Studies and Assessment of the Risk of Disease*, 363 NEW ENG. J. MED. 166, 166 (2010); Cornelius A. Rietveld et al., *Common Genetic Variants Associated with Cognitive Performance Identified Using the Proxy-Phenotype Method*, 111 PROC. NAT'L ACAD. SCI. 13790, 13791 (2014); Austin L. Hughes, *Me, My Genome, and 23andMe*, 40 NEW ATLANTIS, Fall 2013 at 3, 9.

the purported effects of deprivation, which are the focus of discussions of the social implications of deprivation neuroscience. 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 that happen to be 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 efficacy of interventions (thereby obviating the need to determine external versus genetic causation directly) is to conduct controlled prospective clinical trials. In fact, in her dialogue with James Thompson, Kimberly Noble suggested as much by stating that "the bottom line is that, to truly establish the direction of causality, we need a random experiment."[138] She announced 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."[139] 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."[140] In other words, if the children of poor families receiving cash transfers show significantly larger brains and better cognitive functioning than SES-matched controls who do not receive the 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 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 Kimberly Noble) may offer the potential to answer some discrete policy questions.

Unfortunately, many aspects of low SES resist correction.[141] The contributors to disadvantage are complex, poorly understood, and difficult or unethical to manipulate. Children cannot be assigned to different families, home environments, cultures, neighborhoods, or parents.[142] Given these realities, researchers will never be able to provide complete answers to most of the crucial questions, including whether and to what extent parenting style directly causes, or is

138. Noble, *supra* note 129.

139. *Id.*

140. *Id.*

141. *See, e.g.*, Sebastiãn J. Lipina & M. Soledad Segretin, *Strengths and Weakness of Neuroscientific Investigations of Childhood Poverty: Future Directions*, 9 FRONTIERS HUM. NEUROSCIENCE, 2015, at 1, 3–4.

142. FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 342–44.

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 may well resist researchers' efforts to modify them.[143]

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.[144] 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, yielding results that are ambiguous, unreliable, or simply unrevealing.[145] Although the expansion of genome-wide analysis offers great promise for identifying the genetic basis of common human traits and variations, that promise lies largely in the future.[146] The main point, though, is that the studies that will end up illuminating the origins of the biology and behavior correlated with SES represent an extension of the large and growing body of work generated by behavioral genetics. This research stands outside the ambit of deprivation neuroscience as currently practiced.

IV. PRACTICAL IMPLICATIONS

As already noted, the relative contribution of innate versus environmental factors to any behavioral deficit associated with a deprived upbringing has important implications for public policies and interventions designed to help the disadvantaged. As the Thompson critique of the Noble study suggests, a significant role for genetic transmission in producing deficits associated with lower SES implies that the behavioral and brain effects of external factors like poverty might be modest; and therefore, efforts to enhance poor children's performance by improving their circumstances might show little effect.

To be sure, the existence of a heritable component does not rule out alleviation through improved environmental conditions. Because genetic tendencies and environment interact, enriching children's circumstances 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 ease 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 address the associated behavioral difficulties, and policies that are designed to improve on developmental conditions may have a modest or negligible effect. As noted, the vast majority of suggestions and recommendations that make reference to deprivation neuroscience are geared towards preventing and improving on the circumstances that supposedly cause harm in the first

143. *Id.* at 286.
144. Lipina & Segretin, *supra* note 141.
145. Manolio, *supra* note 137, at 173–74.
146. Hughes, *supra* note 137, at 3.

place. On that score, the state of the science, and its inability to identify causal mechanisms, counsels a noncommittal, 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.[147]

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 adversity. 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 the process of connecting external interventions to the kinds of behavioral improvements that policymakers are seeking.

In the absence of techniques to manipulate brains directly, we are thrown back on trying to control the ambient social circumstances that produce or perpetuate disadvantage, or to create the real-life experiences that can cure its negative effects. Unfortunately, even if brain science can help identify the complex social inputs that harm or benefit the brain, it can tell us nothing about how to prevent or produce them. 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 coupled with the generic understanding, long predating recent neuroscientific work, that distinct brain states underlie different observed behaviors. Nor can brain science

147. *See* AMY L. WAX, RACE, WRONGS, AND REMEDIES 22–23, 25, 26–27, 117 (Peter Berkowitz & Tod Lindberg eds., 2009) (discussing the "myth of reverse causation").

tell us how to bring about the changes in society that will improve conditions for the poor.

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. Likewise, 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. And the recommendations that commentators purport to derive from deprivation neuroscience could be—and have been—put forward as plausibly without the benefit of that body of work.[148] Indeed, the agenda commentators favor, which stresses governmentally funded and coordinated services and programs, is no more consistent with, or compelled by, existing studies than alternative, more traditional, approaches to the social problem of poverty.

A. Addressing Poverty and Its Effects

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. The secondary literature that seeks to glean recommendations from the science likewise tilts towards assuming the dominance of environmental factors and, thus, finds a broad ambit for programmatic interventions.[149]

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

148. *See, e.g.,* Gregg J. Duncan et al, *Economic Deprivation and Early Childhood Development*, 65 CHILD DEV. 296, 297 (1994).Rosalind Edwards et al., *Brain Science and Early Years Policy: Hopeful Ethos or "Cruel Optimism*," 35 CRITICAL SOC. POL'Y 167, 175–76 (2015).

149. *See, e.g.*, Anna Almendala, *How Poverty Stunts Kids' Brain Development*, HUFFINGTON POST (July 24, 2015, 12:10 PM), http://www.huffingtonpost.com/entry/how-poverty-stunts-childrens-brain-development_us_55b13476e4b08f57d5d3f990 (discussing the study published by Hair et al., *supra* note 49); Erika Hayakashi, *How Poverty Affects the Brain*, NEWSWEEK (Aug. 25, 2016, 7:10 AM), http://www.newsweek.com/2016/09/02/how-poverty-affects-brains-493239.html (discussing the studies by Hair et al., *supra* note 49 and Noble et al., *supra* note 53); Andrew M. Seaman, *Poverty Might Be Wrecking Brain Development in Some Children*, BUS. INSIDER (July 22, 2015, 10:51 AM), http://www.businessinsider.com/r-effect-of-poverty-on-brains-may-explain-poor-kids-lower-test-scores-2015-7 (discussing the study published by Hair et al., *supra* note 49).

interventions."[150] A different research study expressed the hope (as yet unrealized) of discovering an effective "pharmacological intervention,"[151] while another offered a general reassurance that none of the findings reported imply immutability, or resistance to effective correctives.[152] Yet another report touted programmatic efforts designed to "enhance cognitive stimulation,"[153] whereas two others advance the general assertion that developmental neuroscience holds promise for redesigning public and educational programs for the disadvantaged.[154]

In a similar vein, 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."[155] 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.[156] 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 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."[157] Another group touts a cluster of small, intensive,

150. Kim et al., *supra* note 57, 18445.

151. *See* Van Goozen et al., *supra* note 10, at 227.

152. Hackman & Farah, *supra* note 20, at 71; *see also* Lawson et al., *supra* note 52, at 649 (noting that the prefrontal cortical thickness disparities by SES observed in the study "do[] not in any way imply that these SES differences are innate or unchangeable"); Steinberg, *supra* note 4, at 72 (seeking to dispel the notion of "differences in brain structure or function as conclusive evidence that the relevant behaviors must therefore be hard-wired").

153. Hackman et al., *supra* note 9, at 655; Helen Neville et al., *Commentary: Neurocognitive Consequences of Socioeconomic Disparities*, 16 DEVELOPMENTAL SCI. 708, 708 (2013).

154. *See* Mackey et al., *supra* note 60; Bruce S. McEwen & Peter J. Gianaros, *Central Role of the Brain in Stress and Adaptation: Links to Socioeconomic Status, Health, and Disease*, 1186 ANNALS N.Y. ACAD. SCI. 190, 210 (2010).

155. Farah et al., *supra* note 12, at 280–81.

156. Lipina & Segretin, *supra* note 141, at 3.

157. Luby et al., *supra* note 59, at 1141.

much studied early childhood programs, initiated in the 1970s (the Perry preschool program, the Chicago child-parent study, and the Abecedarian project)[158] that, in long-term follow-up, have produced positive, albeit modest, improvements in rates of employment, criminal offending, and marriage, but not cognitive ability.[159] Other proposals include visiting nurse programs, Moving to Opportunity housing vouchers, intensive cognitive training,[160] and cash transfers to single parents.[161] Perhaps the most specific recommendation comes from the observation by Stevens and her colleagues: underprivileged children are less able to suppress distracting sounds when listening to stories and spoken language.[162] Another study Stevens led developed a training program to counteract that effect, for which there is evidence of limited, short-term success.[163]

The discussions of policy in scientific reviews and the secondary literature tend to make bolder claims and set out more detailed proposals.[164] 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.[165] 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,[166] and providing more generous

158. *See* Pamela Hines et al., *Laying the Foundation for Lifetime Learning*, 333 SCI. 951 (2011) (introduction to a special section: "Investing Early in Education"); Neville et al., *supra* note 153, at 709.

159. Lawrence J. Schweinhart, Benefits, Costs, and Explanation of the High/Scope Perry Preschool Program (Apr. 26, 2003) (unpublished paper presented at the meeting of the Society for Research in Child Development in Tampa, Florida), https://highscope.org/documents/20147/43309/benefits-costs-explanation-perry.pdf/7dd5d9b6-de4b-4171-9a0d-75d7d93f6ec8.

160. Neville et al., *supra* note 153, at 708–09.

161. *See* Noble et al., *supra* note 53, at 778; Greg J. Duncan & Katherine Magnuson, *The Long Reach of Early Childhood Poverty*, PATHWAYS, Winter 2011, at 22, 27; *see also* FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, at 295–96, 418.

162. *See* Stevens et al., *supra* note 70, at 634.

163. Stevens et al., *supra* note 72, at 61.

164. *See* FROM NEURONS TO NEIGHBORHOODS, *supra* note 5, ch. 14; JENSEN & NUTT, *supra* note 4, at 275–77; Clare Huntington, *Neuroscience and the Child Welfare System*, 21 J.L. & POL'Y 37 (2012); Hines et al., *supra* note 158; Neville et al., *supra* note 153, at 709; Ross A. Thompson, *Bridging Developmental Neuroscience and the Law: Child-Caregiver Relationships*, 63 HASTINGS L.J. 1443 (2012); Steinberg, *supra* note 4; Paul Tough, *The Poverty Clinic*, NEW YORKER, Mar. 21, 2011, at 25, 31; 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 Presentation at the Annual Meeting of the Am. Ass'n for the Advancement of Sci. (Feb. 15, 2008), https://www.researchgate.net/publication/237647940_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.

165. FROM NEURONS TO NEIGHBORHOODS, *supra* note 5.

166. *Id.* at 388–92.

funding to "improve the qualifications and increase the compensation" for "children's nonparental caregivers."[167] The authors do not explain how this lengthy wish list is tied to particular neuroscience findings, as opposed to behavioral observations or evidence from social science 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 studies on deprived children's brains.

In the same vein, Helen Neville and colleagues, in deriving lessons from deprivation neuroscience, urge an intensive focus on "developing methods for ameliorating" observed SES disparities.[168] 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."[169] 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.[170] 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 initiatives to enhance language comprehension, reading ability, executive function, memory, and learning generally.[171] Once again, the handful of small, intensive early childhood programs (Perry preschool, Abecedarian, Chicago parent-child initiative) is highlighted.[172] Other proposals include investing in teacher enrichment and quality, creating programs aimed at improving parenting and caregiving, and providing training designed to enhance children's self-control, executive function, memory, basic reasoning, mindfulness, selective attention, and language skills.[173] 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.[174]

In a report on a recent interdisciplinary conference on the policy implications of developmental and deprivation neuroscience, The Institute for Research on Poverty notes that the participants were focused on "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."[175] The document contains many caveats and bemoans the lack of standardization in the "norms regarding what types of social, economic, and

167. *Id.* at 393.
168. Neville et al., *supra* note 153, at 709.
169. *Id.*
170. *Id.*
171. Hines et al., *supra* note 158.
172. *Id.*
173. *Id.*
174. *See id.* and the other articles contained within the special section of the journal issue.
175. *Deepening Connections Between Neuroscience and Public Policy to Understand Poverty*, *supra* note 76, at 1.

demographic data are collected in brain studies and how such factors should be measured."[176] The report expresses the vague hope that "[n]euroscience studies focused on socioeconomic questions could offer the advantage of helping policymakers design interventions that are better targeted and more cost effective," and that "neuroscience may lead to better insights about whether an intervention leads to the intended result."[177] Beyond that, the report is short on the specifics of how neuroscience can be put to work to identify interventions that will actually prove effective in reducing the harmful consequences of poverty.

Finally, in a spring 2016 report published 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."[178] 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.[179] The report contained a comprehensive set of recommendations for pediatric practice based on the research findings, including enhanced nutrition efforts, 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," such as "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."[180]

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.[181] These include having primary care providers "evaluate and address social needs such as housing, employment, education and food," as well as "environmental mediators of neurodevelopment" such as "parenting stress, [and] cognitive stimulation."[182] The article goes on to propose that pediatricians participate in projects with child development specialists to provide "parent-child interaction coaching" and to

176. *Id.* at 2.

177. *Id.* 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." *Id.* The report speculates that the "effects of . . . interventions might be captured far earlier using brain scans or other biological measures." *Id.* 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. Thus, any correspondence between brain changes and behavior can only be established, at least initially, through years of observation and follow-up.

178. Council on Cmty. Pediatrics, *supra* note 75, at 1.

179. *Id.*

180. *Id. at* 7; *see also* Johnson et al., *supra* note 76.

181. *See* Johnson et al., *supra* note 76.

182. *Id.* at 12.

"support play and shared reading."[183] The report also urges pediatricians to advocate for "expanding high quality community resources for families, as well as coordinated systems to implement them."[184] It suggests that "pediatricians may serve as ideal advocates for programs and supports that provide financial benefits to poor families," and predicts that these measures will produce improvements "in long-term cognitive and health outcomes."[185]

The article gives few details on how and whether pediatricians' advocacy will actually change poor children's behavior, brain morphology, or brain functioning. Nor does it provide data on the creation, enactment, and implementation of proposed interventions, or describe the concrete results anticipated 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 connects socioeconomic status to outcomes for disadvantaged children. In sum, the value added by neuroscience, over and above other knowledge and information, is neither explored nor explained, but simply taken for granted.

Law professors have also gotten into the business of citing deprivation neuroscience to justify legal and policy recommendations. In touting a proactive approach to child welfare and arguing against "minimalism" in assisting "at risk" children, 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."[186] 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 "[c]hildren's relationships with their primary caregivers" are "at the core of brain development."[187] When these relationships are deficient, she states, "the developing child's brain is deeply affected."[188] The author concludes that, because the science teaches that the brain changes occasioned by poor caregiving can be hard to reverse, "prevention is essential."[189] While acknowledging that the field of neuroscience may well be too nascent to form the basis of specific proposals,[190] 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."[191] 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

183. *Id.*
184. *Id.*
185. *Id.*
186. Huntington, *supra* note 164, at 40.
187. *Id.*
188. *Id.*
189. *Id.* at 56.
190. *Id.* at 53.
191. *Id.* at 50.

figure for a very young child can be devastating."[192] Thus, "the child welfare system should seek to treat the whole family."[193]

Once again, missing from this exposition is an explanation of how particular neuroscientific 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 has tried his hand at exploring the practical payoffs from the emerging body of developmental neuroscience.[194] In a lengthy article on how "understanding . . . the biological foundations of human development" has "implications for legal analysis,"[195] Thompson recommends a set of initiatives that he claims grows 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, the significance of caregiving quality for buffering stress, and the enduring consequences of early adversity."[196] According to Thompson, the evidence of "brain plasticity" carries "the most important legal implications,"[197] because it "accounts for the efficacy of preventative and intervention[al] efforts targeted to children in adversity."[198] 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."[199] 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 targeted programs to address the specific causes of parental inadequacy."[200]

In light of Professor Thompson's sweeping recommendations, two points are worth noting. First, observational studies of behavior, 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,

192. *Id.* at 56.
193. *Id.*
194. Thompson, *supra* note 164.
195. *Id.* at 1443.
196. *Id.*
197. *Id.* at 1456–57.
198. *Id.* at 1443.
199. *Id.* at 1456.
200. *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 "reduce the consequences of exposure for children." *Id.* at 1453–54.

Thompson's proposals echo those repeatedly 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 results to behavioral data demonstrating or predicting the effectiveness of particular programs.[201]

As a general matter, the secondary literature that purports to find unprecedented programmatic insights in developmental neuroscience offers no novel suggestions. Rather, it reiterates recommendations that have long been popular among developmental experts from a range of social science fields in light of long-standing behavioral observations, evidence accumulated over time, or just plain common sense. For example, the eminent economist James Heckman, who studies human capital and skill formation, has long stressed the importance of early intervention to enhance the life-chances 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

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201. 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, which profiles a young pediatrician, Nadine Burke, reports on a study undertaken at Kaiser Permanente in the 1990s, called the Adverse Childhood Experience study. Tough, *supra* note 164, at 25, 27. 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." *Id.* at 29–30. While asserting that knowledge of how adversity "disrupt[s] 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. *Id.* at 31 (quoting Professor Jack P. Shonkoff, Harvard Medical School). Rather, it echoes the general observation, set out by Huntington and Thompson, that "intervening early can improve later outcomes" in health, education, and behavior. *Id.* 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." *Id.*

Another example of claiming policy payoffs from neuroscience is to be found in a *Guardian* U.K. article reporting on the 2015 Noble study shortly after it was published. Ian Sample, *Brain Development in Children Could Be Affected by Poverty, Study Shows*, GUARDIAN (Mar. 30, 2015, 11:13 AM), https://www.theguardian.com/science/2015/mar/30/brain-development-in-children-could-be-affected-by-poverty-study-shows. 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 a low income might affect children's brains." Thomas adds that "[i]f 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." *Id.* He does not elaborate further on how neuroscience, as opposed to other behavioral scientific methods, can best perform this "narrowing down" function. *Id.*

Finally, a short article appearing in May 2016 in the *New York Times* reports on a conference of leading researchers in the field. The attendees rely upon neuroscience studies, data on stress reactions, and the Adverse Childhood Experience study to suggest that the damage to the brains of disadvantaged children can be reversed through "city policies like universal prekindergarten," as well as forming "caring, consistent relationships with adults." Dwyer, *supra* note 76. The article provides no further details of how these specific recommendations follow from particular findings in neuroscience, as opposed to knowledge from social science and empirical observation generally.

on skill and "early inputs strongly affect the productivity of later inputs," interventions designed to alleviate early deprivation provide the most effective results.[202] 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. In fact, and unsurprisingly, brain science simply confirms what we know from behavioral studies. It adds nothing to the empirical social science data already available.

In addition to reiterating proposals predating the research in deprivation neuroscience, the policy schemes that purport to rely on the science are largely oblivious to the peculiar obstacles and pragmatic difficulties of influencing private behavior, especially within families. The vast majority of proposals are geared towards progressive, government-sponsored, collectively coordinated programs. They emphasize top-down initiatives and publicly funded efforts, 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 thinking, including critiques of some forms of governmental aid to the poor as creating perverse incentives, promoting bad habits, and discouraging avenues of self-help that might prove more effective in the long run.[203] For example, there is research suggesting that irregular family forms and arrangements, such as multi partner fertility, stepparent and mixed parentage families, households where children cohabit with nonbiologically related males, and families experiencing serial adult partnerships, pose enhanced risks of child abuse and neglect.[204] 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.[205]

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202. For a summary of James Heckman's work on skill development, see James J. Heckman, *Skill Formation and the Economics of Investing in Disadvantaged Children*, 312 SCIENCE 1900 (2006).

203. *See, e.g.*, CHARLES MURRAY, LOSING GROUND (1984); MYRON MAGNET, THE DREAM AND THE NIGHTMARE (Encounter Books 2000) (1993); WILLIAM VOEGELI, NEVER ENOUGH: AMERICA'S LIMITLESS WELFARE STATE (2d ed. 2012); *see also* INST. FOR FAMILY, CMTY., & OPPORTUNITY, 2016 INDEX OF CULTURE & OPPORTUNITY: THE SOCIAL AND ECONOMIC TRENDS THAT SHAPE AMERICA 47 (Jennifer A. Marshall & Rachel Sheffield eds., 2016) (detailing counterproductive and dependence-promoting aspects of welfare and transfer programs), http://thf_media.s3.amazonaws.com/2016/2016IndexofCultureandOpportunity.pdf.

204. MARTIN DALY & MARGO WILSON, THE TRUTH ABOUT CINDERELLA: A DARWINIAN VIEW OF PARENTAL LOVE 26–36 (1998); Martin Daly & Margo Wilson, *Child Abuse and Other Risks of Not Living with Both Parents*, 6 ETHOLOGY & SOCIOBIOLOGY 197 (1985); Margo I. Wilson, Martin Daly, & Suzanne J. Weghorst, *Household Composition and the Risk of Child Abuse and Neglect*, 12 J. BIOSOCIAL SCI. 333 (1980).

205. *See, e.g.*, Kay S. Hymowitz, Marriage and Caste in America: Separate and Unequal Families in a Post-Marital Age, Heritage Foundation Lectures, Lecture No. 1005 (Mar. 23, 2007), http://www1.heritage.org/research/lecture/marriage-and-caste-in-america-separate-and-unequal-families-in-a-post-marital-age; *see also* W. Bradford Wilcox et al., When Marriage Disappears: The Retreat

In choosing between these alternative approaches 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 strategies that would best alleviate the effects of poverty and attendant adversities, whether over the short or long term. Nor does it tell us whether support for traditional families and established structures is 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, improve things or make them worse, is not information that neuroscience can provide. Neuroscience is completely uninformative and indeterminate on these questions. Yet the implicit assumption behind the articles that prescribe policies based on neuroscience seems 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 about the concrete behavioral and social 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.[206] 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 (IDEA), which mandates individually tailored special education programs for covered students, and protects them from certain disciplinary sanctions.[207] Ryan finds fault with the IDEA definition of covered learning disabilities and the manner in which the definition has been interpreted and applied.[208] His central contention is that recent findings in deprivation neuroscience show that the scope of the statute's coverage should be expanded.[209]

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from Marriage in Middle America, Heritage Found. Lectures, Lecture No. 1179 (Feb. 22, 2011), http://www.heritage.org/research/lecture/2011/02/when-marriage-disappears-the-retreat-from-marriage-in-middle-america.

206. Ryan, *supra* note 124, at 1503.

207. Individuals with Disabilities Education Act (IDEA), 20 U.S.C. §§ 1400–1444 (2012). *See* 20 U.S.C. § 1401(10)(F)(14) (2012).

208. *See* Ryan, *supra* note 124, at 1457–58.

209. *Id.* at 1495.

The IDEA protects children with a "disorder in [one] or more of the basic psychological processes" that interfere with the ability to "listen, think, speak, read, write, spell, or do mathematic calculations."[210] Specified disorders that interfere with learning—such as "brain injury, dyslexia, developmental aphasia"—are deemed covered, but the statute contains discrete exclusions.[211] Individuals are not considered learning disabled if their deficits are "primarily the result . . . of environmental, cultural, or economic disadvantage."[212] Based on the statutory definition and exclusions, and in reliance on the consensus of disability experts, the IDEA's protections have been extended almost exclusively to children who meet the so-called "discrepancy" model of disabled learning.[213] Children conforming to this model characteristically combine an overall normal range intelligence with deficits in selective learning-related abilities (such as reading or receptive language).[214]

Ryan's main target is the exclusion from statutory protection of learning deficits associated with (and presumably caused by) social disadvantage.[215] Ryan's argument centers on a key categorical distinction that, in his view, underwrites the express statutory exclusion of deficits traceable to low SES.[216] 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.[217] 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.[218] This divide, he argues, can be gleaned from the included and excluded categories, in conjunction with "neighboring language in the statute."[219] As Ryan sees it, "[t]here also appears to be universal agreement among commentators"[220] that "real" learning disabilities are "an *internal* disorder, innate to the students."[221] The operating assumption is that these types of deficits are rooted in purely biological or neurological defects that are the result of aberrant genes or genetic accidents.[222] In other words, disabled students are "born that way." They do not "become that way" through the agency of external conditions such as poverty and disadvantage.

Ryan draws on the findings of deprivation neuroscience to argue that the exclusion for learning deficits traceable to external factors such as poverty and

210. 20 U.S.C. § 1401 (30) (A).
211. *Id.* § 1401 (B), (C).
212. Ryan, *supra* note 124, at 1457 (quoting 20 U.S.C. § 1401 (30) (C)).
213. *Id.*
214. *See id.* at 1467–68.
215. *Id.* at 1457, 1470–71
216. *Id.* at 1470–72.
217. *Id.* at 1458.
218. *Id.* at 1458, 1466.
219. *Id.* at 1458.
220. *Id.* at 1458 n.14.
221. *Id*. at 1458.
222. *Id.* at 1464, 1480–81.

disadvantage, as opposed to intrinsic defects, is unjustified and incoherent, and that socially acquired difficulties should be treated the same as learning problems stemming from other (purely "innate") causes.[223] The neuroscience, he claims, shows that "a child's environment,"[224] including low SES, alters brain development, structure, and function in "physical mechanistic ways."[225] Those effects, argues Ryan, are "no less concrete and real,"[226] and of no lesser concern for the educational fate and functioning of 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.[227]

Central to Ryan's thesis is his "challenge [to the] the notion that there is a sharp divide between internal and external causes of learning disabilities."[228] 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.[229] Because, Ryan contends, there is now "scientific" proof that poverty alters the brain, and accumulating evidence on how that process occurs, 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 or accompanying behavior at issue, or both. On this meaning, an "innate" brain state is one attributed to purely genetic or inherited factors (and thus independent of external and environmental influence).[230] 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.[231] In this sense, all behaviors are ultimately "innate" in that they all are grounded in a corresponding, specific brain state.

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223. *Id*. at 1458.

224. *Id.* at 1491.

225. *Id.* (quoting Farah et al., *supra* note 12, at 285).

226. *Id.* (quoting Farah et al., *supra* note 12, at 286).

227. *See id.* In attacking the "discrepancy" model of learning disability (LD), 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." *Id.* at 1472. According to Ryan, many studies have revealed similarities between the two groups across a "host of dimensions, including achievement levels, achievement growth, behavior, response to instruction, '[and] even a broad range of neurobiological factors.'" *Id.* (quoting Jack M. Fletcher et al., *Classification of Learning Disabilities: An Evidence-Based Evaluation*, *in* IDENTIFICATION OF LEARNING DISABILITIES; RESEARCH TO PRACTICE 231 (Reneé Bradley et al. eds., 2002)). These findings provide additional support for abandoning the categorical distinctions, including the exclusion for poverty-related learning problems, which are inscribed in the law. *Id.*

228. *Id.* at 1479.

229. *Id.*

230. *Id.*

231. *Id.* at 1458–59

Ryan's use of the term appears to adopt the latter meaning. His principal point seems 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," because they correspond to an alteration or abnormality in the "internal" state of the brain.[232] 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.[233] The focus should be on the state of the brain that has trouble learning, and not how the brain state came about. Given the purpose and goals of the IDEA, the distinction between "born" and "made" learning deficits, argues Ryan, is arbitrary and unjustified.[234] Therefore, the law should treat them equally.

Ryan acknowledges that his reasoning entails *a reductio*. All learning difficulties attributable to environmental influence, or innate factors, or some combination of these, can be candidates for designation as "disabilities," because all shortcomings would appear to have a distinct basis in the brain, whatever the source.[235] As Ryan puts it (repeating the words of Farah and her colleagues), "any difference in cognitive function whether genetic or environmental in origin, reflects a difference in brain function."[236] 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.[237] 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."[238]

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."[239] Similarly, the failure to enroll in a quality preschool program

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232. *Id.* ("The problem today is that the distinction between internal disorders and external circumstances is increasingly untenable. Research into the impact of poverty on brain development and function suggests that external circumstances, such as living in sustained poverty, can have internal effects. To oversimplify, growing up in poverty can physically affect how a child's brain develops and functions.").

233. *Id.* at 1493.

234. *Id.* at 1495.

235. *See id.* 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 to 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 regime based on internal vs. external causation is dubious.

236. *Id.* at 1497 (quoting Farah et al., *supra* note 16, at 169).

237. *Id.* at 1496.

238. *Id.*

239. *Id.*

can produce a lifelong lag in subsequent academic achievement. So can growing up in a culture that de-emphasizes learning, reading, verbal communication, or analytic thought. At the very least, Ryan's critique threatens to demolish the distinction between the learning disabled and "garden variety slow learners" who were never meant to be covered by the statute.[240] Indeed, if the disabled category rightly includes anyone whose performance falls below a certain threshold, regardless of the reason, the critical question for the law then becomes who is sufficiently impaired to be covered by the IDEA. How far short of normal would qualify for preferential treatment? Would a dramatic expansion in the category of children entitled to the special protections and extra resources fit in with the overall goal of IDEA? It's hard to know because that goal is not well-articulated. At the very least, such an expansion would prove highly disruptive and expensive for school districts already strapped for funds. Ryan gestures towards these dilemmas 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.[241] The specific findings of neuroscience, while supporting the insight, add nothing to it. In particular, they provide no independent rationale for impugning the IDEA's definitional distinctions or for revising the law.[242]

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.[243] Their reasoning relies only peripherally on empirical evidence, and then almost entirely on behavioral studies. The main justifications for their position are normative and pragmatic.[244] According to Kelman and Lester, children the IDEA defines as "learning disabled" should not be singled out for special benefits, entitlements, and protections.[245] Rather, a wide variety of pupils, including the "socioeconomically disadvantaged" and "poor achievers" generally, should receive extra help, protection, and resources.[246] Kelman and Lester attack the existing statutory categories mainly on

240. *Id.* at 1497.
241. *See, e.g.,* KELMAN & LESTER, *supra* note 6, at 156–60.
242. *Id.*
243. *Id.* at 160
244. *See id.* at 156–57.
245. *Id.*
246. *Id.* at 156.

grounds of justice.[247] Based on "competing principles" of redistribution that "demand that we compensate for environmental or both environmental and genetic disadvantages,"[248] the authors deny that learning disability (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.[249] In none of these cases are the students at fault or causally responsible for their difficulties. Rather, they are all victims of bad luck (either from an unfortunate biological endowment or from being born into poverty and disadvantage). Thus, society is obligated to treat them equally by offering them aid and holding them harmless to the extent feasible. Additionally, Kelman and Lester question the evidence that classic learning disabled, or LD, students benefit more from special help than other "slow learners" and "poor readers" without such a diagnosis.[250] 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

247. Kelman and Lester, like Ryan, argue that the categorical distinction between children with a "bona-fide" learning disability 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 difficulties. *Id.* at 29.

248. *Id.* at 156.

249. *See id.* at 195–96. 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 antidiscrimination 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." *Id.* at 197. Children who are merely poor or culturally deprived, in contrast, are not regarded as belonging to a distinct group of social victims, subject to special forms of mistreatment and thus entitled to a higher degree of solicitude.

250. *See id.* at 219; *see also id*. at 33–34, 138. Ryan, for his part, does not completely ignore the question of interventional efficacy. Ryan, *supra* note 124, at 1467. He points out that, in 2004, the IDEA was modified to allocate 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. *Id.* 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. *Id.* at 1476. 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." *Id.*

people who are disadvantaged through no choice or fault of their own.[251] 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. Because these shortcomings are not their fault, why does it matter how they got that way? Moral and normative considerations, and not any information revealed by neuroscience, render the IDEA's entitlements unjust and incoherent.[252]

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 functional or anatomical features 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 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, nor to the question of who should receive its benefits.

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251. For a recent exposition of the luck egalitarian approach to societal compensation and redistribution see Peter Vallentyne, *Brute Luck and Responsibility*, 7 POL. PHIL. & ECON. 57 (2008). *See also Justice and Bad Luck*, STAN. ENCYCLOPEDIA PHIL., https://plato.stanford.edu/entries/justice-bad-luck/ (last updated Apr. 11, 2014).

252. *See* KELMAN & LESTER, *supra* note 6, at 218–26; 194–226. 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 children's learning difficulties, 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 bad luck of adverse 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, and thus the potential for perverse consequences, may differ depending on category and circumstances. *Id.* at 192. On the potential for abuse from classifying children as suffering from mental disabilities, see, e.g., Patricia Wen, *Case Spotlights Disability System*, BOSTON GLOBE (Jan. 17, 2010), http://archive.boston.com/news/local/massachusetts/articles/2010/01/17/case_spotlights_disability_system/?page=full.

V. 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 problems 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 adversity 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 demography.

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 is simply to reaffirm 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 influence 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 activity associated with specific behavioral deficits found more commonly among lower SES individuals. But that association offers little help in formulating effective proposals to reduce 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 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 preventing or 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 will not necessarily bring improvements.[253] 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

253. Thompson, *supra* note 96.

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, stressful neighborhoods, cultural practices, 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 neurofunctional 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 devising 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 consequences. Rather, 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 crafting effective interventions.

Might neuroscience nonetheless offer the promise of a streamlined methodology? Can it get us closer to the answers we need, and do so faster? It has been suggested that, if neuroscience could eventually establish an airtight connection between brain states and designated behavioral effects, neuromorphological or physiological markers might then serve as proxies that can supplant or stand in for behavioral measures.[254] 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.[255] And the link

254. For a discussion of the suggestion, see *Deepening Connections Between Neuroscience and Public Policy to Understand Poverty*, *supra* note 76, at 2.

255. *See, e.g.,* Adelle E. Forth et al., *The Assessment of Psychopathy in Male and Female Noncriminals: Reliability and Validity*, 20 PERSONALITY & INDIVIDUAL DIFFERENCE 531 (1996); Robert D. Hare & Craig S. Neumann, *Psychopathy as a Clinical and Empirical Construct*, 4 ANN. REV. CLINICAL PSYCHOL. 217 (2008); Kent A. Kiehl et al., *Temporal Lobe Abnormalities in*

between the proxy and the behavior is difficult and cumbersome to establish.[256] The complexity of behavior and its circumstances suggests that myriad trials, in a range of situations, would be necessary to connect neural markers to behavior, and would always leave open the possibility of unanticipated circumstances and imperfect prediction.[257] 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 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 has 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 will not help address or 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 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 as yet 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 neurodeprivation 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 point to effective solutions.

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

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Semantic Processing by Criminal Psychopaths as Revealed by Functional Magnetic Resonance Imaging, 130 PSYCHIATRY RES.: NEUROIMAGING 297 (2004).

256. Kiehl et al., *supra* note 255, at 305.

257. *Id.*

most promising avenue for disentangling causal mechanisms and identifying effective interventions. But the practical ambit of such trials is limited. 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.

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 behavioral and social science. Designing policies depends on real-world constraints and behaviors, and the feasibility—ethical, practical, and political—of their manipulation. Neuroscience offers no help with this project.