

THE BIOLOGY OF INEQUALITY

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ABSTRACT

We have known for quite some time that disadvantaged individuals suffer from poorer health outcomes and lower life spans than the advantaged. The disadvantaged do not perform as well on educational tests than their wealthier peers. In some situations, racial discrimination intersects with poverty to worsen these outcomes for minorities. With the notion that poverty becomes implanted in an individual's genes and brain, science helps explain how these disparate lifespans and variations in cognitive outcomes come to be. This Article collectively refers to these scientific theories as embodied inequality. Embodied inequality explains why it is so difficult for individuals to escape the effects of socioeconomic disadvantage.

Rhetorically, embodied inequality challenges traditional narratives that assume that individual genes and individual behavioral choices are the primary causal agents for social outcomes. Individual action plays a role, but biologists and brain scientists now understand that the environment, along with one's genes, pulls many of the strings toward particular social outcomes. While social-policy theorists have long advocated for government intervention to create a more robust social safety net and a more nurturing society, this Article is the first to apply these emerging scientific theories to these legal and policy issues.

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INTRODUCTION

Inequality has life or death consequences.¹ Despite marked advances in science and medicine, disadvantaged people live shorter lives and suffer from worse mental and physical health than more advantaged individuals.² As this Article shows, this disparity stems not from hunger or other physical forms of deprivation but from the experience of living in stressful disadvantaged environments with little social security and control over one's individual circumstances.

In 1969, Johan Galtung proposed the concept of “structural violence” to explain how bureaucratic and political forces sometimes fail to prevent a preventable death.³ As an example, Galtung explained that people continued to die from tuberculosis even though modern medicine could easily prevent deaths from this disease.⁴ In this instance, death happened because

1. See James Banks et al., *Disease and Disadvantage in the United States and in England*, 295 JAMA 2037, 2037 (2006) (stating that disparities in health outcomes are the greatest at the lowest end of the socioeconomic spectrum); Daniel A. Hackman et al., *Socioeconomic Status and the Brain: Mechanistic Insights from Human and Animal Research*, 11 NATURE REVIEWS 651, 651 (2010) (“Growing up in a family with low SES [Social Economic Status] is associated with substantially worse health and impaired psychological well-being”); 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, 190 (2010) (explaining that stressful experiences can produce a maladaptive effect that leads to “interacting behavioral, cognitive, physiological, and neural changes that promote vulnerability to ill health.”). When race intersects with socioeconomic forces, there are also negative results. See David H. Chae et al., *Discrimination, Racial Bias, and Telomere Length in African-American Men*, 46 AM. J. PREVENTIVE MED. 103, 103 (2014) (“Black American men experience aging-related diseases earlier in life and suffer greater severity and worse consequences of disease compared to other groups.”); Christopher W. Kuzawa & Elizabeth Sweet, *Epigenetics and the Embodiment of Race: Developmental Origins of US Racial Disparities in Cardiovascular Health*, 21 AM. J. HUM. BIOLOGY 2, 2 (2008) (explaining that Black Americans, in general, suffer from cardiovascular disease at a much higher rate than the rest of the population).

2. Michael Marmot, *Status Syndrome*, SIGNIFICANCE, Dec. 2004, at 150, 150 [hereinafter Marmot, *Status Syndrome*].

3. Johan Galtung, *Violence, Peace, and Peace Research*, 6 J. PEACE RES. 167, 170–71 (1969). Galtung uses the term “structural” in a sociological context. In this context, the term (structural and its nominalized form, structuralism) is generally concerned with studying the organizational forms that emerge from human interactions. See Bruce H. Mayhew, *Structuralism Versus Individualism: Part I, Shadowboxing in the Dark*, 59 SOC. FORCES 335, 335–39 (1980); John W. Mohr, *Introduction: Structures, Institutions, and Cultural Analysis*, 27 POETICS 57, 57 (2000) (explaining that structuralists “are concern[ed] with identifying deeper, underlying . . . patterns that find expression in surface level cultural forms”); Susan Carle, *Structure and Integrity*, 93 CORNELL L. REV. 1311, 1313 (2008). Susan Carle uses the term structural to refer to how social structures determine inequalities of power and resources that can in turn affect how lawyers approach advocacy for their clients.

4. Galtung, *supra* note 3, at 168.

resource-allocation decisions impeded access to modern medicine.⁵ Beginning in the 1960s and 1970s, Michael Marmot found a correlation between status and health outcomes in a British civil-servant hierarchy.⁶ The higher up an employee was on the social ladder, the lower his risk of death. Marmot labeled this phenomenon “status syndrome.”⁷

Structural violence and status syndrome are not just abstract theories. We are now beginning to understand the mechanics of how this happens in the body. Through the mechanism of stress, social and economic inequality produces measurable changes in the human body at the genetic⁸ and synaptic level.⁹ These changes produce negative health outcomes in the form of higher disease rates, shorter life spans, and greater chances for becoming mentally ill.¹⁰ Growing up in a disadvantaged environment correlates with greater social and psychological problems, such as anxiety, impulsiveness, and depressiveness. These issues can exacerbate the cycle of poverty and predispose individuals to make choices that place them within the criminal justice system.¹¹ A disadvantaged environment can also negatively impact cognitive performance, creating a tragic circle as lower cognitive performance creates barriers to education and work, which then obstruct social mobility.¹²

The biological concepts of epigenetics and neuroplasticity shed light on how one’s material environment can get under one’s skin and into one’s genetic and brain pathways. Epigenetics is the study of how environmental stimuli alter the expression of individual genes without modifying the

5. *See id.*

6. Marmot, *Status Syndrome*, *supra* note 2; Michael G. Marmot, *Status Syndrome: A Challenge to Medicine*, 295 JAMA 1304, 1304 (2006) [hereinafter Marmot, *A Challenge to Medicine*].

7. Marmot, *Status Syndrome*, *supra* note 2.

8. Stress impacts the body at the genetic level by altering the way that a gene is expressed. *See* NESSA CAREY, THE EPIGENETICS REVOLUTION: HOW MODERN BIOLOGY IS REWRITING OUR UNDERSTANDING OF GENETICS, DISEASE, AND INHERITANCE 244–45 (2012) (explaining a study wherein brain samples of suicide victims with a history of abuse showed higher levels of methylation than victims with no abusive history); Chris Murgatroyd et al., *Dynamic DNA Methylation Programs Persistent Adverse Effects of Early-Life Stress*, 12 NATURE NEUROSCIENCE 1559, 1559 (2009) (discussing how, for mice, stress impacts an animal’s genes level through the methylation process).

9. Stress impacts the body at the synaptic level in the brain by altering the brain’s structure. *See* Bruce S. McEwen, *Brain on Stress: How the Social Environment Gets Under the Skin*, 109 PROC. NAT’L ACAD. SCI. 17180, 17180 (2012).

10. Johnna R. Swartz et al., *An Epigenetic Mechanism Links Socioeconomic Status to Changes in Depression-Related Brain Function in High-Risk Adolescents*, 22 MOLECULAR PSYCHIATRY 209, 209 (2017); Christian H. Cooper, *Why Poverty Is Like a Disease*, NAUTILUS (Apr. 20, 2017), <http://nautil.us/issue/47/consciousness/why-poverty-is-like-a-disease>; *see also* sources cited *supra* note 1.

11. *See* Hackman et al., *supra* note 1.

12. Brandon Keim, *Poverty Goes Straight to the Brain*, WIRED (Mar. 30, 2009, 2:00 PM), <http://www.wired.com/2009/03/poordevelopment>; *see also* Hackman et al., *supra* note 1, at 652 (explaining the relationship between a child’s low socioeconomic status and lower indications for working memory and cognitive control); Sebastián J. Lipina & Michael I. Posner, *The Impact of Poverty on the Development of Brain Networks*, 6 FRONTIERS HUM. NEUROSCIENCE 1, 4–6 (2012) (explaining that, in the context of brain imaging studies, low socioeconomic status leads to discernible differences in how the brain activates in response to performing attention and reading related tasks).

DNA itself.¹³ In this context of this Article, epigenetics shows how the stress of social inequality alters the body at the genetic level.

Neuroplasticity shows how inequality alters the brain. Neuroplasticity refers to how different external conditions correlate with brain structures that differ in size and composition.¹⁴ Environmental differences can produce long-lasting changes in brain structure. Moreover, one's material environment impacts the amount of energy, or bandwidth, one has to devote to cognition tasks. A frenetic environment full of tasks that must be juggled in the mind creates a drag on the mind's cognitive bandwidth that impacts performance on cognitive tests, casting doubt on the theory that intelligence is a pure product of internalized traits.¹⁵

Embodied inequality¹⁶ is both durable and inheritable, but it is not everlasting. Durability comes from the fact that exposure to stress early in life has long-lasting consequences to endocrinal, hormonal, and metabolic systems.¹⁷ These environmentally mediated biological effects can also be passed down from one generation to the next, in utero through the placenta, through the father's sperm, or through maternal behavior.¹⁸ Although embodied inequality is durable and inheritable, it is also reversible. If the material environment that triggers these changes is altered, the changes can be reversed.¹⁹ In this way, embodied inequality does not lend itself to a rigidly deterministic view of biological outcomes.

While exposure to certain environmental agents, such as environmental toxins and hazardous chemicals, can produce negative impacts on the human body,²⁰ this Article focuses on biological changes mediated by social agents, specifically the relationship between stress and economic inequality. Stress is the underlying mechanism by which poverty can get under the skin and inside the brain. For humans, stress is defined in the scientific literature as involving "early maltreatment, conflict-laden familial

13. CAREY, *supra* note 8, at 7–8; RICHARD C. FRANCIS, EPIGENETICS: HOW ENVIRONMENT SHAPES OUR GENES 28–29 (2001).

14. McEwen, *supra* note 9, at 17180–81; McEwen & Gianaros, *supra* note 1, at 191.

15. See *infra* notes 207–15 and accompanying text.

16. Harvard epidemiologist Nancy Krieger has also used this term to describe how racial "discrimination, as one form of societal injustice, becomes embodied inequality." Nancy Krieger, *Methods for the Scientific Study of Discrimination and Health: An Ecosocial Approach*, 102 AM. J. PUB. HEALTH 936, 936 (2012) [hereinafter Krieger, *An Ecosocial Approach*].

17. See Ian C.G. Weaver et al., *Epigenetic Programming by Maternal Behavior*, 7 NATURE NEUROSCIENCE 847, 847 (2004).

18. See *infra* notes 45–50 and accompanying text; see also CAREY, *supra* note 8, at 103–05; Darlene Francis et al., *Nongenomic Transmission Across Generations of Maternal Behavior and Stress Responses in the Rat*, 286 SCI. 1155, 1158 (1999).

19. See Kuzawa & Sweet, *supra* note 1, at 10 (discussing epigenetics); McEwen & Gianaros, *supra* note 1, at 198–99 (discussing neuroplasticity).

20. See Mark A. Rothstein et al., *The Ghost in Our Genes: Legal and Ethical Implications of Epigenetics*, 19 HEALTH MATRIX 1, 14–15 (2009).

relationships, stressful life events, and adverse physical and social conditions—often occasioned by lower socioeconomic environments.”²¹

However, extreme circumstances are not required for stress to become embedded. Socioeconomic status (SES) is a reliable proxy for the kind of stress that can become embodied.²² Living in poverty, even when it does not involve explicit abuse or trauma, nonetheless creates an especially acute kind of stress because “it unites individual and societal lack of control, creates unpredictable adversity, sets conditions that leave people unable to respond, and creates a [deep] sense of helplessness and despair.”²³ Michael Marmot characterizes stress as “arising from the inability to control our lives, to turn to others when we lose control or to participate fully in all that society has to offer.”²⁴ Embedded stress can derive from such commonplace experiences as a bad marriage or social isolation.²⁵ In this context, there is also an intersectional aspect to stress—racial discrimination functions as a “qualitatively distinct stressor.”²⁶

Finally, as developed in Section II.A. of this Article, the stress of not having control over one’s life is deeply connected to neoliberalist policy. The logic of neoliberalism places each individual in the driver’s seat. There is no justification for a collective safety net—each individual actor is able to make their way in the market, and if they cannot, there is something flawed within them. The experience of working and living in this roiling sea of competition creates, for individuals with little power, the exact kind of randomized stress that becomes biologically embedded.

These new scientific theories challenge the idea that individual characteristics are most responsible for how one’s life turns out. This is just

21. McEwen & Gianaros, *supra* note 1, at 191; *see also* S.J. Lupien et al., *Can Poverty Get Under Your Skin? Basal Cortisol Levels and Cognitive Function in Children from Low and High Socioeconomic Status*, 13 DEV. & PSYCHOPATHOLOGY 653, 655 (2001) (“Stress is generally defined as previous or actual exposure to life events that require adaptation from the individual, or else as a state occurring when an individual perceives that the demands of the environment exceeds his or her ability to cope.” (citations omitted)).

22. *See* Jamie L. Hanson et al., *Association Between Income and the Hippocampus*, 6 PLOS ONE, no. 5, 2011, at 1; 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, 1136 (2013).

23. Daniel H. Lende, *Poverty Poisons the Brain*, 36 ANNALS ANTHROPOLOGICAL PRAC. 183, 196 (2012); *see also* Swartz et al., *supra* note 10 (discussing how specific stressors (childhood abuse) and nonspecific stressors (poverty) are both associated with increased methylation of certain gene promoter parts, which then predict greater risk for mental illness such as depression).

24. Marmot, *Status Syndrome*, *supra* note 2, at 153.

25. FRANCIS, *supra* note 13, at 42.

26. *See* Chae et al., *supra* note 1; *see also* Nancy Krieger et al., *The Unique Impact of Abolition of Jim Crow Laws on Reducing Inequities in Infant Death Rates and Implications for Choice of Comparison Groups in Analyzing Social Determinants of Health*, 103 AM. J. PUB. HEALTH 2234, 2239–41 (2013) [hereinafter Krieger et al., *The Unique Impact of Abolition of Jim Crow Laws*] (reporting on study results indicating that the abolition of Jim Crow segregation produced improvements in infant mortality rates for black Americans); Zoë Carpenter, *What’s Killing America’s Black Infants?*, NATION (Feb. 15, 2017), <https://www.thenation.com/article/whats-killing-americas-black-infants/> (explaining that a number of research studies are pointing to racial discrimination, rather than race itself, as being a factor that explains why black infants die at a much higher rate than white infants).

not the case if one's environment contributes to biological and neurological changes, which in turn produce negative health and cognitive outcomes. "Social selection" is the theory most aligned with an individualistic explanation for life outcomes. Social selection posits that individuals select an environment that most aligns with their innate characteristics and cognitive ability.²⁷ For instance, children who enjoy reading will encourage parents to set up a home environment that supports literacy. Social selection theory puts the individual first.

On the other hand, "social cause" theory holds up the material environment as a causal factor for the negative health and cognitive outcomes experienced by disadvantaged persons.²⁸ If an impoverished and stressful environment changes a person's health for the worse at the epigenetic level and negatively impacts the person's brain pathways, the individual lacks complete control over his or her life destiny. Thus, epigenetics and related theories of neuroplasticity challenge a core narrative of liberal individualism.

Because embodied inequality corroborates a social cause theory—that material conditions (rather than individual choice or innate ability) contribute heavily to outcomes—this lends support for the mobilization of collective policy solutions. Here, the hard science empowers new rhetorical approaches that might reframe legal debates about poverty and inequality. The science turns a rigidly deterministic approach to outcomes (you end up where you end up because of your internal merit and cognitive ability) on its head.²⁹ While social Darwinism supports a view that inherited, predetermined traits will predict where you end up in life,³⁰ the science of embodied inequality challenges that view by recognizing that the material environment plays a causal role in life outcomes.

Moreover, the science behind embodied inequality supports progressive theories such as Professor Martha Fineman's vulnerability theory, which contends that the state should provide a support network for those in our society who lack control over their own circumstances.³¹ We now have new science-based arguments that can be used to challenge a host of neoliberal policies—precarious work structures, work schedules, school discipline, mass incarceration—that, as a whole, remove control and stability from individuals' lives. These scientific theories strengthen the argument that we can and should return to a jurisprudential time when large-scale collective solutions to social problems were both entertained and implemented.

27. See *infra* notes 269–93 and accompanying text.

28. See Hackman et al., *supra* note 1, at 653.

29. See Gregory Claeys, *The "Survival of the Fittest" and the Origins of Social Darwinism*, 61 J. HIST. IDEAS 223, 228 (2000).

30. *Id.* (explaining that a Darwinian view of inheritance is a highly deterministic theory).

31. See Martha Albertson Fineman, *The Vulnerable Subject and the Responsive State*, 60 EMORY L.J. 251, 255–56 (2010).

Part I of this Article explores the science, specifically epigenetics and neuroplasticity, reviewing the theories as they relate to both animals and humans and describing the impact that embodied inequality has on life outcomes.

Part II considers how embodied inequality interacts with both rhetoric and policy. Section II.A. illustrates how these new scientific discoveries can be used to reframe powerfully the individualistic rhetoric surrounding inequality and poverty. Section II.B. develops both small-scale and large-scale prescriptions that, as a whole, might improve individuals' material environment and reduce exposure to toxic stress. Included in this discussion are small- and large-scale initiatives that would shore up social security for those most affected by stressful and uncontrollable material environments.

Then, Part III applies the science to specific areas of the law—constitutional law, workplace law, and public-education law. These new scientific theories can be applied to generate novel constitutional theories concerning equal protection. The biology of inequality is relevant for considering whether being poor equates to being in a suspect class, which would trigger higher levels of scrutiny for government discrimination. The science is also relevant for determining whether or not robust governmental remedies for past discrimination are appropriate, if that discrimination can be biologically traced.

From a more specific standpoint, the science might be applied to reform the legal structures that undergird workplace law and public-education law. In the context of work, more worker protection would provide families and children shelter from the stress of living without control, which would in turn ameliorate many of the biological effects of disadvantage. Public education is relevant to this Article because initiatives that foster stable and integrated public schools correlate with positive collateral effects in the material environment (reduced pockets of concentrated poverty, more residential integration). Good, integrated (racial and socioeconomic) public schools can slow down or halt some of the detrimental biological effects mediated by disadvantaged living situations.

I. THE SCIENCE OF INEQUALITY IN THE BODY AND MIND

This section of the Article describes epigenetic and neuroscientific approaches to social inequality. Section I.A. will first explain epigenetics—how one's material circumstances become embedded in DNA through epigenetic imprinting; how this imprinting impacts the brain's structure and stress reaction system; and how these marks can be passed on to subsequent generations. Section I.A. will also explain the connections between epigenetics, stress, and SES. Then, Section I.B. will address neuroscientific explanations for how material conditions negatively impact the mind and brain. Section I.C. explains the intersectional aspects of

biological inequality, which occur when the double disadvantages of socio-economic and racial discrimination combine to inflict harm on the body and mind. Finally, Section I.D. concludes by explaining why social cause (our circumstances are a controlling factor for social outcomes) trumps social selection (as individuals, we choose the direction of our path) as an explanatory theory for these effects. These new scientific theories tell us that the material world is much more responsible for social outcomes than what is currently contemplated by the ingrained mindset of individualized responsibility.

A. Epigenetics: How Inequality Impacts Genetic Expression

Epigenetics refers to the long-term alteration of DNA via chemical processes, without changing the sequence of DNA itself.³² To understand epigenetics we start with the premise that genes are not naked.³³ They are “clothed” in a variety of chemical supplements.³⁴ These chemical additions change the way that genes are expressed.³⁵ By virtue of these chemical clothes, genes can be turned up or turned down.³⁶ And when a gene is turned up or down, this changes the blueprint that cells follow in reproducing themselves.³⁷ These changes to the body’s instruction manual can have a tremendous impact on cells, and ultimately on the body.³⁸ Sometimes epigenetic changes occur randomly.³⁹ But often, epigenetic changes

32. CAREY, *supra* note 8, at 4; FRANCIS, *supra* note 13, at x; Rothstein et al., *supra* note 20, at 3. The epigenetics takeaway—that we are not the sum and substance of our genes, that genetic determinism is not the only narrative that explains where we end up—has roundly captured the public’s imagination. See Maurizio Meloni & Giuseppe Testa, *Scrutinizing the Epigenetics Revolution*, 9 *BIOsocieties* 431, 432 (2014). Recently, Pulitzer prize winning author, Dr. Siddhartha Mukherjee, wrote a compelling article in *The New Yorker* about epigenetics, which came under heavy criticism. Siddhartha Mukherjee, *Same but Different: How Epigenetics Can Blur the Line Between Nature and Nurture*, *NEW YORKER*, May 2, 2016, at 24, 27–28. The criticism was that the article failed to mention more established genetics knowledge bases, which explain how genes become expressed, notably through the RNA transcription process. See Jerry Coyne, *The New Yorker Screws Up Big Time with Science: Researchers Criticize the Mukherjee Piece on Epigenetics*, WHY EVOLUTION IS TRUE (May 5, 2016, 10:33 AM), <https://whyevolutionistrue.wordpress.com/2016/05/05/the-new-yorker-screws-up-big-time-with-science-researchers-criticize-the-mukherjee-piece-on-epigenetics>; see also Jerry Coyne, *Researchers Criticize the Mukherjee Piece on Epigenetics: Part 2*, WHY EVOLUTION IS TRUE (May 6, 2016, 10:15 AM), <https://whyevolutionistrue.wordpress.com/2016/05/06/researchers-criticize-the-mukherjee-piece-on-epigenetics-part-2>. The critique did not dispute the various studies that are beginning to populate the epigenetics field, but it did take issue with the way that Dr. Mukherjee presented the science to the public in the *The New Yorker* article, more as a proven thing than as a theory that is yet to be fully proven. While a detailed inquiry into the debate is outside the scope of this paper, after reading the numerous studies and articles cited in this Article, epigenetics is more than just a half-baked theory. It behooves us to note that epigenetics is a working scientific theory with much left to be proven.

33. FRANCIS, *supra* note 13, at xi.

34. *Id.*

35. *See id.*

36. *Id.* Another way to think about epigenetics is to think of the DNA as the cell’s hardware and epigenetic processes as the cell’s software operating system. See Rothstein et al., *supra* note 20, at 3.

37. *See* FRANCIS, *supra* note 13, at xi.

38. *See id.*

39. *Id.*

result from environmental stimuli.⁴⁰ This is the socially important part of the science—the environment changes how our genes express themselves, which produces longstanding effects in the body.

Epigenetic changes can also be passed on to subsequent generations. One of the first illustrations of this passing on phenomenon occurred in a longitudinal study of Dutch babies born during a famine that occurred at the end of World War II.⁴¹ Babies in utero during the famine were born with a low birth weight but suffered significantly elevated levels of obesity as they grew up.⁴² They also suffered from a higher risk of metabolic illnesses, such as high blood pressure, coronary heart disease, and type two diabetes. In addition, these babies carried a higher risk for schizophrenia and other psychological disorders.⁴³ The effects of the Dutch famine continued into the third generation, affecting the grandchildren of the mothers who lived through it.⁴⁴ Similar findings occurred in a study linking low birth weight and weight at one year in British men with higher death rates for coronary heart disease.⁴⁵

It is not difficult to see how babies born to mothers suffering from a lack of nutrition would be born underweight, but why would the babies suffer from metabolic syndromes later on in life? The answer may derive from how hormones interact with the DNA of the child in utero, making imprints on the child's gene expression. But there are other more radical theories for how epigenetic changes get passed on to subsequent generations. Are epigenetic changes, which arise during one's lifetime, passed on through the germ line during reproduction? The general consensus would respond "no" to this question because, during reproduction, a DNA "cleansing" occurs that erases any epigenetic changes that arose during an individual's lifetime.⁴⁶ However, some recent studies have found "resistance" to this cleaning process, suggesting that some epigenetic changes, modulated by the environment, are passed on, just like one passes on one's genes.⁴⁷

If epigenetic changes are passed through the germ line, this would present an inheritance theory operating on the short term, as Jean-Baptiste

40. *Id.*

41. *Id.* at 1–2; see also CAREY, *supra* note 8, at 2–4.

42. FRANCIS, *supra* note 13, at 3; see also CAREY, *supra* note 8, at 3–4.

43. FRANCIS, *supra* note 13, at 4.

44. CAREY, *supra* note 8, at 4.

45. D.J.P. Barker et al., *Weight in Infancy and Death from Ischaemic Heart Disease*, 334 LANCET 577, 577 (1989).

46. Jonathan Shaw, *Is Epigenetics Inherited?*, HARV. MAG., May–June 2017, at 13, 14 (“[T]here is no evidence that epigenetic information can survive . . . this biochemical cleansing.”).

47. See Walfred W.C. Tang et al., *A Unique Gene Regulatory Network Resets the Human Germline Epigenome for Development*, 161 CELL 1453, 1454 (2015) (observing some resistance to the blank-slate epigenetic cleaning that takes place in the mammalian germline, which gives rise to the possibility that epigenetic imprints can be passed through to the germline).

Lamarck once proposed.⁴⁸ Lamarck's theory has been, for the most part, disproven by Darwinian evolutionary biology,⁴⁹ but now, epigenetics indicates that single-generation inheritance might be possible. Other explanations hold that epigenetic changes are passed to the child by the mother while the child is in the womb⁵⁰ or through the mother's behavior toward the child while the child is in infancy.⁵¹

For purposes of this Article, it does not matter so much how these changes are passed on. The point is that environmental factors can become embedded and can be transmitted to future generations. The external environment "affects us through our genes, by modulating their activity."⁵² The policy implication is that certain toxic environments create biological hardships that then become intractable as they are picked up by subsequent generations. Whether these genetic modifications are transmitted through the germline, in utero, or through maternal behavior, the bottom line is that one's material environment sticks. Children who are exposed to stress, hunger, or other toxicity do not start off with a clean slate.

In order to understand how epigenetics works, we start with the function of DNA. In broad strokes, DNA is analogous to the blueprint for the cell, delivering instructions for how the cells in our body should replicate and differentiate.⁵³ One strand of the DNA's double helix then serves as a template for mRNA⁵⁴ to use in the creation of protoprotein, which then carries out the rest of the building required for the construction of the cell.⁵⁵ Epigenetics is the process by which chemicals interact with our DNA, altering how our genes are expressed.⁵⁶ Most of the time, epigenetic changes serve a useful purpose, directing our cells (all of which have the same underlying DNA code) to differentiate themselves into skin cells, eye cells, organ cells, etc.⁵⁷ As shown more fully below, however, sometimes external conditions (stress, negative maternal environment, trauma)

48. Michael K. Skinner, *Environmental Epigenetics and a Unified Theory of the Molecular Aspects of Evolution: A Neo-Lamarckian Concept That Facilitates Neo-Darwinian Evolution*, 7 *GENOME BIOLOGY EVOLUTION* 1296, 1298 (2015) (describing the distinctions between Charles Darwin's natural selection model, where genetic changes occur over the long term, and Jean-Baptiste Lamarck's theory, which posited that the environment could directly alter an organism's phenotype in the short term).

49. See CAREY, *supra* note 8, at 99 (noting that Lamarckian inheritance rarely occurs through changes passed through the germline).

50. *Id.* at 103 (discussing nongenomic epigenetic changes that occur in the womb).

51. Bruce S. McEwen, *Understanding the Potency of Stressful Early Life Experiences on Brain and Body Function*, 57 *METABOLISM* 11, 11 (2008) (theorizing that differences in maternal care patterns produce epigenetic changes while the animal is in infancy).

52. FRANCIS, *supra* note 13, at 5.

53. *Id.* at 17; see also CAREY, *supra* note 8, at 43.

54. mRNA stands for "messenger RNA." mRNA is one of the three forms of ribonucleic acid polymers that carry out the cell-constructing and protein-building instructions contained in DNA. See HARVEY F. LODISH ET AL., *MOLECULAR CELL BIOLOGY* § 4.4 (4th ed. 2000).

55. FRANCIS, *supra* note 13, at 17; see CAREY, *supra* note 8, at 45.

56. See CAREY, *supra* note 8, at 7-8.

57. See *id.* at 59.

can produce adaptive epigenetic modifications, which then produce negative health and cognitive consequences.

There are three ways that DNA can be modified in an epigenetic manner. The first involves the chemical methylcytosine interacting with DNA, binding to certain portions of the DNA strand so that genes are either expressed (active) or less expressed (inactive).⁵⁸ When areas of DNA become more “methylated,” the volume of genes is turned down, and the genes are prevented from fully expressing themselves in the cell.⁵⁹ On the other hand, when areas of the DNA are less methylated, or “demethylated,” the genes become more expressed.⁶⁰ Another method of epigenetic change occurs through proteins known as histones, which can bind up the DNA so that the genes in the tightly bound area become less expressed.⁶¹ And finally, epigenetic modifications can occur through RNA interference, where certain RNA molecules (responsible in part for carrying out the DNA’s blueprint instructions) bind back to the DNA, limiting the expression of certain genes.⁶² Most of this Article will focus on the first method of epigenetic change, the methylation and demethylation of DNA strands, as this is the area of research that most relates to how material conditions can get under the skin and impact human development and health.

The early childhood environment—specifically, the quality of maternal care that one gets—modulates gene expression. This hypothesis derives from studies of rats conducted by scientist Michael Meaney and his colleagues.⁶³ In Meaney’s studies, rat pups that were frequently licked and nursed by a mother with an arched back (a comfortable nursing position) exhibited less methylation (known as hypomethylation or demethylation) for the gene promoter responsible for glucocorticoid receptors (GR) in the brain.⁶⁴ When there is less methylation on a gene promoter, this means that the gene is more expressed, giving rise to these rats having more GR receptors in their brain.⁶⁵

In terms of rat anxiety, more GR receptors are a good thing. More GR receptors allow the rat pups to better modulate their hormonal reactions to stress, allowing them to calm down in response to anxiety producing stimuli.⁶⁶ Less GR receptors cause a rat’s hormonal stress-response

58. CAREY, *supra* note 8, at 56–58; FRANCIS, *supra* note 13, at 46; Rothstein et al., *supra* note 20, at 5–6.

59. CAREY, *supra* note 8, at 59.

60. Rothstein et al., *supra* note 20, at 5–6, 12.

61. FRANCIS, *supra* note 13, at 60–61.

62. Rothstein et al., *supra* note 20, at 6.

63. Hackman et al., *supra* note 1, at 654.

64. FRANCIS, *supra* note 13, at 44–45; Weaver et al., *supra* note 17, at 847–48; *see* CAREY, *supra* note 8, at 243.

65. CAREY, *supra* note 8, at 243.

66. *Id.* at 240; FRANCIS, *supra* note 13, at 44–45; Hackman et al., *supra* note 1, at 655; Weaver et al., *supra* note 17.

system to become overheated in response to anxiety producing stimuli, preventing the rats from easily calming down or mellowing out.⁶⁷ Rat pups who received less licking and nursing from their mothers showed increased methylation of the gene responsible for expressing cortisol, producing the overheating effect.⁶⁸ Interestingly, rat pups born to good mothers but fostered by poor mothers underwent the same epigenetic changes as rat pups born to and mothered by poor mothers, ending up with fewer GR receptors and an over-reactive stress system.⁶⁹ These epigenetic changes were a long-term life effect.⁷⁰ Thus, maternal behavior permanently altered the development of the rats' genes; the initial changes were not produced through the germ transmission.⁷¹

Finally, the kind of maternal care received by a rat pup in its first week of life influences the maternal behavior that the rat pup will exhibit upon reaching maturity.⁷² Rat pups who received poor mothering grow up to be poor mothers themselves, who can be predicted to reproduce continuing generations of stressed-out rats.⁷³ Professor Michael Meaney theorized that this has something to do with the receptor for the hormone oxytocin and "forms the basis for the intergenerational transmission of individual differences in stress reactivity."⁷⁴

Thus, research indicates that epigenetic changes can be passed down through maternal behavior.⁷⁵ The research summarized above indicates that maternal behavior produces hormone responses that then become associated with higher or lower methylation patterns on the DNA for certain genes.⁷⁶ These patterns influence behaviors, particularly maternal behavior, which then reproduce the epigenetic changes in subsequent generations. Epigenetic changes might also be passed down to subsequent generations through the placenta or maternal lactation.⁷⁷

Newer research indicates that nongenomic changes, mediated by the environment, might be transmitted through an organism's sperm. While most epigenetic attachments disappear during the production of sperm cells and egg cells (a reprogramming process that provides a clean slate to start with),⁷⁸ newer research indicates that methylation patterns can stay in

67. FRANCIS, *supra* note 13, at 47; see Hackman et al., *supra* note 1, at 654–55.

68. CAREY, *supra* note 8, at 240.

69. Francis et al., *supra* note 18, at 1156; Weaver et al., *supra* note 17.

70. Weaver et al., *supra* note 17, at 852.

71. *Id.* at 847.

72. Michael J. Meaney, *Maternal Care, Gene Expression, and the Transmission of Individual Differences in Stress Reactivity Across Generations*, 24 ANN. REV. NEUROSCIENCE 1161, 1161, 1172 (2001).

73. *Id.*

74. *Id.* at 1161.

75. FRANCIS, *supra* note 13, at xv (discussing the effects of maternal behavior and intrauterine environment).

76. See *supra* notes 61–72 and accompanying text.

77. Kuzawa & Sweet, *supra* note 1, at 7.

78. See CAREY, *supra* note 8, at 103–04; Tang et al., *supra* note 47, at 1453.

the male germline (in sperm cells) and get passed on to children in this way.⁷⁹ In other words, some environmentally induced epigenetic changes may resist the reprogramming process as sperm cells and eggs are produced, causing these changes to persist in offspring for a short time or even transgenerationally.⁸⁰

For instance, scientists have recently found that exposing a mouse to separation and unpredictable maternal stress impacted small noncoding RNA in the animal's sperm.⁸¹ In this study, scientists exposed male mice to unpredictable maternal separation and maternal stress.⁸² For two subsequent generations, these mice exhibited depressive symptoms of behavioral stress as well as metabolic symptoms.⁸³ Progeny of the mice exposed to maternal stress and separation showed distinctive markers in the RNA in their sperm, although these marks disappeared in the third generation.⁸⁴ Further, when wild mice were inseminated with the sperm of mice who had been exposed to early-life trauma, the resulting offspring exhibited depressive behavioral symptoms and metabolic changes, similar to those observed in the mice who suffered the trauma firsthand.⁸⁵

As discussed above,⁸⁶ the means by which these changes are transmitted, particularly in humans, are still very much up for debate. Nonetheless, it remains undisputed that epigenetic and other environmentally modulated changes carry on through to subsequent generations.⁸⁷ It does not matter so much whether the transmission occurs in the womb, through behavioral means in early life, or through the germline. The policy implications are the same—children who are exposed to trauma are at risk and are also likely to pass these toxic biological effects to their offspring.

Although epigenetic changes are durable, a change in the environment can reverse the trend. For instance, when rat pups born to poor moth-

79. Tamara B. Franklin et al., *Epigenetic Transmission of the Impact of Early Stress Across Generations*, 68 *BIOLOGICAL PSYCHIATRY* 408, 413 (2010) (finding that male mice subjected to unpredictable maternal separation suffered from depressive symptoms and showed DNA methylation of several genes in their sperm/germline, indicating that male mice suffering from poor maternal care pass on the epigenetic changes to their offspring).

80. Tang et al., *supra* note 47, at 1465.

81. See Katharina Gapp et al., *Implication of Sperm RNAs in Transgenerational Inheritance of the Effects of Early Trauma in Mice*, 17 *NATURE NEUROSCIENCE* 667, 667 (2014). RNA coding interacts with an animal's genes in a similar way to epigenetic processes, but it is not exactly the same thing as epigenetics. *See id.*

82. *Id.*

83. *Id.* at 667–68. As for the behavioral effects, the mice were more fearful in a maze, spent more time floating in a forced swim test (“a test of behavioral despair”), and gravitated toward the light, when given a choice between light and dark. *Id.* As for the metabolic symptoms, the mice had altered levels of glucose, insulin, and body weight. *Id.* at 668.

84. *Id.* at 669. The scientists theorized that the third generation of mice, which still exhibited the behavioral and metabolic distinctions, could be transmitted through other nongenomic means such as epigenetic modifications. *Id.*

85. *Id.*

86. *See supra* notes 45–50 and accompanying text.

87. *See supra* notes 41–47, 72–85 and accompanying text.

ers are fostered by good mothers, the negative consequences (stress reactivity, etc.) are markedly lessened.⁸⁸ This supports the theory that a change in the environment can ameliorate or reverse the epigenetic changes.⁸⁹ Scientists have also theorized that epigenetic changes might be reversible through drugs or hormone injections.⁹⁰

Meaney's work on rats provides an analogue to human development; specifically, childhood trauma and stress can produce long-term epigenetic changes that might be passed down to subsequent generations.⁹¹ While it is difficult to study gene expressions in the human brain (one needs to be able to perform an autopsy), Meaney conducted one epigenetics study of human brains, looking for differences in gene expression based on the presence of childhood trauma.⁹² Meaney looked at human brain samples from individuals who committed suicide.⁹³ One set of samples consisted of suicide victims who experienced childhood abuse or neglect.⁹⁴ Another set consisted of suicide victims who were not abused or neglected.⁹⁵ Within the samples, Meaney found higher methylation in samples of people who were abused or neglected and lower methylation for those who were not abused.⁹⁶ This led Meaney to theorize that childhood trauma does in fact cause epigenetic changes for the genes responsible for expressing hormonal receptors in the human brain.⁹⁷

Scientist Rachel Yehuda and her colleagues have found evidence of trauma-induced epigenetic changes in Holocaust victims and their offspring.⁹⁸ First, Yehuda and her colleagues found that survivors of the Holocaust had increased methylation and thus less expression for the GR receptor gene, meaning that Holocaust survivors had more stress hormones circulating in their bodies at any given time.⁹⁹ In looking at the offspring of Holocaust survivors, Yehuda and her colleagues found that offspring of Holocaust survivors had less methylation at the same genetic marker.¹⁰⁰ The methylation levels were not altered in a consistent way; they were

88. Meaney, *supra* note 72, at 1171.

89. Kuzawa & Sweet, *supra* note 1, at 11.

90. *See id.*

91. Meaney, *supra* note 72, at 1161–62.

92. CAREY, *supra* note 8; Hackman et al., *supra* note 1, at 655 (citing Patrick O. McGowan et al., *Epigenetic Regulation of the Glucocorticoid Receptor in Human Brain Associates with Childhood Abuse*, 12 NATURE NEUROSCIENCE 342–48 (2009)).

93. CAREY, *supra* note 8, at 244.

94. *Id.* at 245.

95. *Id.*

96. *Id.*

97. *Id.*

98. Rachel Yehuda et al., *Holocaust Exposure Induced Intergenerational Effects on FKBP5 Methylation*, 80 BIOLOGICAL PSYCHIATRY 372, 372 (2016).

99. *Id.*; see also Tori Rodriguez, *Descendants of Holocaust Survivors Have Altered Stress Hormones*, SCL. AM. MIND (Mar. 1, 2015), <http://www.scientificamerican.com/article/descendants-of-holocaust-survivors-have-altered-stress-hormones>.

100. Yehuda et al., *supra* note 98.

higher in the first generation and lower in subsequent generations of Holocaust survivors.¹⁰¹ Nonetheless, the amount of methylation visible in a parent accurately predicted methylation changes in his or her children, at the same genetic marker for the GR receptor gene.¹⁰² Yehuda and her colleagues posited that the differences in methylation could be an adaptive effect; the offspring's lower methylation levels could reflect an adaptive response to increased exposure to stress hormones.¹⁰³

Yehuda and her colleagues have also found that the offspring of Holocaust survivors suffering from post-traumatic stress syndrome (PTSD) exhibited different levels of methylation for the GR promoter gene.¹⁰⁴ (Note that the GR promoter gene is the same gene implicated in Meaney's rat-pup studies.)¹⁰⁵ The children of Holocaust-survivor fathers with PTSD carried a much higher risk for depression.¹⁰⁶ The children of Holocaust-survivor mothers with PTSD carried a higher risk for PTSD.¹⁰⁷ Children of Holocaust survivors sometimes report PTSD-related nightmares, in which they are "chased, persecuted, tortured, or annihilated, as if they [are] re-living the Second World War over and over again. At these times, they suffer from debilitating anxiety and depression which reduce their ability to cope with stress and adversely impact their occupational and social function."¹⁰⁸ Essentially, offspring of Holocaust survivors have inherited the "unconscious minds of their parents."¹⁰⁹ Yehuda and her colleagues are uncertain of how these epigenetic changes are transmitted, but theorized that they are passed down through parental behavior, intrauterine transmission, or the male germ line.¹¹⁰ Walfred Tang and his colleagues theorized that some enduring epigenetic changes, particularly those related to stress and the brain, might come through to subsequent generations when the gene expressors "escape" the reprogramming process that usually occurs at conception.¹¹¹

In humans, an over-reactive stress system, induced epigenetically, correlates with increased risk for deleterious health outcomes.¹¹² A stressful environment produces epigenetic modifications to the receptors for stress hormones, which then cause the body's stress system to overheat

101. *Id.* at 372, 375.

102. *Id.* at 375, 377.

103. *Id.* at 378.

104. Rachel Yehuda et al., *Influences of Maternal and Paternal PTSD on Epigenetic Regulation of the Glucocorticoid Receptor Gene in Holocaust Survivor Offspring*, 171 AM. J. PSYCHIATRY 872, 875 (2014).

105. See CAREY, *supra* note 8, at 243; FRANCIS, *supra* note 13, at 45; Weaver et al., *supra* note 17, at 848.

106. Yehuda et al., *supra* note 104.

107. *Id.*

108. Natan P.F. Kellermann, *Epigenetic Transmission of Holocaust Trauma: Can Nightmares Be Inherited?*, 50 ISR. J. PSYCHIATRY & RELATED SCI. 33, 33 (2013).

109. *Id.*

110. Yehuda et al., *supra* note 98, at 377–78; Yehuda et al., *supra* note 104, at 878–79.

111. Tang et al., *supra* note 47, at 1465–66.

112. FRANCIS, *supra* note 13, at 58–59; Yehuda et al., *supra* note 98.

and retain a high level of stress hormones in the body.¹¹³ Over time, the large amount of stress hormones in the body causes internal organs to become too sensitive and creates a risk for diabetes, obesity, heart disease, and other metabolic diseases.¹¹⁴ Specifically, exposure to repeated stress causes alterations to the body's hypothalamic–pituitary–adrenal (HPA) axis, which regulates stress hormones.¹¹⁵ Activity within the HPA axis can be measured by the amount of cortisol present in the body during the day-time.¹¹⁶

Nutritional deprivation—a kind of stress—is one mechanism that induces epigenetic changes in the human body that can then be transmitted to subsequent generations. For instance, epigenetics helps explain the generational effects observed in data surrounding the Dutch babies born during World War II. Babies exposed to the famine during the second and third trimesters were born with a low birth weight but in adulthood experienced obesity levels roughly twice that of babies born before or after the famine.¹¹⁷ These low-birth-weight babies also had “higher incidence[s] of elevated blood pressure, coronary heart disease, and type [two] diabetes.”¹¹⁸ Finally, the Dutch famine babies themselves gave birth to children with lower birth weights and other ill effects, indicating an intergenerational effect.¹¹⁹ Scientists now believe that epigenetics, changes to the expression of genes as an adaptive mechanism to this deprivation, played a role in these outcomes.¹²⁰

Maternal stress can also transmit changes to the human epigenome. Recall the rats who did not receive high-quality mothering (high quantity of licking and comfortable nursing) and, as a result, developed over-reactive stress systems. When these young rats matured into mothers, their stress system likely contributed to a low-quality mothering style, which reproduced the same deleterious epigenetic changes in their offspring.¹²¹ Analogously, human mothers who live a highly stressful way of life transmit higher stress hormone levels to their children, which then correlates with higher rates of metabolic syndromes, such as obesity and diabetes.¹²²

113. See FRANCIS, *supra* note 13, at 58–59; see also Amy S. Desantis et al., *Developmental Origins of Flatter Cortisol Rhythms: Socioeconomic Status and Adult Cortisol Activity*, 27 AM. J. HUM. BIOLOGY 458, 458 (2015) (theorizing that the hypothalamus-pituitary-adrenal (HPA) axis functions as the medium by which stress gets inside the body and the brain).

114. FRANCIS, *supra* note 13, at 58–59.

115. Sonia J. Lupien, *Effects of Stress Throughout the Lifespan on the Brain, Behavior, and Cognition*, 10 NATURE REVIEWS NEUROSCIENCE 434, 434, 440 (2009).

116. Desantis et al., *supra* note 113.

117. CAREY, *supra* note 8, at 3–4; FRANCIS, *supra* note 13, at 3.

118. FRANCIS, *supra* note 13, at 4.

119. CAREY, *supra* note 8, at 4; MATT RIDLEY, *THE AGILE GENE* 156 (2003).

120. FRANCIS, *supra* note 13, at 6.

121. Francis et al., *supra* note 18, at 1156; Meaney, *supra* note 72, at 1172.

122. FRANCIS, *supra* note 13, at 58–59.

Thus, severe kinds of stress originating in childhood—such as the malnourishment experienced in the Dutch famine,¹²³ the severe trauma experienced during the Holocaust,¹²⁴ and sexual abuse¹²⁵—can trigger epigenetic changes. But evidence also indicates that more mundane forms of stress are correlated with these negative health outcomes.¹²⁶ In childhood, “[p]ersistent emotional neglect, family conflict, and conditions of harsh inconsistent discipline all serve to compromise growth and intellectual development and to increase the risk for adult obesity, depression, and anxiety disorders to a level comparable to that of abuse.”¹²⁷ The forms of stress that can trigger epigenetic changes in childhood may be subtle, such as the stress that derives from minimal parental bonding or cold and distant parent–child relationships.¹²⁸

Biologically, the chronic stress experienced by individuals living in low-SES environments has been found to alter the way that an individual’s HPA axis regulates the stress hormone cortisol.¹²⁹ Individuals exposed to psychosocial stress (from living in an environment of disadvantage, measured by SES) have been found to have altered levels of cortisol in their systems as compared with individuals from more advantaged environments.¹³⁰ Specifically, low-SES individuals have been found to exhibit “flatter” declines in diurnal cortisol levels than individuals with higher SES.¹³¹ A flatter, or less steep, slope of daily cortisol levels is associated with metabolic and cardiovascular problems and mental health problems.¹³² As discussed more fully in Section C of this Part,¹³³ beyond SES, racial discrimination functions as a unique stressor that modulates cortisol levels to produce these same metabolic and cardiovascular health effects.¹³⁴

There are interdigitated connections between stress, epigenetic changes, and SES. “[I]ndividuals from lower [socioeconomic backgrounds] report greater exposure to stressful life events”¹³⁵ This kind

123. *Id.* at 59.

124. *See supra* notes 96–106 and accompanying text.

125. Meaney, *supra* note 72, at 1161 (citations omitted).

126. *Id.* at 1161–62.

127. *Id.* at 1161.

128. *Id.* at 1162.

129. Desantis et al., *supra* note 113, at 458, 464.

130. *Id.* at 458, 464, 466.

131. *Id.* at 464.

132. *Id.*

133. *See infra* Section I.C.

134. Emma K. Adam et al., *Developmental Histories of Perceived Racial Discrimination and Diurnal Cortisol Profiles in Adulthood: A 20-Year Prospective Study*, 62 *PSYCHONEUROENDOCRINOLOGY* 279, 279–80, 288 (2015) (perceived racial discrimination (PRD) is a type of psychosocial stress that can produce a type of psychosocial stress that modulates cortisol levels in a way that leads to negative physical and mental health outcomes).

135. Lupien et al., *supra* note 21.

of embodied stress can originate from poverty and social isolation.¹³⁶ Biological changes can be triggered by the everyday stress that comes from living in a socially striated environment: feeling a chasm between oneself and societal ideals, experiencing social alienation, holding a low opinion of one's self, and struggling with the inability to meet basic goals that further a "good life."¹³⁷ Other biological stress conductors, related to SES, might include "economic hardship, marital strife, . . . a lack of social and economic support,"¹³⁸ as well as harsh or inconsistent parenting styles.¹³⁹ Finally, the stress of poverty might be particularly conducive to becoming embedded. The stress of poverty would include "criminal victimization, community violence, reduced access to medical services, economic hardships, and limited educational and employment opportunities."¹⁴⁰

For quite some time, we have known that human health and developmental outcomes decline as one traverses from the most privileged groups to the least privileged groups.¹⁴¹ But now, we are developing a scientific understanding of the mechanistic processes that are responsible for these SES-linked differential health outcomes.¹⁴² The stressful experiences that can get under the skin and epigenetically alter the expression of one's genes (harsh family environments, deprivation, maternal stress, inconsistent childhood discipline) are more likely to be experienced by the least privileged population groups in society.¹⁴³ The working theory is that impoverished environments function as stimuli that trigger deleterious biological changes in one's genes, which subsist throughout the individual's lifetime and are then passed on to subsequent generations.¹⁴⁴

A key link between SES, stress, and the biological embodiment of one's environment is a lack of control. "[S]tatus is [relevant] to two fundamental human needs: to have control over your own life and to be a full social participant with all that implies about being a recognized member

136. FRANCIS, *supra* note 13, at 42.

137. Lende, *supra* note 23 (citations omitted).

138. Meaney, *supra* note 72, at 1176 (citations omitted).

139. Hackman et al., *supra* note 1, at 654 (discussing harsh and inconsistent discipline); *see also* Ashli J. Sheidow et al., *The Role of Stress Exposure and Family Functioning in Internalizing Outcomes of Urban Families*, 23 J. CHILD & FAM. STUD. 1351, 1352–53 (2014).

140. Sheidow et al., *supra* note 139, at 1351.

141. Raj Chetty et al., *The Association Between Income and Life Expectancy in the United States, 2001-2014*, 315 JAMA 1750, 1762 (2016) (noting that in this exhaustive study of U.S. life spans, results indicated that "life expectancy increased continuously with income"); Clyde Hertzman & Tom Boyce, *How Experience Gets Under the Skin to Create Gradients in Developmental Health*, 31 ANN. REV. PUB. HEALTH 329, 331 (2010) ("[S]ocial subordination, even in the very young, is associated with heightened cardiovascular, autonomic, and adrenocortical responses to stress and with disproportionately higher rates of chronic medical conditions and injuries."); Marmot, *Status Syndrome*, *supra* note 2 ("Health and longevity are intimately related to position in the social hierarchy. The lower the status, the higher risk of illness and death, and consequently the shorter the life expectancy.")

142. Hertzman & Boyce, *supra* note 141, at 330, 337 (linking the theory of epigenetics to disparate health outcomes based on SES).

143. *Id.* at 330–31.

144. *Id.*

of society.”¹⁴⁵ Stress, in both animals and humans, is created by removing control.¹⁴⁶ Control over one’s life connotes a certain amount of predictability and stability. For instance, in a study of male mice, scientists replicated a lack of control by removing the mice from their mother at predictable and unpredictable times.¹⁴⁷ It was only when the mice were removed at unpredictable times that the mice developed depressive behaviors that lasted their lifetime.¹⁴⁸ In theorizing about status syndrome, Michael Marmot argues that the stress that “aris[es] from the inability to control our lives, to turn to others when we lose control” is the kind of stress that becomes biologically embodied.¹⁴⁹ And “low control is more common the lower down the pile you find yourself.”¹⁵⁰ Thus, social inequality produces biological inequality.

Enduring ill health, produced by biological reprogramming and triggered by a hostile social environment, presents a frightening picture of inequality. This is not about having less money and less food. Epigenetics runs contrary to the rugged-individualism narrative that holds that any individual can make their way to a positive outcome through grit and effort. This is not the case if the individual is starting out with a deficit of biological assets. From a moral perspective, the child who falls victim to epigenetic reprogramming is blameless. This blamelessness should be a signal call for the state to step in and assist. As will be discussed more fully in Part II, there is a need to dismantle many of the neoliberal policies that produce the stress and lack of control that have become embodied. New approaches to work, education, and poverty law are needed to counter this disturbing type of inequality.

The next section explains how socioeconomic environments can become embodied in neural pathways, which then negatively influence cognitive outcomes. In addition to the disparate health outcomes that are correlated with SES, which we now know are explainable by epigenetic processes, one’s material environment influences cognition. These theories support the conclusion that intelligence does not derive purely from the individual’s makeup and genetics but is rather mediated by the material and social environment one inhabits.

145. Marmot, *Status Syndrome*, *supra* note 2, at 153.

146. *Id.*

147. Franklin et al., *supra* note 79.

148. *Id.* For instance, when the male mice were unpredictably removed from their mothers and mated with stressful mother mice, the depressive behavioral symptoms were passed to subsequent generations. *Id.*

149. Marmot, *Status Syndrome*, *supra* note 2, at 153.

150. *Id.*

B. Neuroplasticity and Neuroscience: How Inequality Gets into the Brain

The previous section explained how stress-induced epigenetic changes to DNA expression induce bodily illness by modulating the body's stress-reaction system. Extensive release of stress correlates with metabolic problems in "the immune, gastrointestinal, cardiovascular, and reproductive systems."¹⁵¹

This section discusses how stress interacts with the mind and brain. When stressed, an animal's body (including a human's) becomes less able to modulate its response to stress; instead, it unleashes a large mass of stress hormones.¹⁵² This overheated stress feedback loop, over time, can alter the structure of the brain.¹⁵³ A change in brain structure may be implicated in cognitive and neurological deficits.¹⁵⁴ Where early stress is not present, there is a greater capacity for cognitive tasks and learning, and reduced declines in age-related learning and memory deficiencies.¹⁵⁵

Thus, a stressful environment, the type of environment where many disadvantaged families live, can negatively influence an individual's cognitive outcomes. In childhood, socioeconomic disadvantage is associated with lower cognitive outcomes, with children from disadvantaged backgrounds scoring approximately one-half to one full standard deviation below their more advantaged cohorts.¹⁵⁶ These disparities in cognitive outcomes, in turn, "have long-lasting ramifications for physical and mental health."¹⁵⁷

Here, the science suggests that one's environment, mediated by SES, influences everything from an individual's performance of cognitive tasks to the size and shape of specific areas of the brain and the amount of cognitive bandwidth that can be directed toward particular goals.¹⁵⁸ One's socioeconomic environment heavily influences one's cognitive resources,

151. Hertzman & Boyce, *supra* note 141, at 336; *see also* McEwen & Gianaros, *supra* note 1, at 205.

152. *See* Hertzman & Boyce, *supra* note 141, at 336–38.

153. *See* McEwen & Gianaros, *supra* note 1, at 194–95 (describing that one of the ways that the body can become overburdened with stress is by a failure to terminate automatic release of stress hormones in response to stressful situations).

154. *See* Hertzman & Boyce, *supra* note 141.

155. *Id.* at 337.

156. Kimberly G. Noble et al., *Neural Correlates of Socioeconomic Status in the Developing Human Brain*, 15 DEVELOPMENTAL SCI. 516, 516 (2012) [hereinafter Noble et al., *Neural Correlates*]; *see also* 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) ("SES explain[s] 32% of the variance in children's scores on phonological and vocabulary tests.") (citations omitted); 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) ("Even before the first day of kindergarten, a child's academic prospects can be predicted based on characteristics of his or her parents, including their income, occupation, and level of education.") (citations omitted).

157. Noble et al., *Neural Correlates*, *supra* note 156.

158. *See infra* notes 155–224 and accompanying text.

which in turn impact outcomes like reading, school grades, and test scores.¹⁵⁹ Here again, as with epigenetics, the science challenges traditional theories of meritocracy, that one's innate intelligence is the primary predictor of one's life outcomes. The science indicates that rather than being the products of preexisting intelligence and traits, mental and cognitive functioning are the products of the material environment's interaction with the brain and the body.

First, we start with the concept of neuroplasticity. Neuroplasticity holds that the brain is malleable and can be shaped by one's social environment.¹⁶⁰ Growing up in a disadvantaged environment can negatively influence the "structural and functional plasticity of the hippocampus, amygdala, and prefrontal cortex—processes collectively referred to as neuroplasticity."¹⁶¹ It can also produce disparities in the size of brain structures¹⁶² and brain activity.¹⁶³

In the context of this Article, neuroplasticity is related to epigenetics. As explained above, epigenetic variations at the gene level modulate the amount of stress hormones released into the human body. A stressful environment, commonly experienced by low-SES individuals, interferes with the expression of the genes responsible for modulating stress hormones, which can cause an individual's stress-axis system to overheat and flood the body with stress hormones.¹⁶⁴ In turn, too many stress hormones in the body, over a period of time, can impact the structure of the brain, shrinking the hippocampus (critical for memory) and increasing the amygdala (related to processing of fear).¹⁶⁵

For neuroplasticity theory, the external environment is an important causal mechanism for these differences in brain size, structure, and performance. By way of example, higher SES children are exposed to quieter home environments (less noise pollution), which correlates with better working memory ability, which is helpful for completing cognitive tasks related to reading and speaking.¹⁶⁶ Moreover, because higher SES children

159. See Kimberly G. Noble et al., *Brain-Behavior Relationships in Reading Acquisition Are Modulated by Socioeconomic Factors*, 9 DEVELOPMENTAL SCI. 642, 642 (2006) [hereinafter Noble et al., *Brain-Behavior Relationships*] (discussing relationships between socioeconomic factors and academic achievement); see also Raizada et al., *supra* note 156.

160. McEwen & Gianaros, *supra* note 1, at 191.

161. *Id.*

162. See Kimberly G. Noble et al., *Family Income, Parental Education and Brain Structure in Children and Adolescents*, 18 NATURE NEUROSCIENCE 773, 773 (2015) [hereinafter Noble et al., *Family Income*].

163. See Noble et al., *Brain-Behavior Relationships*, *supra* note 159.

164. See *supra* notes 108–29 and accompanying text; see also Margaret A. Sheridan, *The Impact of Social Disparity on Prefrontal Function in Childhood*, 7 PLOS ONE, no. 4, 2012, at 10 (finding more variable amounts of the stress hormone cortisol in low-SES children).

165. Luby et al., *supra* note 22.

166. See Michele Tine, *Working Memory Differences Between Children Living in Rural and Urban Poverty*, 15 J. COGNITION & DEV. 599, 608 (2013).

are exposed to more printed material in the home, a rich environment strengthens the brain's pathways used for language acquisition.¹⁶⁷

Links have been found between SES and the size of structures within the brain and activity levels in the brain, which might explain why different SES groups experience different outcomes in cognitive processing, self-control mechanisms, and responsiveness to emotional cues, all of which are mediated by neurological processes.¹⁶⁸ These cognitive effects produce disparities in cognitive performance,¹⁶⁹ which impact life outcomes because educational achievement, test scores, and other markers of cognitive performance matter so much.¹⁷⁰

The size of structures in the brain is one SES-linked factor that connects the environment to individual cognitive performance. In one study, conducted by neuroscientist Kimberly Noble, differences in income were associated with “large differences in [brain] surface areas, . . . [typically] in [brain] regions [relating to] language, reading, executive functions and spatial skills.”¹⁷¹ Professor Noble found that higher SES subjects tended to have brains with a greater cortical surface area and thickness.¹⁷² In a separate study, Noble found SES and parental education predicted differences in the size of the brain's hippocampus and amygdala.¹⁷³ Similarly, another study found a significant association between childhood SES and hippocampal volumes in the brain, an association that continued into adulthood.¹⁷⁴ Yet another study found that higher SES correlated with more gray matter volume in the brain.¹⁷⁵ (Both the hippocampus and gray matter are vital for performing higher level cognitive tasks.)¹⁷⁶

In addition to supporting the relationship between SES and brain size, several studies lend support to the connection between a child's SES-mediated environment and brain activity relating to language processing,

167. See Noble et al., *Brain-Behavior Relationships*, *supra* note 159.

168. See Noble et al., *Family Income*, *supra* note 162, at 774 (finding a positive relationship between SES and the brain's cortical surface area); Roger T. Staff et al., *Childhood Socioeconomic Status and Adult Brain Size: Childhood Socioeconomic Status Influences Adult Hippocampal Size*, 71 ANNALS NEUROLOGY 653, 653, 657 (2012) (“[A] significant association between childhood SES and hippocampal volume . . .”).

169. Katarzyna Jednoróg et al., *The Influence of Socioeconomic Status on Children's Brain Structure*, 7 PLOS ONE, no. 8, 2012, at 4 (finding SES affects cognitive performance); Noble et al., *Brain-Behavior Relationships*, *supra* note 159 (“SES . . . is a robust predictor of children's reading achievement . . .”) (citations omitted); Noble et al., *Neural Correlates*, *supra* note 156 (“[S]ocioeconomic disadvantage in childhood is associated with negative effects on cognitive and socio-emotional development.”) (citations omitted).

170. See LANI GUINIER & GERALD TORRES, *THE MINER'S CANARY* 22 (2002).

171. Noble et al., *Family Income*, *supra* note 162.

172. *Id.* at 774–75. For study subjects with higher income, income had less of an effect on brain morphology. *Id.* at 773.

173. Noble et al., *Neural Correlates*, *supra* note 156, at 522–23. In this study, lower parental education correlated with a larger amygdala (indicating greater exposure to stress) and a smaller hippocampus (an area of the brain critical for memory). *Id.*

174. Staff et al., *supra* note 168, at 657.

175. Jednoróg et al., *supra* note 169, at 5.

176. See Hanson et al., *supra* note 22, at 4.

attention, and working memory. For instance, a study of neuroimages of children found a correlation between higher SES and more brain activity in the left inferior frontal gyrus, the part of the brain that relates to language.¹⁷⁷ Children with lower SES indicators showed weaker neural specialization, or less activity, in language processing areas of the brain.¹⁷⁸ The authors of this study theorize that a richer linguistic environment in the home (more books and printed matter) could be producing greater volume in the brain's pathways responsible for language processing.¹⁷⁹

SES also correlates with children's ability to marshal cognitive resources related to attention. Attention is a cognitive science concept that refers to the ability to perform tasks that require "filtering distracting information, managing response conflict, and regulating behavior."¹⁸⁰ This study found that children from low-SES backgrounds experienced difficulties with aspects of attention, showing a reduced ability to filter out distracting information.¹⁸¹ These difficulties with attention correlate with performance problems in other areas, such as preliteracy and language acquisition.¹⁸² For children with attention issues, the typical classroom is "poorly suited for learning."¹⁸³ Moreover, children need the ability to focus their attention to learn to read.¹⁸⁴ Thus, these attention problems create a cascade effect that creates an achievement gap between low-SES and high-SES students.¹⁸⁵

Finally, a study from Professor Michelle Tine found connections between a child's working memory and the child's SES-mediated environment.¹⁸⁶ Working memory, which refers to the brain's ability to hold information while completing cognitive tasks,¹⁸⁷ can be visuospatial or verbal.¹⁸⁸ Interestingly, Tine's study uncovered neurological distinctions based on whether the child's environment was a low-income urban versus a low-income rural setting.¹⁸⁹ Children from urban environments showed symmetrical differences in both visuospatial and verbal working memory.¹⁹⁰ In other words, in comparison with their more privileged cohorts, children from lower SES environments had deficits in both visuospatial and verbal working memory.¹⁹¹ Children from low-income rural environments also scored less well on working-memory tasks than

177. Raizada et al., *supra* note 156.

178. *Id.* at 1399.

179. *Id.* at 1398–99.

180. Stevens et al., *supra* note 156, at 635.

181. *Id.* at 635, 640, 642.

182. *Id.* at 636.

183. *Id.* at 643.

184. *Id.*

185. *Id.* at 634.

186. Tine, *supra* note 166, at 599.

187. See Sheridan, *supra* note 164, at 2.

188. Tine, *supra* note 166, at 599.

189. *Id.*

190. *Id.* at 599, 607.

191. *Id.*

their higher income cohorts, but low-income rural children, unlike low-income urban children, had worse visuospatial- than verbal-working-memory scores.¹⁹²

Professor Tine theorized that children in low-income rural environments exhibit stronger verbal working memory but weaker visuospatial memory because children in more rural environments are exposed to less noise pollution.¹⁹³ Noise pollution, because of its negative impact on attention, has been correlated with deficits in verbal working memory.¹⁹⁴ On the other hand, low-income urban children may have higher visuospatial working memory because they are exposed to more everyday visual stimulation, such as “traffic, crowds, commercial, residential, industrial buildings and signs, and opportunities to navigate public transportation systems.”¹⁹⁵ Because the brain is plastic and because the environment plays a key role in the brain’s plastic structure, children who are exposed to less visual stimulation may have more attenuated brain pathways that relate to the performance of visuospatial tasks.¹⁹⁶

In a related sense, the environment, mediated by a higher SES, can help augment a child’s neurological activities, producing better cognitive outcomes for those who reside in more advantaged homes. This theory was borne out in a study conducted by Professor Noble and her colleagues.¹⁹⁷ In this study, scientists viewed neuroimages of children’s brains while they were reading.¹⁹⁸ Professor Noble and her colleagues then looked specifically at the area of the brain associated with phonological awareness (PA).¹⁹⁹ When this area of the brain appeared active in the neuroimages, it indicated that the child possessed a strong cognitive ability for language processing.²⁰⁰ In children with higher SES, sometimes the phonological awareness region was not as active.²⁰¹ Yet, these children still showed a higher reading ability than low-SES children with similar PA activity levels.²⁰² Professor Noble and her colleagues theorized that even with a similar neurological activity, higher SES children enjoy an environment in which they can draw upon alternative modes of literary support that derive from their environment—such as increased exposure to printed materials—which buffers reading skills.²⁰³

192. *Id.*

193. *Id.*

194. *Id.* (citations omitted).

195. *Id.* (citations omitted).

196. *Id.*

197. Noble et al., *Brain-Behavior Relationships*, *supra* note 159.

198. *Id.*

199. *Id.*

200. *Id.* at 642, 650–52.

201. *Id.* at 650–51.

202. *Id.*

203. *Id.* at 651–52.

In addition to the studies of SES and brain development in children, other studies show how SES interacts with adult brains. Specifically, SES-related correlations exist in areas of the brain relating to the processing of fear and stress. For instance, in a neuroimaging study, Peter Gianaros and his colleagues found that, in comparison with higher status individuals, individuals with lower social status exhibited greater amygdala activity in response to being presented with threatening and angry facial expressions.²⁰⁴ The amygdala is responsible for regulating the body's stress response system, so greater activity in the amygdala indicates a more stressful response to threatening stimuli.²⁰⁵ The authors of this study theorized that disadvantaged environments measurably impact brain function, which becomes visible with higher levels of amygdala activity.²⁰⁶

Another of Professor Gianaros's neuroimaging studies indicates that human individuals with a perceived low social standing tend to exhibit reduced amounts of gray matter volume in the perigenual anterior cingulate cortex (pACC).²⁰⁷ Gray matter in the pACC is responsible for processes involving "the appraisal of salient environmental and personal events, the experience of emotional states, and the regulation of behavioral and autonomic responses to emotional and stressful stimuli."²⁰⁸ Less gray matter in this part of the brain could explain adverse mental health outcomes, such as problems with self-control, decision making, and over-reactive emotional responses.²⁰⁹

Professor Gianaros and his colleagues suggested that the stress of living a disadvantaged life could structure the brain (through hormonal interactions) to reduce the amount of gray matter in the pACC.²¹⁰ Alternatively, the study authors posited that individuals with comparatively lesser amounts of gray matter could be predisposed to view themselves in a depressive way, contributing to perceptions of having low social status.²¹¹ Regardless of how the correlation between gray matter and SES perceptions comes about, this is a harmful feedback loop. Individuals with less

204. McEwen & Gianaros, *supra* note 1, at 203 (citing Peter Gros et al., *Potential Neural Embedding of Parental Social Standing*, 3 SOC. COGNITIVE & AFFECTIVE NEUROSCIENCE 91, 91–92 (2008)).

205. *Id.*

206. See 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, 18444 (2013) (finding that adults who grew up in poverty possessed increased amygdala activity, which was likely a result of stress exposure, which "provides evidence of neural embedding of childhood poverty").

207. Peter J. Gianaros et al., *Perigenual Anterior Cingulate Morphology Covaries with Perceived Social Standing*, 2 SOC. COGNITIVE & AFFECTIVE NEUROSCIENCE 161, 166 (2007).

208. McEwen & Gianaros, *supra* note 1, at 205 (citing George Bush et al., *Cognitive and Emotional Influences in Anterior Cingulate Cortex*, 4 TRENDS COGNITIVE SCI. 215, 215 (2000)).

209. See Gianaros et al., *supra* note 207, at 161–62, 170; see also John M. Allman, *The Anterior Cingulate Cortex: The Evolution of an Interface Between Emotion and Cognition*, 935 ANNALS N.Y. ACAD. SCI. 107, 114 (2001) (explaining that the anterior cingulate cortex is involved in regulating self-control, attention, and social maturity).

210. Gianaros et al., *supra* note 207, at 169–70.

211. *Id.* at 170 (citing Ronald A. Cohen et al., *Early Life Stress and Morphometry of the Adult Anterior Cingulate Cortex and Caudate Nuclei*, 59 BIOLOGICAL PSYCHIATRY 975, 978–79 (2006)).

gray matter in the pACC are prone to exhibit maladaptive behavioral and mental reactions,²¹² which would be exacerbated in environments of disadvantage.

Another provocative study developed the theory that persons living in disadvantage do not do as well on cognitive performance test because so much cognitive energy (cognitive bandwidth) is expended to survive in socially stressful scenarios.²¹³ In this study, psychologist Anandi Mani and her colleagues divided their study subjects into groups and noted each subject's SES status.²¹⁴ After considering one of two problems, the study participants were then asked to complete a test designed to assess cognitive control and fluid intelligence.²¹⁵ The first problem involved a car breaking down with repairs that would cost \$150.²¹⁶ The second problem involved car repairs costing \$1,500.²¹⁷ For both lower and higher SES subjects, contemplation of the \$150 car repair problem had no appreciable impact on how the subject performed on the subsequent cognitive test.²¹⁸ But for low-SES subjects who were "primed" with the \$1,500-car-repair problem, their performance was "significantly worse" on both the cognitive-control and fluid-intelligence tests.²¹⁹

Having to "manage sporadic income, juggle expenses, and make difficult tradeoffs" produces an immediate cognitive load that creates burdens in performing any other cognitive tasks.²²⁰ The drag on one's cognitive bandwidth caused by being poor is analogous to losing a full night's sleep, suffering from the effects of chronic alcoholism, or losing 13 IQ points.²²¹ This study confirms the recurring theme of this Article, which is that one's material environment does more than just affect one's financial circumstances. Being poor bleeds into the brain and the mind.²²² Cognitive performance, so often held out as a product of innate individual merit, is not

212. See Gianaros et al., *supra* note 207, at 161–62, 170.

213. Anandi Mani et al., *Poverty Impedes Cognitive Function*, 341 SCIENCE 976, 976 (2013).

214. *Id.* at 977.

215. *Id.*

216. *Id.*

217. *Id.*

218. *Id.*

219. *Id.*

220. *Id.* at 976–77.

221. *Id.* at 980.

222. Dr. Brett Ingram, a scholar who is applying neuroscience to communications explains that: The congruencies between people who have suffered brain injuries, and those who have suffered social marginalization, could be attributable to the fact that, at the microscopic level, emotional distress caused by rhetorical or symbolic affronts to one's social standing is manifested in neurological injuries—a deformation of the neural circuitry with which the brain represents itself to itself that effects what amounts to an ontological change in the subject's conscious thought.

Brett Ingram, *Critical Rhetoric in the Age of Neuroscience* 162 (Feb. 2013) (unpublished Ph.D. dissertation, University of Massachusetts Amherst), http://scholarworks.umass.edu/open_access_dissertations/690.

such an easy story. We must accept that the material environment also shapes these outcomes.

As with epigenetics, SES's negative influence on the brain can be explained as stress related. Stressful experiences can change the structure of the brain. Higher levels of stress, in both animals and humans, correlate with reduced gray matter in the hippocampus and orbital prefrontal cortex area of the brain.²²³ And here, the stress of being disadvantaged is exactly the kind of stress that wreaks problematic changes in the function and shape of the human brain. As Bruce S. McEwen and Peter J. Gianaros write: "early maltreatment, conflict laden familial relationships, stressful life events, and adverse physical and social conditions—often occasioned by lower socioeconomic environments—during development and aging can [negatively] influence the structural and functional plasticity of the hippocampus, amygdala, and prefrontal cortex."²²⁴ In this manner, neuro-anthropologist Daniel Lende writes that "[t]he social organization of inequality happens through how social forces shape our neuroplastic and embodied brains."²²⁵ Lende writes that because of these embodied effects, we cannot and should not reduce poverty to an abstracted problem for social theorists and the political economy.²²⁶ As the studies reviewed in this Article show, "people suffer through their embodied brains, through despair and toxic stress and destructive behavior."²²⁷

The brain-science studies discussed in this Article, taken together, illustrate the deleterious cascade effect that a low-SES environment can have on an individual's cognitive outcomes, which then link up with social outcomes. As with epigenetic changes, environmentally mediated effects on the brain are reversible.²²⁸ The plastic brain can be retrained. It might be possible, for instance, to strengthen the brain pathways of children living in noisy or low-literacy environments through targeted brain training.²²⁹ However, we should not let the potential for individual brain retraining distract us from contemplating the embedded *structural* reasons that individuals from lower SES environments are burdened with weaker brain pathways, heavier cognitive loads, and more toxic stress. While the theory of neuroplasticity can be deployed to celebrate a neoliberal vision of the individual subject,²³⁰ it also supports the conclusion that the material conditions that one finds oneself in, starting in childhood, deeply influence the neural pathways responsible for cognitive performance and, in turn,

223. McEwen & Gianaros, *supra* note 1, at 199.

224. *Id.* at 191.

225. Lende, *supra* note 23, at 197.

226. *See id.* at 198–99.

227. *Id.*

228. *See* McEwen & Gianaros, *supra* note 1, at 196, 198.

229. Stevens et al., *supra* note 156, at 634, 636.

230. *See* Victoria Pitts-Taylor, *The Plastic Brain: Neoliberalism and the Neuronal Self*, 14 HEALTH 635, 635 (2010).

social outcomes. Until we, as a society, devise collective solutions to provide a better environment for all children, these disparities will likely continue.

C. Intersectional Effects of Biological Inequality

Biological inequality is intersectional. In the United States, black Americans experience lower birth weights and higher incidences of cardiovascular disease in comparison with white cohorts.²³¹ Scientists Christopher Kuzawa and Elizabeth Sweet argue that these disparities are products of epigenetic mechanisms driven by the hostile social environment that black people inhabit.²³² As referenced above, maternal stress transmits epigenetic changes.²³³ And, as discovered within the Dutch-famine data, low-birth-weight babies are at greater risk for developing metabolic diseases, such as heart disease, diabetes, and high blood pressure.²³⁴ Kuzawa and Sweet argue that low birth weights for black babies are traceable to the stress, discrimination, and lower SES experienced by black American mothers during the course of their lives and pregnancies.²³⁵ The stress of racial discrimination, experienced in employment, housing, education, law, and everyday life is a type of stressor that can become biologically embodied.²³⁶ Thus, Kuzawa and Sweet argue that race-related stress functions as durable “developmental programming” that produces epigenetic changes, which in turn produce persistent, unequal health outcomes for black Americans.²³⁷

Kuzawa and Sweet present evidence that supports the conclusion that the U.S. social environment has become biologically embedded within the black populace and that these biological effects cannot be explained by inherent genetic variations. Their theory is borne out by data indicating that black American newborns have lower birth weights than children born to women who recently immigrated to the United States from Africa.²³⁸ The birthweight of children for subsequent generations of African immigrants then regresses to converge with the lower black American mean.²³⁹ This phenomenon leads Kuzawa and Sweet to theorize that the social en-

231. Kuzawa & Sweet, *supra* note 1, at 2, 8.

232. *Id.*

233. *See supra* notes 117–118 and accompanying text.

234. *See supra* notes 113–116 and accompanying text.

235. Kuzawa & Sweet, *supra* note 1, at 3, 8.

236. *See* Chae et al., *supra* note 1.

237. Kuzawa & Sweet, *supra* note 1, at 2, 4.

238. *Id.* at 8. *See generally* Gopal K. Singh & Stella M. Yu, *Adverse Pregnancy Outcomes: Differences Between US- and Foreign-Born Women in Major US Racial and Ethnic Groups*, 86 AM. J. PUB. HEALTH 837 (1996) (examining the differences between US-born and foreign-born mothers in pregnancy risks); Martha S. Wingate & Greg R. Alexander, *The Healthy Migrant Theory: Variations in Pregnancy Outcomes Among US-Born Migrants*, 62 SOC. SCI. & MED. 491 (2006) (finding internal migrant populations have better birth outcomes).

239. Kuzawa & Sweet, *supra* note 1, at 8.

vironment, rather than genes, is responsible for variations in black Americans' health.²⁴⁰ Moreover, in their paper, Kuzawa and Sweet caution that black American birth outcomes are not a product of the mother's choice.²⁴¹ Rather, "[t]he most important predictors of compromised birth outcomes include factors such as self-perceived discrimination, racism, and chronic stress."²⁴²

When the law changes to legally prohibit discrimination, it improves the health outcomes for black people.²⁴³ This was shown in a recent study, conducted by Harvard epidemiologist Nancy Krieger, which analyzed the repeal of Southern Jim Crow laws on the health of black citizens.²⁴⁴ Krieger documented that after the repeal of Jim Crow laws in the South, health among blacks improved and overall health inequities between blacks and whites decreased.²⁴⁵ Krieger used infant mortality to track these health inequities because infant mortality, in particular, functions as an effective marker for a population's overall health.²⁴⁶ Infant mortality "is highly sensitive to living conditions and access to medical technology during pregnancy and the first year of life and is also reflective of mothers' cumulative health status before and after conception."²⁴⁷ Krieger's data analysis indicated that during Jim Crow, infant-mortality rates for blacks were higher in regions where Jim Crow laws were in effect than in areas without these laws.²⁴⁸ After the 1964 Civil Rights Act abolished de jure segregation, there was a convergence in the infant mortality for black infants inside and outside of jurisdictions that experienced Jim Crow.²⁴⁹ However, the black infant-mortality rate, though it improved in some areas, remained much higher than the white infant-mortality rate.²⁵⁰

Krieger theorizes that while the enactment of the 1964 Civil Rights Act improved health outcomes for black people, it was not enough, given the persistence of the black–white infant-mortality gap.²⁵¹ Structural changes, aimed at de facto forms of discrimination, are necessary to resolve this tragic problem.²⁵² Krieger thoughtfully advocates an "ecosocial"

240. *Id.* at 8, 10.

241. *Id.* at 10. ("[T]he research reviewed here overwhelmingly points to the importance of factors that are symptomatic of structural inequality and discrimination rather than choice.")

242. *Id.* at 10; see also DeAnnah R. Byrd et al., *Infant Mortality: Explaining Black/White Disparities in Wisconsin*, 11 *MATERNAL & CHILD HEALTH J.* 319, 320 (2007) (reporting on women who report racial discrimination and low birth weight for infants as a factor that heavily contributes to risk of infant mortality).

243. Krieger et al., *The Unique Impact of Abolition of Jim Crow Laws*, *supra* note 26, at 2234.

244. *Id.*

245. *Id.*

246. *Id.*

247. *Id.*

248. *Id.* at 2236.

249. *Id.*

250. *Id.*

251. *Id.* at 2241; Nancy Krieger et al., *Jim Crow and Premature Mortality Among the US Black and White Population, 1960-2009: An Age-Period-Cohort Analysis*, 25 *EPIDEMIOLOGY* 494, 503 (2014) [hereinafter Krieger et al., *Jim Crow and Premature Mortality*].

252. See Carpenter, *supra* note 26 (reporting on Dr. Krieger's theories).

approach to racialized public-health outcomes to understand “how we literally biologically embody exposures arising from our societal and ecological context, thereby producing population rates and distributions of health.”²⁵³ Moreover, Krieger recommends that research on race and health outcomes should analyze race not as an “a priori trait of individuals” but as a multidimensional category entwined with political, economic, and social relations.²⁵⁴

David H. Chae and his colleagues have documented a connection between the psychosocial stress of racial discrimination and the deterioration of one’s DNA, which controls the aging process.²⁵⁵ Black men have a life expectancy of 69.7 years, compared to 75.7 years for white men.²⁵⁶ Black men suffer from age-related diseases that appear earlier and show more severity than diseases suffered by other groups.²⁵⁷ Chae and his colleagues discovered that black men had shorter DNA telomeres than other groups.²⁵⁸ Telomeres are pieces of the ends of the DNA that protect the DNA from deterioration.²⁵⁹ Black men who reported experiences of discrimination and held internal antiblack attitudes had shorter telomeres than black men who held more problack attitudes.²⁶⁰ While the Chae study does not encompass epigenetics as the mechanism for how the telomeres become shortened, it does suggest that the social environment coupled with the psychic internalization of negative stereotypes can become embodied and impact life expectancies. “[R]acial discrimination in concert with the internalization of racial bias has pernicious effects on biological aging, and . . . this is one pathway through which social inequities generate greater disease vulnerability in the population.”²⁶¹

Finally, Northwestern University Professor Emma Adam has found data supporting a theory that race-based stress influences the HPA (the hormonal apparatus that regulates stress) in a way that produces highly negative physical and mental health outcomes.²⁶² Specifically, Adam and her colleagues observed cortisol levels (which measure HPA activity) in a large set of longitudinal data.²⁶³ Adam’s found that individuals (both black and white) who reported high levels of perceived racial discrimination

253. Krieger, *An Ecosocial Approach*, *supra* note 16.

254. Krieger et al., *Jim Crow and Premature Mortality*, *supra* note 251.

255. Chae et al., *supra* note 1, at 107.

256. *Id.* at 103.

257. *Id.*

258. *Id.* at 104.

259. *Id.*

260. *Id.* at 107. Amy J. Schulz and her colleagues reported similar findings with respect to depression in black American women. Amy J. Schulz et al., *Discrimination, Symptoms of Depression, and Self-Rated Health Among African American Women in Detroit: Results from a Longitudinal Analysis*, 96 AM. J. PUB. HEALTH 1265, 1265, 1267 (2006). In this study, Schulz found that black American women who experienced an increase in racial discrimination over time had a correlated increase in symptoms of depression. *Id.*

261. Chae et al., *supra* note 1, at 108.

262. Adam et al., *supra* note 134, at 280, 288.

263. *Id.* at 288.

(PRD) showed cortisol levels with a flatter slope than individuals who did not report PRD.²⁶⁴ Notably, among those individuals reporting PRD, black subjects reported different cortisol levels in the morning and lower overall cortisol levels throughout the day.²⁶⁵ These racially disparate results led Adam to theorize that her study's black subjects were "actively mobilizing [their stress response systems] to cope with the anticipated discriminatory experiences" that they know they will face during the day.²⁶⁶

In a more recent paper, Adam and her colleagues theorized that the physiological impact of race discrimination might explain racial and ethnic academic-achievement gaps.²⁶⁷ The racial discrimination primarily experienced by black and Latino youth is linked to physiological effects, such as overactivation of the body's HPA system and disruptions in an individual's sleep cycle.²⁶⁸ While low SES is also associated with these physiological effects, which mediate academic achievement, members of racial minorities suffer from double disadvantage; the stress of living in economic disadvantage is compounded by the stress of being a target of discrimination.²⁶⁹ Adam and her colleagues explain that both the HPA-system response (which can lead to behavioral and mood changes, like increased anxiety and anger) and disruptions in individual sleep cycles (which can also lead to fatigue induced behavioral issues) can explain the racial and ethnic educational-achievement gap, which has remained stable since the 1990s, even between individuals at higher SES levels.²⁷⁰

Adam's work debunks the highly offensive idea, propagated by Richard Herrnstein and Charles Murray, that race-based disparities on IQ tests and other cognitive metrics are a product of genetic differences between different races.²⁷¹ While scholarly consensus has rejected Murray and

264. *Id.* It has been theorized that stress exposure produces flatter diurnal cortisol rhythms, which in turn correlate with higher incidents of depression, anxiety disorders, PTSD, fibromyalgia, cardiovascular disease, and metabolic disorders—the ill health effects that have been identified throughout this paper. *Id.* at 280, 288.

265. *Id.* at 288. Lower daily cortisol levels indicate hypocortisolism—"a pattern of low and less dynamic cortisol levels that is thought to result from past chronic stress or traumatic stress, and is associated with negative health outcomes." *Id.* Having a flat (as opposed to steep) pattern of cortisol produces negative health outcomes, but low overall levels of the stress hormones is an additional negative indicator. *Id.*

266. *Id.*

267. Dorainne J. Levy et al., *Psychological and Biological Responses to Race-Based Social Stress as Pathways to Disparities in Educational Outcomes*, 71 AM. PSYCHOLOGIST 455, 455–56 (2016).

268. *Id.* at 461–64.

269. *Id.* at 458–59.

270. *Id.* at 456–57, 461–64. Although the SES achievement gap has widened and is now twice as large as the black/white achievement gap, it does not fully explain the racial/ethnic achievement gap because that gap has not changed since the 1990s, and persists at higher income levels. *Id.* at 456–57 (citing Sean F. Reardon, *The Widening Income Achievement Gap*, EDUC. LEADERSHIP, May 2013, at 10, 11–12).

271. RICHARD J. HERRNSTEIN & CHARLES MURRAY, *THE BELL CURVE: INTELLIGENCE AND CLASS STRUCTURE IN AMERICAN LIFE* 269, 270 (1994).

Herrnstein's theory,²⁷² the idea that innate individual characteristics (including racial characteristics) produce positive or negative life outcomes remains ingrained in U.S. conservative culture. While many scholars have persuasively theorized that social, psychic, and economic conditions experienced by historically oppressed racial minorities have more to do with achievement gaps than genetics,²⁷³ these newer biological studies add helpful strength and legitimacy to this point.

The one-two punch of racial discrimination and economic disadvantage has the capacity to produce enduring injury. In considering the work of Rachel Yehuda and her colleagues, which documented epigenetically programmed stress disorders in Holocaust survivors and their offspring,²⁷⁴ we can reasonably hypothesize that the trauma of slavery, the terroristic enforcement of Jim Crow segregation, and the continuing subordination of black people have become embodied, producing a biological inequality that continues to be transmitted to subsequent generations. This enduring biological inequality belies the argument that black people can and should overcome discrimination through grit and individual effort. Such a quest is not fully possible when past and present violence and discrimination continue to invade, infect, and sicken.

D. Social Cause Versus Social Selection

Is one's inherited material environment the primary causal factor that determines one's life outcomes? This is the theory of social cause. Or, does one inherit certain cognitive traits that predispose one to choose certain environments over others? This is the theory of social selection.

Social selection theory posits that differences in inherited cognitive ability predispose people to certain SES environments, which then become embodied.²⁷⁵ In the context of the brain, social selection theory holds that

272. Christopher Jencks & Meredith Phillips, *Introduction to THE BLACK-WHITE TEST SCORE GAP* 1, 2 (Christopher Jencks & Meredith Phillips eds., 1998) (“[D]espite endless speculation, no one has found genetic evidence indicating that blacks have less innate intellectual ability than whites.”); Richard E. Nisbett, *Race, Genetics, and IQ*, in *THE BLACK-WHITE TEST SCORE GAP*, *supra*, at 86, 89 (“Despite the assertions of some scholars, including Herrnstein and Murray, a review of the evidence in both areas provides almost no support for genetic explanations of the IQ difference between blacks and whites.”).

273. Stereotype threat presents a social and cultural explanation as to why racially disadvantaged perform less well on standardized tests. Claude M. Steele & Joshua Aronson, *Stereotype Threat and the Test Performance of Academically Successful African Americans*, in *THE BLACK-WHITE TEST SCORE GAP*, *supra* note 272, at 401–02, 422–23. Professor Claude Steele developed stereotype threat theory in a series of studies showing that in a cognitive testing environment, racially disadvantaged students perform less well when they are told the test is a measure of their cognitive ability and also when their racial identity is primed. *Id.* at 401–23. Being confronted with a negative racial stereotype in a high-pressure testing environment depresses performance on the test. *Id.* at 422–23. Meredith Phillips and her colleagues provide a succinct explanation of the social theories that explain the black-white academic performance gap. See Meredith Phillips et al., *Family Background, Parenting Practices, and the Black-White Test Score Gap*, in *THE BLACK-WHITE TEST SCORE*, *supra* note 272, at 103, 109–10 (stating black genes “cause social and economic inequalities that affect cognitive skills”).

274. See *supra* notes 96–107 and accompanying text.

275. Hackman et al., *supra* note 1, at 653.

“all human capacities are present in the exuberantly wired brain” and the individual simply seeks out those preferred external stimuli that will influence the individual’s brain structure.²⁷⁶ Social selection has also been described as a theory that actively correlates individual genes to the environment.²⁷⁷ As an example, because children with above average verbal abilities will enjoy reading, the children’s positive reactions to reading encourage parents to set up a home environment that supports literacy.²⁷⁸ Social selection holds that people are born with certain traits and characteristics that function as neural limits, which produce a selective effect.²⁷⁹ Individuals with high cognitive abilities end up with greater SES, whereas individuals with lesser abilities end up in the lower classes.²⁸⁰

The conflict between social cause and social selection figures into the debate concerning policy solutions to remedy (or not remedy) the deeply unequal outcomes experienced by the haves and have nots in the United States. The standard conservative position is that inherited differences in intelligence are the controlling a priori factors that determine one’s place in society.²⁸¹ The authors of the infamous book *The Bell Curve* favorably frame the founding fathers’ belief that people should be sorted into a natural aristocracy, with the smartest taking on the responsibility for governance.²⁸² If an individual’s place in society is primarily a product of *inherited* cognitive ability, then it does not make much sense to marshal resources to provide more equal outcomes to everyone.²⁸³ Thus, social selection, with its emphasis on inherited traits, is aligned with a conservative position that would decline to devote collective resources to achieving more equal outcomes.

Although it matches up with neoliberal or conservative conceptions of the self, social selection is not the only explanatory theory for the environment–outcome relationship. Social selection and social cause are not mutually exclusive—both have support in the scientific literature—the debate centers on how much emphasis one should be given over the other.²⁸⁴

276. JOSEPH LEDOUX, *SYNAPTIC SELF: HOW OUR BRAINS BECOME WHO WE ARE* 87 (2002).

277. See Phillips et al., *supra* note 273, at 110.

278. See *id.*

279. Lende, *supra* note 23, at 188.

280. *Id.*

281. HERRNSTEIN & MURRAY, *supra* note 271, at 10 (describing the theory that IQ determines social standing).

282. See *id.* at 530–33.

283. *Id.* at 530.

284. For a view into the two ideological poles of this debate, see Herrnstein & Murray’s *The Bell Curve* and the book written in response to it—*The Black-White Test Score Gap*. See generally HERRNSTEIN & MURRAY, *supra* note 271; THE BLACK-WHITE TEST SCORE GAP, *supra* note 272. In the introduction of *The Black-White Test Score Gap*, the editors explain that “despite endless speculation, no one has found genetic evidence indicating that blacks have less innate intellectual ability than whites.” Christopher Jencks & Meredith Phillips, *Introduction*, *supra* note 272, at 1, 2. For a recent argument that expresses deep skepticism for whether social cause theories should support arguments for more robust antipoverty programs, see Amy L. Wax, *The Poverty of Neuroscience of Poverty: Policy Payoff or False Promise?*, 57 JURIMETRICS J. 239, 241–42 (2017). Professor Wax argues that because neuroscience cannot definitively quantify how much of poverty’s embodied effects derive

For debates about remedies for lessening unequal outcomes, recent advances in epigenetics and neuroplasticity indicate that the balance should be tipped more heavily in favor of social cause.

In contradistinction to social selection, the theory of social cause elevates the material environment as a causal factor for the various negative health and cognitive outcomes associated with disadvantaged status.²⁸⁵ Individual action does play a role, but biologists and neuroscientists now understand that the environment, along with one's genes, pulls many of the strings toward particular social outcomes.²⁸⁶ While "[g]enes may play an essential role in placing a function in the brain of every human, . . . at the same time [they] make a relatively small contribution to differences in the way that function is wired in individuals."²⁸⁷

Social cause theory is supported by studies showing that small income increases lessen the rate of childhood mental-health issues in impoverished families; that SES-mediated environments explain IQ differences in adopted children; and that the impact of poverty on children is greater in early childhood than late childhood.²⁸⁸ As Michael Marmot points out, social cause theory is substantiated by "[b]irth cohort studies show[ing] that social position *precedes* the development of ill-health."²⁸⁹ And, Marmot's studies of British civil servants indicated that "job promotion led to better health, rather than the reverse."²⁹⁰

Moreover, the kind of stress that mediates embodied inequality is not a matter of selection. The stress produced by living in an impoverished environment "involves both brain and body and is often driven by reactions to environmental stressors, both real (e.g., a fistfight) and perceived (e.g., thinking a fistfight could happen)."²⁹¹ The research on embodied inequality supports the concept that culture and social structure shape the individual, just as much as or perhaps even more so than the characteristics that the individual is born with.²⁹²

A disadvantaged environment (e.g., an uncaring mother, harsh discipline, fear of violence in the neighborhood) impacts biology at the individual level.²⁹³ The connection between epigenetic changes and maternal

from social causes as opposed to genetic characteristics, then the science should not be used to support antipoverty reform. *Id.*

285. Hackman et al., *supra* note 1, at 653.

286. *Id.* (explaining that social selection and social cause are not mutually exclusive and may operate together); Hertzman & Boyce, *supra* note 141, at 341 (explaining the theory of "gene-by-environment" to explain differences in cognitive achievement between high-income and low-income children); Kuzawa & Sweet, *supra* note 1, at 2–3 (explaining how genetics alone fail to explain for racial disparities in health outcomes).

287. LEDOUX, *supra* note 276, at 91.

288. Hackman et al., *supra* note 1, at 653.

289. Marmot, *Status Syndrome*, *supra* note 2, at 152 (emphasis added).

290. *Id.* at 150, 152.

291. Lende, *supra* note 23, at 191.

292. *See id.* at 191–93.

293. Hackman et al., *supra* note 1, at 654; McEwen & Gianaros, *supra* note 1, at 192.

care makes it easy to blame the individual, in this case mothers. Indeed, Charles Murray and Richard Herrnstein believe that poverty and its related stress effects are in large part perpetrated by “incompetent” “single women of low intelligence.”²⁹⁴

Such a callous, reductive framing, however, is a shortsighted approach for both the causes of and solutions to embodied inequality.²⁹⁵ Embodied inequality is a structural problem.²⁹⁶ While biologically impactful stress operates at the individual level (parenting, discipline), it also originates from a cascade of mundane aggressions, from everyday life occurrences that normally do not come under criticism²⁹⁷ to choices and institutional arrangements that maintain equilibrium between groups.²⁹⁸ Moreover, there is comparative evidence that stress is related to the amount of inequality in a society’s hierarchy; the steeper the hierarchy in a given society, the worse the health outcomes are for those at the bottom.²⁹⁹ In this way, it is not likely that intervention aimed exclusively at the individual level will fully solve the problem because of how deeply inequality runs in the material environment, our bodies, and our brains.³⁰⁰ Accordingly, the biological embodiment of inequality is a problem that requires structural approaches, collective solutions capable of changing the degree of inequality that exists in our society.

The next sections consider what, if anything, law and policy can do to remodel the landscape of our capitalistic democracy in a way that removes or ameliorates the toxic material conditions that can get under the skin. From a rhetorical and legal standpoint, solving this problem requires engagement with the logic of neoliberalism because neoliberalist policies are, in great part, producing the stress and uncertainty that then become embodied.

II. RHETORIC AND POLICY RESPONSES

While the big problems driving biological inequality—poverty, education, income inequality, racial inequality—may not be entirely solvable through legal means, the scientific theories explained in this Article, as

294. HERRNSTEIN & MURRAY, *supra* note 271, at 519 (“[I]nadequate nutrition, physical abuse, emotional neglect, lack of intellectual stimulation, a chaotic home environment . . . are very difficult to improve . . . when the single mother is incompetent.”).

295. Kuzawa & Sweet, *supra* note 1, at 10 (arguing that embodied inequality is best seen as “symptomatic of structural inequality and discrimination rather than [poor] choice[s]”).

296. *Id.*

297. Hertzman & Boyce, *supra* note 141 (“[I]t is often the less memorable but hurtful and far more prevalent misfortunes of childhood that become embedded in neural circuitry and produce the vulnerabilities of adult life.”).

298. See generally Christine Stephens & Annemarie Gillies, *Understanding the Role of Everyday Practices of Privilege in the Perpetuation of Inequalities*, 22 J. COMMUNITY & APPLIED SOC. PSYCHOL. 145, 145–56 (2012) (arguing that solving inequality must involve analyzing how the actions of middle class groups (however unintentional) keep those beneath them in their place).

299. Marmot, *Status Syndrome*, *supra* note 2, at 150, 152–53.

300. Lende, *supra* note 23, at 197 (arguing that any attempt to address inequality at the individual level will not fully address the problem because of how deeply rooted the problem is).

they evolve, provide new openings for novel legal arguments and approaches to policy making. Ostensibly, neoliberalism and neoliberalist rhetoric, with their relentless individualism and abstracted economic and market focus, have produced the bodily harm suffered by the poor and disadvantaged. Countering neoliberal thinking by cataloging and describing harm to individual bodies gives these arguments a new visceral fire-power. Thus, the biology of inequality provides progressive advocates with powerful new science-based evidence to support arguments that would elevate collective, communitarian goals over individual interests. For quite some time, progressive advocates have grappled with a de facto narrative of virtue and merit that places individual striving front and center.³⁰¹ But now there is evidence that this narrative does not function in a material environment of deprivation and disadvantage, produced by faceless capitalistic forces, which damages bodies and brains so much.

Armed with science, progressive legal scholars and legal policy makers now have the ability to push for policy and legal remedies that could reshape the material experiences of disadvantaged citizens in such a way as to reverse these harmful and intractable biological effects. Such remedies could be small scale, focused on interventions at the family level, or larger scale, such as legal interventions in constitutional, workplace, and education law.

The new science supports policy solutions to strengthen the safety net in a way that lessens capitalism's effects on society's most vulnerable. Within the law, changes to workplace law and education law represent universal remedies that could ameliorate the kind of randomized stress that becomes embedded and passed on to subsequent generations. The science could also re-animate the deployment of race-based remedies (affirmative action and reparations) on the ground that biological harm is now traceable to the psychosocial stress produced by slavery, Jim Crow terror, and today's pervasive institutional racism. Section II.A. explains the new rhetorical strategies that the science makes available. Then, Section II.B. outlines some of the policy changes that could heal these wounds. Part III will address remedies specific to law.

A. Rhetorical Strategies: Countering Neoliberalist Logic

Embodied inequality can mount a strong challenge to the logic of neoliberalism and advance arguments for collective solutions for society's most durable problems—failing public education, poverty, and income inequality. This Article directly engages with neoliberal logic because neoliberalist policies engender the kind of stress and lack of control that becomes embedded in the body and brain.

301. See ROLLO MAY, *THE MEANING OF ANXIETY* 180–81, 184 (rev. ed. 1977) (explaining the deep folkways within Western culture that emphasize virtue, individualism, and competition against others).

Neoliberalism refers to the political logic that foregrounds the market and individuals in competition as the primary actors in society; the government is relegated to the background, stripped of the power to intervene and remedy social problems.³⁰² As a mode of thought, it is most associated with policies advanced in the 1980s by Margaret Thatcher and Ronald Reagan, policies that sought deregulation, stable private-property systems, and the dismantling of the welfare state to shift the role of government from public to private.³⁰³ Influenced by the economic theory of Friedrich Hayek, neoliberalism posits that the only thing the state should do is support the market, protecting the mechanisms by which individuals can compete and be entrepreneurs.³⁰⁴ The system, according to the theory, does not privilege one set of values over another.³⁰⁵ The market is morally neutral.³⁰⁶ Thus, neoliberalism differs somewhat from other incarnations of conservative individualism because it presents itself as value free. Rather than emphasizing the virtuosity of values, like a striving work ethic, it declares that the neutral market is the best system for organizing social relations.³⁰⁷ However, the longstanding positive valence placed on individualism remains in the thought system as an enthymeme, an implicitly understood premise.³⁰⁸

Neoliberalism, operating through its contention that it is value free, might be thought of as individualism on steroids. The subject's virtue is measured by a capacity for "self-care," the ability to provide for one's own need and ambitions.³⁰⁹ Poor social outcomes are solely the fault of the individuals who made poor choices in the market.³¹⁰ "The neoliberal model of [individual] choice does not recognize the material constraints that limit an individual's choices because those constraints are seen as merely the product of her previous choices."³¹¹

Under neoliberalist logic, if a person fails, it is her own fault.³¹² Sociologist Loic Wacquant refers to this phenomenon as "[t]he cultural trope

302. See WENDY BROWN, UNDOING THE DEMOS: NEOLIBERALISM'S STEALTH REVOLUTION 10, 17, 28, 30, 42 (2015). In neoliberalist economies, global capital remains dependent on the state's diffuse and variegated network of law, treaties, and quasi-governmental institutions. Jackson Lears, *The Long Con*, 37 LONDON REV. BOOKS 28, 29–30 (2015) (reviewing STEVE FRASER, THE AGE OF ACQUIESCENCE: THE LIFE AND DEATH OF AMERICAN RESISTANCE TO ORGANIZED WEALTH AND POWER (2015)).

303. Corinne Blalock, *Neoliberalism and the Crisis of Legal Theory*, 77 L. & CONTEMP. PROBS. 71, 83 (2014).

304. *Id.* at 72, 73, 85, 99.

305. *Id.*

306. *Id.* at 99.

307. *Id.*

308. See *id.* at 88, 89, 102 (explaining that neoliberalist logic/rhetoric is so powerful because it is "inextricably tied to [deep-seated] beliefs about liberty, dignity, and individual choice, as well as corresponding beliefs about the capacities and limits of the state to effectuate change").

309. *Id.* at 88 (quotations omitted).

310. *Id.*

311. *Id.*

312. *Id.*

of individual responsibility,” a rhetoric that enables corporations and governments to abdicate responsibility for individuals’ wellbeing.³¹³ If you do not do well in the market, it is your own fault.³¹⁴ In this way, neoliberalism privileges a highly atomistic view of human relations that denies the necessity of communal or collective solutions to provide stability and security to working people.³¹⁵ As theorized in more depth below, neoliberalist philosophy produces uncertainty and anxiety, which then morph into the kind of stress that can become embodied.

The neoliberalist perspective efficiently dispenses with arguments that seek to remedy unequal social outcomes. The law should ensure equal opportunity but not concern itself with the absence of equal outcomes.³¹⁶ According to this logic, the state should not interfere with social sorting mechanisms because differences in social outcomes result from differences in cognitive talent.³¹⁷ Inequality becomes the norm, “a market formulation of winners and losers.”³¹⁸

In this way, antipathy to interventions for public welfare sets neoliberalism apart from liberalism. Although liberalism emphasized the virtues of individual striving and competition, it also viewed law as an appropriate mechanism (particularly Keynesian³¹⁹ approaches) for accomplishing the objectives of a society.³²⁰ However, in the words of Margaret Thatcher, “[t]here is no such thing as society.”³²¹ Two strains of neoliberal thought support the hostility to large-scale state or government programs that seek to achieve social security for all, as a public good.³²² The first concept is that the market is such a complex and fragile ecosystem that anything that might interfere with it should be avoided.³²³ And second, drawing upon Cold War experiences, is the belief that it is simply impossible for the state

313. Loïc Wacquant, *Crafting the Neoliberal State: Workfare, Prisonfare, and Social Insecurity*, 25 Soc. F. 197, 213–14 (2010).

314. See *id.*; Blalock, *supra* note 303, at 88.

315. Blalock, *supra* note 303, at 87.

316. In their book, Richard J. Herrnstein and Charles Murray provide an example of the conservative thought that advocates for equal opportunity without regard to outcomes. See HERRNSTEIN & MURRAY, *supra* note 271, at 530–34.

317. *Id.* (discussing differences in cognitive ability against the backdrop of affirmative action). See generally CHRISTOPHER HAYES, *TWILIGHT OF THE ELITES: AMERICA AFTER MERITOCRACY* (2012) (discussing how American individualism views unequal outcomes as a natural sorting based on innate individual differences).

318. BROWN, *supra* note 302, at 41.

319. John Maynard Keynes (for whom Keynesian economics is named) theorized that a state’s government can and should be actively involved in managing the economy, through fiscal policy and government spending. See Alan S. Blinder, *The Fall and Rise of Keynesian Economics*, 64 ECON. REC. 278, 278–83 (1988).

320. Blalock, *supra* note 303, at 84.

321. *Id.* at 73 (quoting Interview by Douglas Keay with Margaret Thatcher, Prime Minister of the U.K., in London, U.K. (Sept. 23, 1987), <http://www.margaretthatcher.org/document/106689>).

322. Blalock, *supra* note 303, at 87 (“The neoliberal framework, premised on the impossibility of enacting a collective substantive vision, clearly cannot ground the state’s legitimacy in democratic authority and pursuit of the common good the way liberalism does.”).

323. *Id.* at 85–86.

to successfully effectuate social policy for the public good; the market is the only mechanism that works.³²⁴

Specifically, neoliberalism eschews any commitment, grounded in a sense of the collective good and moral responsibility, to provide individual workers with a secure, lifelong job that carries a living wage.³²⁵ After World War II, as more competition emerged from postwar economies, the American economy morphed from an industrial economy to a finance economy.³²⁶ In this transition, the focus moved from making money by manufacturing things, which created fairly stable jobs at the firm level, to an emphasis on inflating stock prices, for which mass layoffs created the best payoff.³²⁷ Neoliberalism has ushered in an era of disaggregated production, offshore jobs, and temporary staffing models.³²⁸

Thus, within the neoliberal political economy, the distressing effects of material inequality are not a moral problem to be reckoned with. Inequality is just the natural and probable consequence of faceless market forces.³²⁹ Neoliberalism thus enshrines an incredibly harsh style of social Darwinism that was absent from liberalism, which was at least animated by humanistic values, which in turn supported social-welfare policies to help vulnerable subjects.³³⁰ Unlike liberal Keynesian approaches, neoliberalist logic abdicates all responsibility for individuals who falter. “The state is not responsible if individuals do not properly respond to the market’s incentive structures, but it is responsible for the pernicious consequences of sheltering individuals from the market’s disciplinary effects.”³³¹ Thus, neoliberalist logic views social welfare programs as anathemas, shelters that shield individuals from the consequences of their own actions. The government’s only concern is giving people access to markets and the ability to pursue their own interests.³³²

Rhetorically, the social insecurity inherent in the neoliberal economy has been reframed as free agency. All individuals, regardless of their circumstances, are reduced to human capital or capitals exercising choice in the market.³³³ And, as human capitals, everyone must be competitively

324. *Id.* at 93–94.

325. Lifelong social support in exchange for a life’s work is referred to as the Fordist-Keynesian social compact. See Wacquant, *supra* note 313, at 201.

326. Lears, *supra* note 302.

327. *Id.*

328. *Id.*

329. Blalock, *supra* note 303, at 88.

330. See BROWN, *supra* note 302, at 187–88 (discussing the post-WWII view that liberal arts exposure would cultivate humanistic values, helpful for citizenship and democracy); Wacquant, *supra* note 313, at 198–99 (referencing the acceptance of social welfare initiatives during the middle of the twentieth century).

331. Blalock, *supra* note 303, at 88.

332. *Id.*

333. Blalock, *supra* note 303, at 87; BROWN, *supra* note 302, at 37.

entrepreneurial, constantly working to increase value and return on investment.³³⁴ Think of the person who works in a low-wage job during the day and is a sharing-economy “entrepreneur” for Uber at night.³³⁵ By categorizing all workers as entrepreneurial, neoliberalism obscures class visibility.³³⁶ Workers are just like management (just exceedingly unequal to individuals in management or executive positions).³³⁷ And yet, ordinary workers “have no guarantee of security, protection, or even survival.”³³⁸

From a rhetorical perspective, neoliberalism gains its power because its logic is based around an enthymeme, a syllogism in which the premises (inequality is normal; the market is an effective, value-free organizing institution) are left unstated and thus untouched. Neoliberalism, by not directly engaging with democratic principles oriented around the collective good—or “*the demos*” as Wendy Brown frames it—has been able to conduct a “stealth revolution” and erase intelligent, legitimate alternatives to the current political rationality, held collectively.³³⁹ Neoliberalism is hegemonic.³⁴⁰ As Jackson Lears quipped, neoliberalism is “everywhere and nowhere; its custodians are largely invisible.”³⁴¹

Thus, on the one hand, neoliberalist rhetoric focuses on individual responsibility that says that if one fails to obtain a meaningful place in life, then it is one’s own fault.³⁴² On the other hand, neoliberalism employs an “illusion of amorality”³⁴³ along with neutral market and economics language to extract individual personhood from corporate actors in a way that entirely absolves them from responsibility.³⁴⁴ In fact, neoliberalism’s neutrality allows it to be “indifferently embraced by politicians of the Right

334. BROWN, *supra* note 302, at 36, 65.

335. See Robert Reich, *Robert Reich: The Sharing Economy Will Be Our Undoing*, SALON (Aug. 25, 2015, 2:15 AM), http://www.salon.com/2015/08/25/robert_reich_the_sharing_economy_will_be_our_undoing_partner.

336. BROWN, *supra* note 302, at 65.

337. See *id.*

338. *Id.* at 37.

339. *Id.* at 68–69, 115–16. Brown refers to neoliberal logic as “more termitelike than lionlike.” *Id.* at 35.

340. Blalock, *supra* note 303, at 89.

341. Lears, *supra* note 302. Looking at this submerging phenomenon from a slightly different angle, Loïc Wacquant argues that certain aspects of neoliberalism (particularly its tendency toward mass incarceration of the poor and racially oppressed) operate in a highly visible way, through the police, the courts, and popular culture that celebrates law enforcement (*Law & Order*, *CSI*, *Dog the Bounty Hunter*, etc.). See Wacquant, *supra* note 313, at 206. Nonetheless, the carceral arm of neoliberalism effectively submerges the bodily pain and suffering experienced by those in the bottom ranks of society by invisibilizing and containing them—behind prison walls. See *id.*

342. See *supra* notes 304–07 and accompanying text.

343. Blalock, *supra* note 303, at 99.

344. See Linda Nader, *The Life of the Law - A Moving Story*, 36 VAL. U. L. REV. 655, 662 (2002) (describing consumer/business disputes as moving from a face-to-face method of conversation to a fact-to-faceless culture).

or the Left.”³⁴⁵ Neoliberalist rhetoric is brilliant in its power to both scapegoat (in a neutral way) individuals who are at the bottom rungs of society and mask responsibility for those at the top. This dual rhetoric relies on both submersion (not seeing the palpable harm that the capitalistic apparatuses create) and abstraction (giving corporate actors a mask of invisibility).

Finally, despite its claim to being value-free, abstract neoliberalist rhetoric descends from the conservative political rhetoric, devised by Richard Nixon adviser Lee Atwater and successfully deployed to dampen the public sentiment garnered by the civil rights, anti-war, and other 1960s social-justice-oriented movements. In the 1960s, political and social information were presented in a new, highly visual way that galvanized audiences.³⁴⁶ Progressives gained rhetorical ground in part by emphasizing (on television and in photojournalism) the visual and visceral harm happening *to bodies*. Television and print media exposed audiences to black American civil rights protesters being mauled by police dogs and collapsing under high-pressure fire hoses, as well as Southern school children facing hateful heckling in their journey to a newly desegregated school.³⁴⁷ Viewers also beheld the corpses of the murdered Emmett Till and Dr. Martin Luther King, together with universal images of human grieving and despair in photographs of Mamie Till and Coretta Scott King.³⁴⁸ Also in context, audiences were moved to oppose the war in Vietnam upon viewing the slain student protesters at Kent State and haunting photographs of agonized children fleeing napalm in Vietnam.³⁴⁹

In response to this leftward shift in public sentiment, Lee Atwater’s “Southern Strategy” replaced visibly racial rhetoric (racial epithets, repeated calls for segregation, etc.) with abstract language focusing on individual freedom: freedom of association (as an argument against forced desegregation), freedom from forced busing of children (an argument against school desegregation efforts), and freedom from the dangerous inner-city crime.³⁵⁰ This more abstracted rhetoric was much more middle-class and

345. Wacquant, *supra* note 313, at 209. In fact, Democratic President Bill Clinton’s 1990s welfare reforms and tough on crime positions integrated neoliberalism fully into the U.S. political economy. *See id.*

346. *See* Christine Harold & Kevin Michael DeLuca, *Behold the Corpse: Violent Images and the Case of Emmett Till*, 8 Rhetoric & Pub. Aff. 263, 267 (2005). *See generally* Maurice Berger, *For All the World to See: Visual Culture and the Struggle for Civil Rights* (2010) (providing a comprehensive examination of the ways images inspired audiences to join the civil rights movement).

347. Kevin M. Kruse, *White Flight: Atlanta and the Making of Modern Conservatism* 210 (2005).

348. *See* Harold & DeLuca, *supra* note 346, at 265.

349. *See* Rodger Streitmatter, *Mightier Than the Sword: How the News Media Have Shaped American History* 177–87 (2012) (discussing the impact of news and photojournalism on the public’s view of the Vietnam War).

350. Rick Perlstein, *Exclusive: Lee Atwater’s Infamous 1981 Interview on the Southern Strategy*, *NATION* (Nov. 13, 2012), <http://www.thenation.com/article/exclusive-lee-atwaters-infamous-1981-interview-southern-strategy>; *see also* Kruse, *supra* note 347, at 6–8, 164, 194 (explaining Southern

respectable than populist racialized rhetoric (e.g., the rhetoric of George Wallace).³⁵¹ Accordingly, the Southern Strategy successfully drew middle-class white voters to the Republican Party throughout the 1970s and 1980s.³⁵²

In tandem with this rhetorical shift from demagoguery to a more abstract focus on rights, brute physical violence gave way to legitimized forms of social control administered through the police, courts, prisons, and the deployment of “law and order” and “broken windows” narratives.³⁵³ Loïc Wacquant refers to these popular-culture narratives as “law and order pornography,”³⁵⁴ expressed in television shows like *Cops*, *Law & Order*, *CSI*, etc. These shows allow the law and order narrative to become a “core civic theater onto whose stage elected officials prance to dramatize moral norms and display their professed capacity for decisive action, thereby reaffirming the political relevance of Leviathan³⁵⁵ at the very moment when they organize its powerlessness with respect to the market.”³⁵⁶ Also in this same time period, the United States became four or five times more punitive, imprisoning scores of mostly poor and minority citizens.³⁵⁷ In this way, conservative rhetoric and action has been able to divert attention away from highly visible and visceral *bodily* depictions of oppression hurting people living in the social and racial underclass. The poor and oppressed living in the nether spaces of society are taken to the mass prison system where they are contained, warehoused, and disappeared from view.³⁵⁸

In response to this invisibilization trend, the biology of inequality directs the eye back toward the palpable injuries suffered by disadvantaged adults and children, simply as a result of living in capitalistic society. Refocusing on the visceral, corporal effects of a previously abstracted and decontextualized system could prove to be highly persuasive.³⁵⁹ Using visual imagery to detail the harm and pain flowing from embodied inequality

conservatism’s successful transformation from bald-faced racist demagoguery to new arguments premised on a rhetoric of “rights, freedoms, and individualism”).

351. KRUSE, *supra* note 347, at 7.

352. *Id.* at 6.

353. See generally MICHELLE ALEXANDER, *THE NEW JIM CROW: MASS INCARCERATION IN THE AGE OF COLORBLINDNESS* 40 (2012) (arguing that a racial caste system in America exists through the targeting of black men through the criminal justice system); Jeffrey Fagan & Garth Davies, *Street Stops and Broken Windows: Terry, Race, and Disorder in New York City*, 28 *FORDHAM URB. L.J.* 457 (2000) (arguing that the “broken windows” theory of policing disproportionately targets people of color living in poorer neighborhoods).

354. Wacquant, *supra* note 313, at 206.

355. Thomas Hobbes’s Leviathan (a biblical sea monster) metaphorically illustrates how a powerful sovereign imposes order and discipline over a mass of citizens. See generally THOMAS HOBBS, *LEVIATHAN* (Christopher Brooke ed., Penguin Classics 2017) (1651).

356. *Id.*

357. *Id.* at 199 n.4, 208.

358. *Id.* at 203–04.

359. See, e.g., Kevin S. Douglas et al., *The Impact of Graphic Photographic Evidence on Mock Jurors’ Decisions in a Murder Trial: Probative or Prejudicial?*, 21 *L. & HUM. BEHAV.* 485, 486

produces an inherently visceral reaction in an audience.³⁶⁰ Visual rhetoric (whether in the form of images or imagistic language), particularly as it is focused on injury to the human body, has the potential to demand a “force [of] reckoning, an active redistribution of knowledge that is different from what had sufficed before.”³⁶¹ Moreover, rhetoric itself operates on an embodied level:

[W]hen rhetoric influences us, it does so in an embodied way, triggering electro-chemical reactions that traverse our neural pathways, [outside] the purview of our conscious thought. Although it sounds like a science fiction concept, the biological and embodied nature of rhetoric is in line with the beliefs of the ancient Sophists, who understood rhetoric to have the same kind of effect on the brain as a drug.³⁶²

Thus, attacking neoliberal thought patterns by using rhetoric that trains our focus on the body operates on two different levels: (1) the subject matter of the rhetoric operates on a visceral level; and (2) the rhetoric itself, in general, has an embodied impact. Using rhetoric that elevates principles of community, care, and nurturance over those of individualism and competition has the potential to forge new collective thought patterns, thereby influencing collective values and policy choices.³⁶³

Thus, as a rhetorical strategy to respond to neoliberalism, progressive scholars and theorists should focus on the body and strive to concretize and surface the tragic uncaringness that underlies neoliberalism. One part of this project is to dredge up and display, through scientific description, the acute bodily infections that occur from living in unforgiving environments of social insecurity and racialized oppression. We can emphasize how years of physical and psychic threats, fueled by racial oppression, have infected countless black men, women, and children, who continue to experience negative mental and health outcomes³⁶⁴ as well as higher rates of mortality than their white counterparts.³⁶⁵

There should also be emphasis on the universal pain inflicted by biological inequality. We now know that white working-class males are also experiencing serious setbacks in mortality, which are best explained by concomitant socioeconomic setbacks that have occurred since the 1970s—

(1997) (gruesome imagery has a special ability to “generate a sense of moral outrage that demands someone be found responsible and held accountable”).

360. *See id.*

361. Harold & DeLuca, *supra* note 346, at 281 (discussing the impact that images of Emmett Till’s corpse had on the public).

362. Lucy A. Jewel, *Neurorhetoric, Race, and the Law: Toxic Neural Pathways and Healing Alternatives*, 76 MD. L. REV. 663, 663 (2017).

363. *See infra* notes 357–65 and accompanying text.

364. *See supra* notes 225–67 and accompanying text.

365. ANNE CASE & ANGUS DEATON, MORTALITY AND MORBIDITY IN THE 21ST CENTURY 8–9 (2017), https://www.brookings.edu/wp-content/uploads/2017/03/casedeaton_sp17_finaldraft.pdf.

the disappearance of secure jobs along with the family and social stability that these good jobs bring.³⁶⁶

Unlike other policy arguments seeking to call out neoliberalist logic and advocate interventions for inequality, arguments related to the biological embodiment of inequality bring two new things to the table: (1) the ability to induce moral outrage and (2) scientific legitimacy. The moral outrage (life and death should ignite deep-seated normative reactions) presented by this problem could shock the legal system into a new catalytic state where large-scale collective solutions to societal problems are seriously entertained.³⁶⁷ We are talking about life and death, and death comes more quickly if you are poor, and even more quickly if you are poor and a member of a historically oppressed group. In fact, both Hillary Rodham Clinton and Bernie Sanders referred to the disparate life expectancies between the poor and the rich in their presidential campaigns.³⁶⁸

Rhetorically, when progressives confront the logic of neoliberalism, we might take advice from cognitive scientist George Lakoff. Lakoff identifies two deep-seated metaphors to explain left and right thought patterns in the United States—the strict father and the nurturing mother.³⁶⁹ Neoliberal or conservative logic mostly relies on the strict father metaphor—the individual who fails does so as a result of his own poor choices or weakness in the face of competition; that individual must be disciplined.³⁷⁰ More progressive policies draw upon the nurturing mother metaphor.³⁷¹ Lakoff advises progressive political advocates to frame the government in the role of the empathic mother and emphasize that the “first responsibility of the government is to protect and empower its citizens” through an “ethics of care.”³⁷² These two family metaphors, though they are oppositional, are deep-seated and embodied in our minds.³⁷³ Most humans respond to both of these metaphors.³⁷⁴ One side can gain a cognitive rhetorical advantage by repeatedly emphasizing one metaphor, or frame, in a way that trumps the other frame.³⁷⁵

366. *Id.* at 2, 8, 29–34.

367. GEORGE LAKOFF, *THE POLITICAL MIND: A COGNITIVE SCIENTIST’S GUIDE TO YOUR BRAIN AND ITS POLITICS* 53 (2008) (“To get the public to adopt progressive moral positions you have to activate progressive moral thought in them by openly—and constantly—stressing morality, not just the interests of demographic groups.”).

368. See Sabrina Tavernise, *Disparity in Life Spans of the Rich and the Poor is Growing*, N.Y. TIMES, Feb. 12, 2016, <http://www.nytimes.com/2016/02/13/health/disparity-in-life-spans-of-the-rich-and-the-poor-is-growing.html>.

369. See LAKOFF, *supra* note 367, at 76–77.

370. *Id.* at 78–79.

371. See *id.* at 81.

372. *Id.* at 47–48.

373. *Id.* at 82–84.

374. See *id.* at 76–84.

375. *Id.* at 53, 114.

Scientific knowledge produced through empirical data collection and laboratory studies also legitimizes legal and policy arguments.³⁷⁶ The consequences of inequality infect the human body and brain at the genetic and synaptic levels, and are then passed on to subsequent generations. The science lends empirical credibility to the argument that deepening inequality is more than just a policy problem, it is a medical issue that carries far more complexity than a facile narrative of winners and losers, sorted based on inherent “merit.”

Here, the science on embodied inequality could generate novel legal arguments that might return us to a jurisprudential time when large-scale collective solutions to social problems were both entertained and implemented. As Professor Jack Balkin has stated, individualism and communalism constitute the most important pair of opposed ideas in legal and moral thought.³⁷⁷ So many legal quandaries turn on how much responsibility to give to the individual versus how much responsibility the community should have collectively.³⁷⁸

Progressive scholars generally advocate that the legal continuum should swing more in the direction of collective responsibility over individual responsibility. For instance, such a collective approach is found in Martha Fineman’s vulnerability theory, that the state should become more responsive and actively step in and protect its citizens, who are all vulnerable.³⁷⁹ Here, Professor Fineman’s vulnerability theory dovetails nicely with the embodiment theories discussed in this Article. For Professor Fineman, we humans are inherently vulnerable because of our embodiment; we are always susceptible to bodily harm and injury from accidents and catastrophes.³⁸⁰ Similarly here, the theories of epigenetics and neuroplasticity are aligned with Professor Fineman’s theory. Because our bodies and brains are shaped by negative material- and social-environmental conditions and, in many instances, these conditions exist outside of our control, the biological embodiment of inequality substantiates a theory that we are all vulnerable and we all need protection. In accordance with Professor Fineman’s theory, collective solutions, delivered by a robust state, can and should ameliorate some of the conditions that can get under the skin. The next section considers how embodied inequality might support policy initiatives grounded in a collective approach to solving large-scale social problems.

376. See Timothy Zick, *Constitutional Empiricism: Quasi-Neutral Principles & Constitutional Truths*, 82 N.C. L. REV. 101, 162 (2003) (quoting MICHEL SERRES & BRUNO LATOUR, *CONVERSATIONS ON SCIENCE, CULTURE AND TIME* 87 (1995)).

377. Jack M. Balkin, *The Crystalline Nature of Legal Thought*, 39 RUTGERS L. REV. 1, 12–13 (1986).

378. *Id.* at 16.

379. Martha Albertson Fineman, *The Vulnerable Subject: Anchoring Equality in the Human Condition*, 20 YALE J.L. & FEMINISM 1, 9–10 (2008).

380. *Id.*

B. Policy Responses

This Part of the Article focuses on potential policy choices that could be pursued to reverse the bodily harm that is so highly correlated with economic, social, and racial degradation. Scientists have theorized that epigenetic and neuroplastic changes to the body can be reversed by altering an individual's environment.³⁸¹ If the material environment can be shifted from punitive to curative, then there is the possibility of healing. Drawing upon the scientific theory, if we can change the material environment to give individuals more of a sense of control, this will decrease the acute levels of stress responses associated with living with no control. Accordingly, the ill health effects discussed *supra*, might be reversed.

With this general end in mind, Section II.B.1. discusses small-scale solutions and Section II.B.2. discusses large-scale structural changes that would improve disparities in biological outcomes.

1. Small-Scale Solutions

Small-scale solutions would be aimed at improving environments at the individual and family level. The goal is to teach and build mental and neural pathways that are resilient, that are better able to handle the types of stress that can become embedded.³⁸² For example, the Perry Preschool Project studied the effects of parent coaching in the form of weekly home visits with participating families (low SES).³⁸³ At three and four years old, children in this project also attended a free, high-quality preschool program.³⁸⁴ The Perry Preschool Project's robust intervention resulted in short-term improvements in cognitive performance, long-term impacts on high school graduation rates, self-sufficiency, and reduced incarceration.³⁸⁵ Unfortunately, few of the thousands of programs based on the Perry Preschool Project have been able to replicate its structure and benefits (for funding reasons or otherwise).³⁸⁶

Similarly, in response to its black infant-mortality crisis,³⁸⁷ Milwaukee has initiated Blanket of Love, a program of nurse home visits to black

381. Kuzawa & Sweet, *supra* note 1, at 11 (discussing epigenetics); McEwen & Gianaros, *supra* note 1, at 198–99 (discussing neuroplasticity). There is also reason to believe that epigenetic and neuroplastic changes can be reversed with drugs or hormones. See McEwen & Gianaros, *supra* note 1, at 190, 198–99. For moral and ethical reasons (a discussion of which is beyond the scope of this Article), the policy of injecting disadvantaged individuals with drugs or hormones to try to reverse embodied inequality is not an ideal solution.

382. See, e.g., Nat'l Sci. Council on the Developing Child, *Supportive Relationships and Active Skill-Building Strengthen the Foundations of Resilience* 1 (Ctr. on the Developing Child at Harvard Univ., Working Paper No. 13, 2015), http://developingchild.harvard.edu/resources/reports_and_working_papers/working_papers/wp13.

383. Jack P. Shonkoff, *Changing the Narrative for Early Childhood Investment*, 168 JAMA PEDIATRICS 105, 105 (2014).

384. *Id.*

385. *Id.*

386. *Id.*

387. See *supra* notes 236–47 and accompanying text.

mothers residing in high-poverty areas, an initiative that has proven effective in other cities.³⁸⁸ Women living in poverty in Milwaukee face “a particularly brutal slate of risk factors and stressors” that make pregnancy and parenting deeply challenging.³⁸⁹ Blanket of Love sends out nurses and other professional caregivers to engage with at-risk pregnant women and “wrap the pregnant woman up in love.”³⁹⁰ The volunteers in this program intervene to reduce the stressors correlated with the conditions of racialized poverty by finding women homes or furniture; helping them feel confident in talking to doctors; providing education on safe sleeping conditions; or in some cases, helping women escape abusive relationships.³⁹¹ While these individualized interventions have been shown to help, those who are studying the infant mortality crisis in Milwaukee note that much of the problem goes beyond the individual level and touches on large-scale issues like mass incarceration, housing precarity, and the cloud of racism and bias that infects everyday experiences at work and other settings (like Ob-Gyn doctor’s offices).³⁹²

Small-scale solutions should be pursued, but not at the expense of larger scale solutions, as politically difficult as those solutions may be. Embodied inequality cannot be solved with prenatal support, parental education, or cognitive stimulation alone.³⁹³ While embodied inequality can be reversed, that reversal happens only if the material environment is substantively changed. This supports the adoption of large-scale structural remedies.

2. Large-Scale Solutions

This section of the Article proposes policy solutions, broad in scope, that would strengthen social security in the United States and give large portions of citizens a greater sense of stability and control over their lives and their children’s lives.

Large-scale solutions should be aimed at reducing poverty and persistent racial inequality and bias. In the context of this Article, poverty concentrates stress at toxic levels by creating unpredictable adversity that leaves people unable to respond. The resulting lack of control, at the individual and societal level, in turn releases ruinous toxins in the body and mind.³⁹⁴ Those with racial disadvantage suffer these effects twofold, the so-called double jeopardy of poverty and racial disadvantage.

A move toward social democracy, with a massive shoring up of social-welfare programming, would ameliorate the stress that stems from the

388. Carpenter, *supra* note 26.

389. *Id.*

390. *Id.*

391. *Id.*

392. *Id.*

393. Lende, *supra* note 23, at 194.

394. See *supra* notes 23–24, 145–50 and accompanying text.

unbridled uncertainty of living in poverty. The state should broadcast the message that we will catch you if and when you fall. It is worth noting that citizens in Greece, with a much lower gross national product per capita but a much stronger social-welfare support system, enjoy a higher life expectancy than citizens in the United States.³⁹⁵ Thus, an inference can be drawn that robust social security systems positively impact lifespan numbers.

Beyond the project of making the United States look more like Western Europe (the success of which, in this climate, is politically dubious), other large-scale initiatives³⁹⁶ that would give individuals more control over their lives include the following:

- *A living wage*—a living wage would reduce stress by giving individuals certainty over how and whether they will be able to support their families.³⁹⁷
- *Paid family and sick leave policies*—these policies would reduce stress for workers by eliminating having to choose between sustaining income and being able to care for loved ones.³⁹⁸
- *Universal healthcare*—having access to healthcare increases certainty and a sense of control over unpredictable health issues; moreover, unsurprisingly, the underinsured and uninsured have a higher chance of dying than the privately insured.³⁹⁹
- *An end to mass incarceration*—mass incarceration leads to disenfranchisement; civic alienation; and exclusion from employment, housing, education, and other benefits, all of which negatively impact the health of those affected. These negative health consequences likely impact the incarcerated and their partners and children.⁴⁰⁰

III. LEGAL SOLUTIONS

As addressed in this Part, the scientific theories discussed in this Article can be applied to generate novel constitutional theories concerning equal protection. The biology of inequality is relevant for considering

395. Marmot, *Status Syndrome*, *supra* note 2, at 153.

396. See McEwen & Gianoros, *supra* note 1, at 210 (“[B]asic education, housing, taxation, setting of a minimum wage, and addressing occupational health and safety and environmental pollution regulations are all likely to affect the brain and health via a myriad of mechanisms.”).

397. COUNCIL ON CMTY. PEDIATRICS, AM. ACAD. OF PEDIATRICS, POVERTY AND CHILD HEALTH IN THE UNITED STATES, PEDIATRICS 4, 7 (2016).

398. CURTIS SKINNER & SUSAN OCHSHORN, NAT’L CTR. FOR CHILDREN IN POVERTY, PAID FAMILY LEAVE: STRENGTHENING FAMILIES AND OUR FUTURE 5–6, 9 (2012).

399. Andrew P. Wilper et al., *Health Insurance and Mortality in US Adults*, 99 AM. J. PUB. HEALTH 2289, 2292 (2009).

400. See Krieger, *An Ecosocial Approach*, *supra* note 16, at 938 (discussing the negative biological impact of mass incarceration); Carpenter, *supra* note 26 (discussing the work of Arline Geronimus, who theorized that pregnant women who experience the incarceration of a partner or spouse experience chronic stress, which releases harmful chemicals, which are then passed to her child in utero).

whether being poor equates to being in a suspect class, which would trigger higher levels of scrutiny for government discrimination.⁴⁰¹ The science is also relevant for determining whether robust governmental remedies for past discrimination are appropriate, if that discrimination can be biologically traced.

From a more specific standpoint, the science might be applied to reform the legal structures that undergird workplace law and public-education law. In the context of work, more worker protection would provide families and children shelter from the stress of living without control, which would in turn ameliorate many of the biological effects of disadvantage.

Public education is relevant to this Article because initiatives that foster stable and integrated public schools correlate with positive collateral effects in the material environment (reduced pockets of concentrated poverty, more residential integration).⁴⁰² Thus, a strong inference can be drawn that good, integrated (racial and socioeconomic) public schools can slow down or halt some of the detrimental biological effects mediated by disadvantaged living situations.

A. Constitutional Jurisprudence

Two areas of U.S. constitutional jurisprudence could be impacted by the scientific theories discussed in this Article. First, the science supports arguments that impoverished people might be considered a suspect class for the purpose of equal protection analysis. Second, if and when racial harm becomes biologically traceable, this will challenge existing Supreme Court holdings restricting the use of race to remedy past and continuing racial harm. Further development of these arguments will hopefully appear in a subsequent article. In particular, the biology of inequality supports new arguments: that (1) poverty should be considered a suspect characteristic; and (2) existing jurisprudence concerning race remedies (affirmative action, reparations) can be discarded because racial harm may soon become biologically traceable.

With respect to poverty, the Supreme Court has definitively held that impecunity is not a suspect class characteristic.⁴⁰³ In *San Antonio Independent School District v. Rodriguez*,⁴⁰⁴ Justice Powell, writing for the majority, held that poor people, as a class, are “not saddled with such disabilities, or subjected to such a history of purposeful unequal treatment, or

401. See *supra* notes 392–95 and accompanying text.

402. See *infra* note 489 and accompanying text.

403. *San Antonio Indep. Sch. Dist. v. Rodriguez*, 411 U.S. 1, 19–22, 25 (1973). Julie Nice wrote a masterful critique of the Court’s holding on this point. Julie Nice, *No Scrutiny Whatsoever: Deconstitutionalization of Poverty Law, Dual Rules of Law, & Dialogic Default*, 35 *FORDHAM URB. L.J.* 629, 646–47 (2007).

404. 411 U.S. 1 (1973).

relegated to such a position of political powerlessness as to command extraordinary protection from the majoritarian political process.”⁴⁰⁵

As this Article has described, the new science of epigenetics—and to a certain extent, neuroplasticity—roundly challenges the Court’s reasoning on this point. The new science indicates that persons living in poverty do become saddled with persistent disabilities, as the ill effects of deprived material environments seep into the body and mind and get passed on, through biology (i.e., in utero exposure to stress hormones) and physicality (repeated early exposure to toxic material environments). While some might incorrectly construe the science as supporting a socially determinist argument that poor people suffer because they are innately weak, these new scientific theories reject that argument out of hand. The persistent biological suffering discussed in this Article primarily results from the environment that one is born into and comes of age into, rather than from any kind of innate characteristic or individual choice.

Under current Supreme Court jurisprudence, state actors cannot use race in affirmative action programs to heal and repair the effects of past racial discrimination unless a specific constitutional or statutory violation has been shown.⁴⁰⁶ In *Bakke*, Justice Powell, writing for a plurality, reasoned that “[t]here is no principled basis for deciding which groups would merit ‘heightened judicial solicitude’ and which would not. Courts would be asked to evaluate the extent of the prejudice and consequent harm suffered by various minority groups.”⁴⁰⁷ But what if it becomes possible to biologically trace the harm that one group has inflicted on another? Multiple scientists are developing the theory that the racial and social environment has become biologically embedded within the black American populace.⁴⁰⁸ Soon it might be possible to trace the biological harm deriving from the atrocities of slavery; the terror of Jim Crow; the despair of discrimination in housing, finance, employment, education, militarized policing, and incarceration; and the stress of daily exposure to interpersonal bias and institutionalized racism. The health gap (infant mortality rates and lifespans) between whites and blacks cannot be explained by inherent genetic variations.⁴⁰⁹ Immigrants to the United States from Africa do not suffer from these same ill-health consequences.⁴¹⁰ The cloud of U.S. culture and institutions continues to infect black citizens in a particular and unique way. If racial harm becomes biologically traceable, that opens up new arguments for the justness and efficacy of reparations. Reparations are no longer a remedy that is too far removed from the original harm because the original harm endures.

405. *Id.* at 28.

406. *Regents of the Univ. of Cal. v. Bakke*, 438 U.S. 265, 302, 308–09 (1978).

407. *Id.* at 296–97.

408. *See supra* notes 225–67 and accompanying text.

409. *See supra* notes 232–33 and accompanying text.

410. *See supra* notes 233–34 and accompanying text.

While the biological traceability concept bolsters the argument that the harm of racism can possibly be traced to a chain of de jure and de facto causes, it does not address the problem of the innocent member of the majority who “bear[s] no responsibility for whatever harm [a minority group member] . . . [has] suffered.”⁴¹¹ At this point, the analysis shifts into narratives of individual responsibility, when a more appropriate analysis might focus on an inquiry into the problem of unequal distributions of *biological* capital.

B. Specific Legal Solutions

This section makes prescriptive arguments and observations about two areas of the law, workplace law and education law, that might be reformed to make positive impacts on material environments. Workplace law might be reformed to give families more certainty and control over their income and work time. Public education can be structured to construct healthy environments that produce positive educational outcomes as well as collateral benefits for the community and the economy. Reform in both of these areas could reshape expectations about work and wages to create more certainty and control and reduce toxic stress.

1. Workplace Law

Biologically embedded stress derives, in great part, from a perceived lack of control over one’s life.⁴¹² In the context of working-poor and impoverished people, embedded stress is exacerbated by neoliberalist work policies that, by their very nature, create a deep sense of uncertainty and unpredictability. As Wendy Brown writes, neoliberalism places every individual, no matter what the individual’s particular contextual circumstances, in the role of a capital agent, responsible for the individual’s own wellbeing in the market.⁴¹³ The logic of neoliberalism holds that because everyone is an *independent* entrepreneur and capitalist, there is no need for anyone to be *dependent* upon collective support structures.⁴¹⁴ In a system where every person is for themselves, people do not know whether or not their work will earn them enough income to pay for shelter and food.

Low-wage workers, in particular, struggle with a lack of control over working hours, work time, and the overall security of their jobs. This lack of control affects the stability of the environment that parents are able to provide for their children,⁴¹⁵ which can produce long-term physical and mental health damage. Thus, workplace laws could be changed to provide more certainty to low-wage workers. As mentioned above, a living wage would go far, but laws could also mandate a certain number of consistent

411. *Bakke*, 438 U.S. at 310.

412. *See supra* notes 23–26, 140–45 and accompanying text.

413. BROWN, *supra* note 302, at 22.

414. *See id.* at 22–23, 30, 34–37, 40–41.

415. LEILA MORSY & RICHARD ROTHSTEIN, ECON. POLICY INST., PARENTS’ NON-STANDARD WORK SCHEDULES MAKE ADEQUATE CHILDCARE DIFFICULT 2 (2015).

hours; more heavily regulate just-in-time or on-call employment practices; or enact more across-the-board protections for workers, such as shifting some at-will work relationships into more protected positions.

For instance, the just-in-time or on-call aspects of labor practices under neoliberalism exacerbate the lack of control experienced by low-wage workers. With just-in-time or on-call labor practices, employers use big-data algorithms to dictate when workers may work and get paid, or not work and not get paid.⁴¹⁶ These scheduling systems are emblematic of neoliberalism's dedication, in the name of flexibility, to slashing all possible expenses deriving from human labor, "allowing [companies] to staff stores during busy times and save on payroll during slow days."⁴¹⁷ Under this system, workers might make childcare and travel arrangements to get to work, only to be told, hours before the shift is to start, that there are not enough customers available to justify the employee's presence.⁴¹⁸ Not knowing whether one will have hours to work during a day, whether one's child care arrangements are for naught, or the amount of one's paycheck creates the exact kind of loss of control that fuels chronic stress that then becomes embodied.

The end result for people operating in this environment is stress and anxiety, total uncertainty over the things—income, health, shelter—necessary to sustain life. Creating a sense of control, in this context, involves creating more predictability and less uncertainty in terms of the job itself as well as work hours.⁴¹⁹ Unionized jobs offer better protections to workers in terms of stability of work schedules, and some states now require employers to provide minimum pay and pay guarantees for employees called in to work.⁴²⁰ But more stringent federal regulation of workplace policies would go further.⁴²¹ Guaranteed minimum hours of pay and required stability in employee scheduling would give low-wage workers the certainty to both work and plan family care.

416. Lauren Weber, *Retailers Are Under Fire for Work Schedules*, WALL ST. J., Apr. 12, 2015, <http://www.wsj.com/articles/retailers-under-fire-for-work-schedules-1428890401>.

417. *Id.*

418. *See id.*

419. For stressful events, predictability functions as a mitigating factor. People who experience anxiety producing events (i.e., shocks administered in a psychological experiment) feel less distress when the events are predictable. Randy Katz & Til Wykes, *The Psychological Difference Between Temporally Predictable and Unpredictable Stressful Events: Evidence for Information Control Theories*, 48 J. PERSONALITY & SOC. PSYCHOL. 781, 781 (1985); *see also* B. Kent Houston, *Control Over Stress, Locus of Control, and Response to Stress*, 21 J. PERSONALITY & SOC. PSYCHOL. 249, 255 (1972) (noting study subjects "found a threatening situation in which they had no control [the ability to stop the administration of electric shocks by performing well on a test] more anxiety provoking than one in which they had some control over the situation").

420. *See* Charlotte Alexander, Anna Haley-Lock & Nantiya Ruan, *Stabilizing Low-Wage Work*, 50 HARV. C.R.-C.L. L. REV. 1, 19–30 (2015).

421. Such an approach would require amendment of the Fair Labor Standards Act (FLSA), 29 U.S.C. §§ 201–14 (2012), to mandate "uniform protection for all workers and further incentivize employers to minimize instability in low-wage work," Alexander, Haley-Lock & Ruan, *supra* note 420, at 35–37. Or, the Department of Labor could adopt a new interpretation of the FLSA that would treat

Precarity in work—not knowing if one is going to continue to have a job or not—is stressful. A more radical (but not unthinkable) approach would be to consider moving U.S. law from an at-will model—a worker can be terminated for no reason or any reason—to a for-cause model—a worker can only be terminated for good cause.⁴²² In the United States, by virtue of private-law protection, privileged individuals (high-ranking corporate workers, tenure-track professors, etc.) are often sheltered from arbitrary employment terminations. In the United States, as elsewhere, privileged individuals live longer than the less privileged.⁴²³ In other high-income countries, more levels of workers receive protection from arbitrary employment termination.⁴²⁴ U.S. citizens live shorter lives than citizens residing in these other high-income countries that have more substantive protection from job termination.⁴²⁵ The connection between at-will employment and overall life expectancy, at this point, is supported by inference rather than causation. But as further research is conducted, it is probable that further correlations between employment structures and health outcomes will emerge. Returning to a wide-lens focus, for-cause employment would increase certainty and a feeling of control in employees. And the presence of certainty and control is associated with better mental and physical health in the long- and short-term.⁴²⁶ There will, of course, be points of critique that this kind of labor realignment will come at too great an economic cost. The reasoned response would be to study and analyze the economies of those Western European countries (e.g., Germany) that offer robust forms of protection to their workers and enjoy economic health.

2. Education Law

Public education is relevant to this Article because it necessarily engages with the material environment and it is still considered a public good. Whether this sentiment derives from a liberal understanding that

workers scheduled using just-in-time practices as “on call” workers. “For these workers, the hours spent waiting to be called to work would be compensable.” Alexander, Haley-Lock & Ruan, *supra* note 420, at 37.

422. CHARLES J. MUHL, BUREAU OF LABOR STATISTICS, THE EMPLOYMENT-AT-WILL-DOCTRINE: THREE MAJOR EXCEPTIONS (2001); *see also* Moshe Z. Marvit & Shaun Richman, *American Workers Need Better Job Protections*, N.Y. TIMES, Dec. 28, 2017, <https://www.nytimes.com/2017/12/28/opinion/american-workers-job-protections.html>.

423. *See* Marmot, *A Challenge to Medicine*, *supra* note 6.

424. *See generally* Samuel Estreicher & Jeffrey Hirsch, *Comparative Wrongful Dismissal Law: Reassessing American Exceptionalism*, 92 N.C. L. REV. 343 (2014) (describing the employment laws of other countries).

425. One study explained this disparity in the fact that in the United States, more individuals die from accidents, particularly from prescription opioid overdoses. *See* Andrew Fenelon et al., *Major Causes of Injury Death and the Life Expectancy Gap Between the United States and Other High-Income Countries*, 315 JAMA 609, 610 (2016). The higher levels of prescription opioid overdoses are, however, connectable to declining levels of social security, particularly within middle-class white men. *See* CASE & DEATON, *supra* note 365, at 8.

426. *See supra* note 408 and accompanying text.

public education functions for the collective good or a neoliberal understanding that it enables individuals' entry into the market, the bottom line is that the state, in administering public schools, is authorized to design healthy environments for the schoolchildren that pass through its doors. Despite the neoliberalist line of thought that collective social solutions are too impossible to administer,⁴²⁷ public school environments remain one of the only public spaces that the state (local or federal) orchestrates. Public school environments, in this sense, might function as an oasis from the harsh effects of a child's disadvantaged home and neighborhood environment.

As explained below, when school districts can sustain racial and economic integration, which occurs when collective interests for all schoolchildren are pursued over the interests of wealthy individual families who wish to select their public school based on where they live, school districts are able to produce biologically healthy material environments, in the form of more stably integrated neighborhoods, smaller pockets of high-poverty areas, and economic growth.

The optimal public school educational environment is an integrated one, on economic and racial axes.⁴²⁸ Despite *Brown v. Board of Education*,⁴²⁹ many public U.S. schools remain racially and economically segregated,⁴³⁰ and the environments within them function like petri dishes for the physical, mental, and social ills wrought by disadvantage.⁴³¹ Segregated schools also impact the cognitive performance of the schoolchildren that are sheltered within their doors, visible in persistent achievement gaps between white students and black and Hispanic students, and between middle-class and poor students.⁴³² As an example of a curative material environment, "integrated schools boost academic achievement (defined in terms of test scores, attainment (years in school and number of degrees),

427. Blalock, *supra* note 303, at 94.

428. See JAMES COLEMAN ET AL., NAT'L CTR. FOR EDUC. STATISTICS, ED012275, EQUALITY OF EDUCATIONAL OPPORTUNITY SURVEY (1966); HALLEY POTTER ET AL., THE CENTURY FOUND., A NEW WAVE OF SCHOOL INTEGRATION 1, 4 (2016); Myron Orfield, *Milliken, Meredith, and Metropolitan Segregation*, 62 UCLA L. REV. 364, 424 (2015).

429. 347 U.S. 483 (1954).

430. Orfield, *supra* note 428, at 420–22.

431. See Angela Valenzuela, *Ogbu's Voluntary and Involuntary Hypothesis and the Politics of Caring*, in MINORITY STATUS, OPPOSITIONAL CULTURE, AND SCHOOLING 496, 496, 498, 503–04 (John U. Ogbu ed., 2008).

432. POTTER ET AL., *supra* note 428, at 4–5 (discussing black/Hispanic and white, as well as poor and middle-class, achievement gaps); Orfield, *supra* note 428, at 424–26 (explaining how integration boosts academic achievement). In a podcast, public education researcher Nikole Hannah Jones explains that the one thing that "really worked, that cut the achievement gap between black and white students by half" was "the one thing that we are not really talking about, and that very few places are doing anymore." *The Problem We All Live with – Part One*, THIS AM. LIFE (July 31, 2015), <https://www.thisamericanlife.org/radio-archives/episode/562/transcript>. That one thing is integration. *Id.*

and expectations), improve opportunities for students of color, and generate valuable social and economic benefits (better jobs with better benefits, greater ease of living, and diverse future work environments).”⁴³³

Segregated public schools also engender, in a cascade effect, deprived material environments in the form of ailing cities and dying neighborhoods. Professor Myron Orfield has masterfully shown that deeply segregated school systems (mostly a result of the withering away of Supreme Court remedial jurisprudence and white-flight demographic shifts) destroy communities and deter economic growth.⁴³⁴ Specifically, in his study of two school districts (Detroit and Louisville), Professor Orfield described Detroit’s trajectory after the Supreme Court’s *Milliken v. Bradley*⁴³⁵ decision, which rejected district-wide integration efforts.⁴³⁶ Professor Orfield then described Louisville, which had successfully adopted a district-wide, metropolitan integration plan.⁴³⁷ A few months after *Milliken*, the Sixth Circuit distinguished Louisville from Detroit (in part because Louisville only had two school districts whereas Detroit had fifty-three) and allowed a metropolitan-wide remedy to stand.⁴³⁸

After *Milliken*, Detroit schools became acutely segregated.⁴³⁹ In 2000, for instance, the average Detroit student attended a school that was ninety-eight percent black.⁴⁴⁰ This return to segregation was driven in great part by white flight.⁴⁴¹ Whites fled inner city Detroit for school districts in white suburbs.⁴⁴² The loss of population within Detroit decimated its tax base and its school system.⁴⁴³ One hundred Detroit schools closed, the school board had to be taken over by an emergency manager, and the city itself had to be taken over by the state of Michigan.⁴⁴⁴ Detroit’s economy sank into failure.⁴⁴⁵ Residential segregation also worsened. In the 1990s and 2000s, Detroit’s inner-ring suburbs briefly became more diverse as blacks moved out of the inner city.⁴⁴⁶ Within a few years, however, ethnic minorities comprised the primary demographic as whites fled

433. Orfield, *supra* note 428, at 424–25.

434. *Id.* at 368.

435. 418 U.S. 717 (1974).

436. *Id.* at 756; Orfield, *supra* note 428, at 447–62.

437. Orfield, *supra* note 428, at 447–51.

438. *Id.* at 417–18 (citing *Newburg Area Council, Inc. v. Bd. of Educ.*, 510 F.2d 1358 (6th Cir. 1974), *cert. denied*, 421 U.S. 931 (1975)).

439. By 2010, Detroit schools were “the third most racially segregated among the nation’s fifty-largest regions.” Orfield, *supra* note 428, at 447.

440. *Id.*

441. *Id.* at 371–72, 436–40, 458–59.

442. *Id.* at 371.

443. *Id.* at 451.

444. *Id.* at 453–55.

445. *Id.* at 451–52.

446. *Id.* at 456–57.

further toward the exurbs.⁴⁴⁷ Those inner-ring suburbs are now sites of extreme poverty.⁴⁴⁸ Detroit's trajectory matches those of other large cities with multiple metropolitan school districts and without a county-wide integration plan.⁴⁴⁹

By adopting and administering a county-wide integration plan, Louisville was able to block the incentives for white flight. Because the broad integration plan impacted all of Jefferson County, there were no white municipal enclaves to retreat to.⁴⁵⁰ Including most of white suburbia in the metropolitan-wide plans also ensured that schools, in large areas of the county, would remain majority middle-class and majority white, though integrated.⁴⁵¹ After Louisville's integration plan was implemented, Louisville became the eleventh-most-racially-integrated school district among the nation's top fifty regions.⁴⁵² In Louisville, the average black student went to a school that was fifty percent white.⁴⁵³ After Louisville adopted its plan, academic performance improved for black American students.⁴⁵⁴ Tellingly, black American students in Louisville scored much higher on reading and math than Detroit students did.⁴⁵⁵ Louisville's neighborhoods grew more stably integrated and its economy boomed.⁴⁵⁶ Integration in its schools enabled Louisville to support the growth of healthier educational and residential environments.

Despite the palpable harm produced by segregated schools, the project of public school integration in the United States remains a deeply rooted, "wicked problem."⁴⁵⁷ An in-depth exploration of this topic is beyond the scope of this paper, but the perspicacious problem of school seg-

447. *See id.*

448. *Id.* at 457. Detroit's residential patterns in its suburbs mirror that within the rest of the nation. "Ferguson[, Missouri,] is a prime example of how concentration of poverty is moving from the inner-city to the suburbs. As recently as 1990, Ferguson was 75 percent white, but by 2010 it was about two-thirds black. The poverty rate shot up from 7 percent to 22 percent over that period. Three out of ten neighborhoods in Ferguson now have poverty rates of more than 40 percent." PAUL A. JARGOWSKY, THE CENTURY FOUND., ARCHITECTURE OF SEGREGATION: CIVIL UNREST, THE CONCENTRATION OF POVERTY, AND PUBLIC POLICY 14 (2015).

449. Orfield, *supra* note 428, at 434–38.

450. *Id.* at 439, 441. After Louisville adopted its integration plan, there was a short period of limited white flight out of the county altogether, but soon thereafter, "enrollment increased and stabilized." *Id.* at 419.

451. *Id.* at 439.

452. *Id.* at 447.

453. *Id.* at 447 (citation omitted).

454. *Id.* at 419–20.

455. *Id.* at 449.

456. *Id.* at 450–52. It helped that Louisville pursued housing integration initiatives in tandem with its school integration plan. *See* Alana Semuels, *The City That Believed in Desegregation*, ATLANTIC, Mar 27, 2015, <https://www.theatlantic.com/business/archive/2015/03/the-city-that-believed-in-desegregation/388532>.

457. A "wicked problem" is a complex problem that cannot readily be solved in a straightforward linear fashion. *See* Judith Welch Wegner, *Reframing Legal Education's "Wicked Problems,"* 61 RUTGERS L. REV. 867, 870–71 (2009).

regation and integration has to do with shifts in the Supreme Court's approach to equal protection in education.⁴⁵⁸ The Court has shifted away from its post-*Brown*⁴⁵⁹ jurisprudence that mandated eradication of public school discrimination "root and branch"⁴⁶⁰ to lifting federal desegregation orders to return schools as soon as possible to local control,⁴⁶¹ hostility for large-scale busing remedies that would support integration between municipal school districts, on the basis that federal courts lack the power to remedy patterns categorized as de facto segregation,⁴⁶² and the striking down of voluntary integration plans using race as a school assignment factor.⁴⁶³ This shift aligns with the contemporary Supreme Court's conservative and neoliberal jurisprudence, which holds that race and class inequality can usually be explained as a product of individual choices and personal preferences.⁴⁶⁴ According to the Court's current jurisprudence, because these forms of racial and social inequality cannot be traced to a specific animus, collective governmental intervention should be blocked.⁴⁶⁵ In addition, the Supreme Court has rejected the argument that education is a fundamental constitutional right and that discriminatory school spending based on the wealth of particular property tax districts violates the equal protection clause.⁴⁶⁶ This means, under federal law, public schools can be unequal based on economic resources received, as long as each school provides a baseline adequate education. The Supreme Court's precedents on integration remedies and school finance also mean that it will be increasingly rare to see a federal court mandate a large-scale integration remedy.⁴⁶⁷ However, some school districts have voluntarily adopted system-wide integration plans that seek to achieve both racial and socioeconomic diversity.⁴⁶⁸ But other metropolitan areas have stuck with the fragmented approach seen in Detroit.⁴⁶⁹

Although Louisville's metropolitan plan started out as federally mandated and disfavored, within a few years, it became popular among a wide majority of parents.⁴⁷⁰ But a small group of parents remained dissatisfied

458. See generally Orfield, *supra* note 428 (exploring the Supreme Court's desegregation decisions and its effect on the schools and cities in America).

459. *Brown v. Bd. of Educ.*, 347 U.S. 483 (1954).

460. *Green v. Cty. Sch. Bd.*, 391 U.S. 430, 438 (1968).

461. Orfield, *supra* note 428, at 420 (citing *Freeman v. Pitts*, 503 U.S. 467 (1992); *Bd. of Educ. v. Dowell*, 498 U.S. 237 (1991)).

462. *Milliken v. Bradley*, 418 U.S. 717, 756 (1974).

463. *Parents Involved in Cmty. Sch. v. Seattle Sch. Dist. No. 1*, 551 U.S. 701, 743–44 (2007).

464. Orfield, *supra* note 428, at 428.

465. *Id.* at 371, 412, 428.

466. *San Antonio Indep. Sch. Dist. v. Rodriguez*, 411 U.S. 1, 19–22 (1973).

467. Remedies in school finance and equal protection have successfully moved forward on the basis of *state* constitutional rights for education and equal process, which can go further than federal rights. See Myron Orfield, *The Region and Taxation: School Finance, Cities, and the Hope for Regional Reform*, 55 *BUFF. L. REV.* 91, 108–09 (2007).

468. POTTER ET AL., *supra* note 428, at 5; Orfield, *supra* note 428, at 438.

469. Orfield, *supra* note 428, at 364.

470. Semuels, *supra* note 456 (contrasting surveys done in the 1970s in which ninety-eight percent of suburban parents opposed Louisville's integration plan with a survey done in 2011 where

with the plan. After some litigation and a potential resolution involving Louisville's plan in the 1990s, in 2003, one parent challenged Louisville's plan as violating the Constitution's Equal Protection Clause on the basis that it gave more choices to black students than to white students.⁴⁷¹ The resulting case, *Meredith v. Jefferson County Board of Education*,⁴⁷² became the companion case to *Parents Involved in Community Schools v. Seattle School District No. 1*.⁴⁷³ A sharply divided Supreme Court, pivoting on Justice Kennedy's controlling concurrence, struck down Louisville's plan on the basis that it used ethnicity too crudely in its assignment plans.⁴⁷⁴ Currently, Louisville has changed its plan so that it makes assignments based on a student's residential census block, which is ranked based on the percentage of minority residents, educational income of adults, and average household income.⁴⁷⁵ Louisville no longer uses race as a stand-alone factor, but it is built into the equation. In addition, Louisville's use of census-block data to make assignments allows it to use SES as a strong factor for school assignments.⁴⁷⁶ SES, unlike race, does not raise the specter of strict scrutiny under federal law.⁴⁷⁷

During the *Meredith* litigation, a wide-ranging coalition emerged to protect the system and the positive economic, educational, and neighborhood effects that had been flowing into Louisville. Louisville's Chamber of Commerce (usually a bastion of conservatism)⁴⁷⁸ submitted an amicus brief in favor of Louisville's plan.⁴⁷⁹ Turning local-control-based federalist arguments upside down, the Chamber of Commerce argued that "[t]he School Board should be allowed to formulate a student assignment plan suitable to the local community to promote racial integration without interference from a federal court."⁴⁸⁰ The brief touted the positive industry and business effects of having an integrated school system and advocated that Louisville had used means narrowly tailored to achieve its compelling

eighty-seven percent of parents supported integration as a goal and eighty-nine percent of parents were satisfied with the quality of their child's education). Some parents disapproved of the plan because it does not guarantee that a child can attend the closest school within their neighborhood and also, in some instances, requires long bus rides. *Id.* This conflict pits the longstanding individualistic view that public schools belong exclusively to neighborhood residents versus the community approach that views public education as a public good that belongs to everybody. See KRUSE, *supra* note 347, at 143–44 (describing white perceptions of integration as an attack on "our" schools).

471. Orfield, *supra* note 428, at 442–47.

472. 547 U.S. 1178 (2006).

473. 551 U.S. 701 (2007).

474. Orfield, *supra* note 428, at 446 (citing *Parents Involved*, 551 U.S. at 782 (Kennedy, J., concurring)).

475. Semuels, *supra* note 456.

476. *See id.*

477. Because wealth is not a suspect class that triggers strict scrutiny, Orfield, *supra* note 428, at 395, there is more leeway in how it can be used to achieve diversity in public and higher education.

478. *See generally* ALYSSA KATZ, *THE INFLUENCE MACHINE: THE U.S. CHAMBER OF COMMERCE AND THE CORPORATE CAPTURE OF AMERICAN LIFE* 61 (2015).

479. Brief Amici Curiae of the Louisville Area Chamber of Commerce, Inc. (D/B/A Greater Louisville Inc.) & Louisville Metro Mayor Jerry E. Abramson in Support of Respondents at 12, *Meredith v. Jefferson Cty. Bd. of Educ.*, 547 U.S. 1178 (2006) (No. 05-915), 2006 WL 2927086.

480. *Id.*

interest of achieving diversity in its schools.⁴⁸¹ The Chamber of Commerce further argued that Louisville's public school system effectively prepared students for work in diverse settings.⁴⁸² In tandem with the probusiness arguments in favor of integration, Louisville citizens voiced progressive arguments, asserting that the integration plan recognized values of "society as a collective group" and "communal benefits."⁴⁸³

In Louisville's effort to maintain its integrated school system, collective values triumphed over the libertarian every-person-for-himself approach. This victory was enabled by a unique confluence of pragmatic business interests and progressive values. In this example, neoliberalist values (promote competition, access to labor markets) aligned with progressive communitarian values (protect the public good). Without giving up on the critical project of dismantling the logic of neoliberalism and untrammelled capitalism, we can still appreciate pragmatic points for policy reform. In terms of practical strategies for obtaining legal support for more curative material environments, a regional and local, rather than federal, situs might be the best approach.⁴⁸⁴ Indeed, vibrantly progressive state court decisions like California's *Serrano v. Priest*⁴⁸⁵ (wealth is a suspect class under the California constitution's equal protection clause)⁴⁸⁶ and New Jersey's *NAACP v. Mount Laurel*⁴⁸⁷ doctrine ("land use regulations should provide a realistic opportunity for decent housing for at least some part of its resident poor who now occupy dilapidated housing")⁴⁸⁸ provide a template for progressive reform at the state and local level.

A return to the Warren Court's invigorated approach to federal rights would be ideal as it would further substantive equality for all. But nostalgia will not change the Supreme Court's current composition and deeply conservative jurisprudence. Thus, federalism offers a vessel of hope, imperfect as it is. Local and regional approaches are, at this point, a place where substantive, structural change might be accomplished and maintained.

In summary, public-education policy is highly relevant to this Article because education policy correlates with the germination of toxic environments associated with deleterious health and mental effects that are then passed on to subsequent generations. As this Article has shown, living in environments of economic disadvantage and racial discrimination removes an individual's sense of stability and control and generates toxic

481. *Id.* at 4–5.

482. *Id.* at 7–12.

483. Semuels, *supra* note 456 (summarizing the words of one Jefferson County public schools graduate who opposed the dismantling of Louisville integration plan).

484. Orfield, *supra* note 467, at 135.

485. 557 P.2d 929 (Cal. 1976).

486. *Id.* at 951.

487. 456 A.2d 390 (N.J. 1983).

488. *Id.* at 418.

levels of stress that penetrate and interact with gene expressions, stress-hormone systems, and brain structures. All of this combines to produce negative physical-health and mental outcomes, which in turn produce overall worse life outcomes.

Segregated schools are associated with biologically toxic environments; they engender residential racial and socioeconomic segregation, intransigent poverty, and economic stagnation.⁴⁸⁹ Areas of the country that have been able to successfully integrate their schools have enjoyed better residential integration, smaller pockets of concentrated poverty, more robust economies, and better race relations.⁴⁹⁰ In the context of creating positive material environments, public education provides an environment that millions of children spend their days in. Further, public education is one environment that most of the public agree should be a common good, available to all school children.⁴⁹¹ Thus, solving the problem of segregation in schools is one remedy that could ameliorate some of the environmentally mediated biological harm that has been discussed in this Article. And there is hope, in the form of local and regional approaches, supported by alignments between business and progressive interests, that school integration can be achieved and maintained.

CONCLUSION

Whether it is at the genetic level or in the brain, toxic and stressful effects related to poverty and discrimination can “get under the skin.” Embodied inequality challenges traditional narratives that assume that individual genes and individual behavioral choices are the primary causal agents for social outcomes. The violent injustice of embodied inequality (experienced in disparate health outcomes and age spans) can fuel progressive legal solutions that might lessen the harshness of these deleterious biological and health outcomes.

From the standpoint of law-related rhetoric, the biological embodiment of inequality adds, in a very novel way, scientific legitimacy to arguments for remedying structural inequality and poverty. In the framework of George Lakoff, the science affords a rhetorical opportunity to shift the debate toward a frame of collective nurturance and caring, a frame that ultimately has the capacity to heal.⁴⁹²

Potential legal and policy solutions include broad-based solutions that would make the U.S. landscape more socially democratic and more nurturing. Small-scale and large-scale solutions designed to ameliorate the structural conditions that perpetuate poverty and racial oppression should,

489. Orfield, *supra* note 428, at 368.

490. *Id.*

491. See Larry Cuban & Dorothy Shipps, *Introduction* to RECONSTRUCTING THE COMMON GOOD IN EDUCATION: COPING WITH INTRACTABLE AMERICAN DILEMMAS 1, 2 (Larry Cuban & Dorothy Shipps eds., 2000) (tracing the longstanding American belief that “publicly funded, locally controlled schools open to all children would promote the common good and improve society”).

492. See *supra* notes 358–64 and accompanying text.

based on the scientific theories, also heal the biological harms that flow from these wounds. To the extent that studies are able to connect specific biological harm to recurring experiences of racial subordination, these scientific theories support radical jurisprudential approaches, including evaluating whether poverty is a suspect class characteristic and whether race-based remedies, such as affirmative action, can be used to remedy or compensate for past and continuing biological harm, which can be traced to a causal chain of *de facto* and *de jure* discrimination.

On a more discrete level, the science also supports concrete legal remedies applied universally to remedy inequality, such as interventions in the workplace and in public education. In the workplace, this Article suggests enacting changes that would give employees more control and certainty over work. For public education, the point is to promote curative environments, the brick-and-mortar school itself as well as the collateral effects that flow from the presence of good (integrated) schools. With public education, local, state, and regional action might be more pragmatic to achieve these initiatives than reliance on federal rights.

The theories discussed in this Article—that the structure of inequality can become embodied and heritable—raise intense policy and moral questions. The crushing mental and physical consequences suffered by individuals living in disadvantage are now visible through the legitimizing lens of science. While the science of disadvantage is still in a nascent stage, the data set is growing. The stress of poverty and discrimination can literally make one sick. In comparison with more advantaged individuals, a person saddled with inequality's negative health and mental effects does not enjoy a level playing field. In this context, the hyper-individualistic mantra "every man for himself"⁴⁹³ conflicts with the very idea of equal opportunity. The biology of inequality, as developed in this Article, supports the marshaling of collective resources to promote deeper economic and racial equality. Within the longstanding conflict between libertarian individualism and democratic communitarianism, these new theories can shift the pendulum toward potent healing solutions.

493. See MAY, *supra* note 301, at 180.