Modern Nature vs. Nurture: Why the Genetic Lottery and Epigenetics Matter for Social Justice

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Background

The nature versus nurture debate is a long-standing source of controversy between fields of science and those interested in how our personalities take form into the adult life. The phrase was first used in the mid-1800s, but it was recognized that the two parameters were separate and contributed individually to the end result (Serpell). To disentangle the effects of genes and environment, many have attempted various adoption and twin studies, few have been able to distinguish the origin or various traits, but none have found evidence to suggest one side of the argument is the sole determinant of social advancement.

In the nature argument, we have seen how unfairly a strict genetic approach can manifest in society. For example, the Sterilization Act that was passed in Virginia in 1924. In this, legislature sought to prevent "the procreation of persons socially inadequate from defective inheritance. 'Socially inadequate' persons were defined as anyone who 'fails chronically… to maintain himself or herself as a useful member of the organized social life of the state,' as well as the 'feeble-minded,' insane, criminally delinquent, epileptic, alcoholic, syphilitic, blind, deaf, crippled, orphaned, homeless, and 'tramps and paupers'" (Harden). While I would like to assume most people operate on a principle that starts and ends with the respect of all persons, I am cognizant that there are still many people that do not abide by this; however, I do know that most people also know someone, or are related to someone, that is affected by one of the conditions listed in the legislative act. Regardless of where we are as a society on our journey to equality and agreeing that the lives of all matter, we can recognize that at least one, if not all, of the 'inadequacies' listed in the act go against our fundamental beliefs of what makes a human being a human being.

In larger manifestations of the argument, we have seen entire wars dedicated to different eugenic ideologies (as in World War II). In the same vein as the Sterilization Act, eugenic ideology "asserts that there is a hierarchy of superior and inferior human beings, where one's DNA determines one's intrinsic worth and rank in the hierarchy. The social, political, and economic inequalities that proceed from this hierarchy—where the superior get more, and the inferior get less—are, according to eugenic thought, inevitable, natural, just, and necessary" (Harden). In other words, the inequalities that exist in society are no one's fault but the designer of our genes, these inequalities are impossible to avoid, and need not be changed, because everyone, essentially, is put into a lottery where everyone gets one ticket – if you win, you win; but if you lose, you lose out on opportunity for the rest of your life.

Though this is an extreme example of what it looks like to believe in solely the *nature* side of the debate and posit that this is fair and just, it sets up a comparison for what it could look like if we send the *nurture* debate into the extreme. To imagine this, we could imagine a hypothetical world in which everyone grows up in the same environment—same parents, same school, same neighborhood. If we attempt to make this world a reality, a person that strictly believes in the *nurture* argument of this debate may suggest that there would be no inequality in our society. We can recognize that such a world is impossible, not only in idea (if we forced everyone to live exactly alike, we would again be forcing a eugenic idea of 'superiority and inferiority' in the ways that we design what constitutes a good or bad life), but also in outcome. Today, we are exceedingly aware of the role of genetics in various conditions such as depression, down syndrome, and cancer. Thus, even if we all lived the same life, many people would still face hardship unjustly because this 'perfect' world is designed against having any genetic diversity.

Of those that err on the side of *nurture* (i.e. the environment to which we are raised) being the most influential factor in life outcomes, most recognize that even this argument is multi-factorial: it is not your neighborhood or your family dynamics or your educational training, but a combination of all of these things. Although all these components of life outcomes are important and require their own attention, it is imperative to connect our knowledge of genetics with our environment. Genetics is not only the genetic makeup we are born with, but the ways in which our genes change because of factors in our environment. In science academia, this concept is known as epigenetics. Generally, epigenetics is the study of how your behaviors and environment can cause changes that affect that way your genes work. Unlike your genetic makeup, these changes do not alter the genetic sequence, but tells the machinery that it should read differently or at a different rate (think an on and off switch). One important concept in epigenetics is the idea of methylation. Essentially, when certain genes are underutilized or overutilized, methyl groups are added to the molecule of DNA (a carbon with three hydrogens attached to it). If you imagine the reader of DNA being a clamp that fits directly around the DNA molecule, you could also imagine that the DNA cannot as easily be read as these methyl groups are added. Thus, methylated DNA usually represses the transcription of this gene from DNA to RNA, ultimately producing fewer protein products. This idea is extremely prevalent in the field of medicine, as various environmental stressors can add or take away methyl groups that are important for the normal functioning of genes. Smokers, for example, have less DNA methylation at certain gene sites because of ingested chemicals, which can cause the body's cellular responses to start to go haywire, whether that is in the form of wrongly destroying healthy cells or proliferating damaged cells, resulting in tumorigenesis or cancers.

Literature Review

Kathryn Paige Harden is a leader in the field of psychology and behavioral genetics (essentially, how our genetics manifest in our observable actions). Classically, she is known as a pioneer in integrating genetic knowledge with developmental insights into human behavior. In contextualizing her work with the mistakes in history that have been made to perpetuate inequality and false superiority, she attempts to create a new narrative that genetic knowledge can help us to create a more just an equal society. In September 2021, she released a book titled *The Genetic Lottery: Why DNA Matters for Social Equality.* The title of this book suggests she heavily relies upon the genes we are born with to define her argument. While the full book contains many caveats and prefaces to her opinion, I am left with the impression that she leans further on the side of nature in our nature versus nurture debate.

To begin, we must understand her metaphor of genetics resembling a 'lottery'. Having a background in genetics, I am aware of the expansiveness of our genome. In each cell's full DNA, there are approximately 25,000 genes and 3 billion base pairs (individual letters, i.e., ATCG). To put this into further perspective, if you had 3 billion one-inch-wide erasers and lined them up around the Earth, they would wrap the circumference of the Earth just under 2 times—in every single cell. If we multiply this by the average number of cells contained in the human body, our base pair erasers would wrap around the Earth 60 trillion times. While most genes are similar between people, since our bodies complete most of the same day-to-day activities, this large genome size leaves a large amount of room for variability. In the world of statistics and probability, we can calculate that "each pair of parents could produce over 70 trillion genetically unique offspring. And that's before you consider the possibility of de novo genetic mutations: brand new genetic changes that arise in the production of gametes" (Harden). In essence, what

you and I contain in our genomes was a 1 in 70 trillion chance interaction that happened when our parents' gametes fused. You have a 1 in 300 million chance of winning the Powerball jackpot. In a way, our genetic composition is an even more extreme version of playing the lottery, with an even smaller chance of winning the genetic 'jackpot'.

Another important concept in her argument is the idea of polygenic traits. That is, traits that are controlled by more than one gene. While we can imagine one gene controlling our intelligence or swimming speed, what we see is the cooperation of dozens, and sometimes hundreds, of genes that steadily could predict you are more likely to be intelligent or fast in a swimming pool. This applies to many other traits—there is not a single trait that will make you outgoing or depressed—these outcomes are polygenic. With this knowledge in mind, there is an exciting realm of possibility for the field of genetics to produce more favorable life outcomes by identifying which dozens of genes are involved with these outcomes. Harden presents some successes of using genome-wide association studies (GWAS) to predict wealth accumulation: "Among the White, retired septuagenarians in this study, people who were low on the polygenic index (the first quartile) had, on average, $475,000 less wealth than people who were high on the polygenic index (the fourth quartile)" (Harden). When I read these results, I immediately wanted my genome tested to know if I am in the wealthy or unwealthy group, but I think these results are overstated in the words of Harden: "Can we really say that genes cause you to be wealthier? (Short answer: yes)" (Harden). One of the first rules in statistics in science is to avoid using the word "causation" when we really mean "correlation". Thus, although it would be groundbreaking if we knew our genes were the direct influencer of how much money we will attain (although it would ruin the idea of the American dream and intergenerational mobility),

what she really means, I believe, is that we see some sort of correlation between the identified genes and wealth attainment.

To make the work that geneticists are doing even more complex, we have tried to focus on the exact changes that produce these outcomes. Instead of identifying if a gene is a simple "yes" or a "no", we have started to sequence each of these genes to identify single base pairs that differ between people. These single base pair differences are called single nucleotide polymorphisms in the science world (SNPs, for short). As we increased the resolution of what is happening at each of these sites, we were ultimately left with more questions than answers: "Schizophrenia and autism and depression and obesity and educational attainment are not associated with one gene. They are not associated with even a dozen different SNPs. They are polygenic and associated with thousands upon thousands of SNPs scattered all throughout a person's genome" (Harden). From there, we may think that once we identify these sites, we could even more accurately predict life outcomes. In practice, however, the results are even less confident than those from our polygenic index: "In samples of White people living in high-income countries, a polygenic index created from the educational attainment GWAS typically captures about 10-15 percent of the variance in outcomes like years of schooling, performance on standardized academic tests, or intelligence test scores" (Harden). While 10-15 percent is significant, it is not convincing enough to leave with the impression of causation. Rather, we are left with a weak correlational relationship if that (this 10-15 percent could presumably be from a sampling bias or experimental error or mere coincidence).

Regardless of the direct causation of genes into our life outcomes, Harden suggests a sense of moral obligation and calls for action, based on what she knows. Harden introduces her

moral argument by taking a logical approach around what most people consider to be unjust and unfair in our society:

> For many people, the distinction they make between inequalities that are fair and those that are unfair is that unfair inequalities are those tied to accidents of birth over which a person has no control, like being born into conditions of privilege or penury. But there is another accident of birth that is also correlated with inequalities in adult outcomes: not to social conditions into which you are born, but the genes with which you are born.

Immediately, we can recognize that her opinion relies heavily on these 'accidents of birth', which implies that she believes most adult outcomes are a result of what you are given at the start of life. Her societal definition that solves this problem, she suggests, is a society "in which these accidents of birth do not determine a person's fate in life" (Harden). Although this 'solution' offers a hypothetical framework we could strive for, we can easily recognize that such a hypothetical is just that – *hypothetical.* Most of our work today seeks to address the consequences associated with poverty – hunger, homelessness, and the maintenance of human autonomy and dignity for every person. Some work, however, attempts to address the *causes* of poverty in the expanding access of healthcare, job centers, and free resources people can use to give them opportunity they otherwise would not be able to afford or realistically obtain. In Harden's argument, this hypothetical world addresses what she suggests is the ultimate cause of poverty: the genes we are born with, but is this really a cause that we can address (*see analysis*)?

An important part of Harden's argument that makes it more convincing is her willingness to address the common hesitations we experience when trying to adopt a *nature* mindset to poverty and inequality. She recognizes these counter arguments swiftly: "many academics hold the conviction that discussing genetic causes of social inequalities is fundamentally a racist,

classist, eugenic project" (Harden). She strengthens this with the results of a study her lab conducted on the ways people perceive genetic influence on our behaviors: "The study speaks to how common it is for people, particularly when they have liberal political ideologies, to see empirical statements about how genes do influence human behavior as incompatible with moral beliefs about how people should be treated equally" (Harden). We have a long history of eugenics movements creating a worldwide panic, as with Nazi Germany and the Ku Klux Klan. Thus, it is only natural for hesitation to arise when we consider a modern-day eugenics movement, which presumably is what people suspect when we start to equate genetics with life outcomes. The argument posited in this novel, however, seeks to flip these negative perceptions of genetic knowledge and instead suggest the knowledge of genetics and its outcomes in life are important in addressing the inequality that exists within our society. Instead of seeking out this information to control others, we should seek out knowledge, so we are better able to provide support where support is due.

On the other hand, much research has been done to try to identify the ways in which our neuronal firing and wiring change with environmental triggers. Under a strictly biological framework, the goal is to understand the molecular mechanics of the behaviors we produce. Because this field of study focuses on the ways in which our brains change, it is considered the backbone of epigenetic research. One area of study prevalent to poverty, and more specifically, the hinderance that anxiety and depression places on life outcomes, focuses on a signaling molecule in the central nervous system called serotonin (or 5-hydroxytryptamine). Serotonin is the modulator of many critical bodily functions including sleep, mood, anxiety, appetite, memory, and perception. To exert and regulate its effects, the released neurotransmitter must encounter one of its high-affinity transporters, like SERT. The SERT protein comes from a

section of the DNA called the SLC6 gene. The protein itself is relatively large, with 12 transmembrane domains (sections that cross from one side of the cell membrane to the other). Because it is such a large protein, it undergoes many steps before its final product is functional, and each of these steps is highly regulated. When SERT meets a serotonin molecule, it is transported into the cell, increasing the intracellular store of serotonin available for usage.

Although the process of creating a mature SERT protein is highly regulated, there are many points in the process that may lead to a dysregulation in serotonin signaling. This dysregulation is linked to many disorders of the central nervous system including anxiety, depression, obsessive compulsive disorder, and autism spectrum disorder. In order to combat this dysregulation, affected individuals can take medication from a class of selective serotonin reuptake inhibitors (SSRIs, i.e. Prozac), to increase the extracellular availability of serotonin and encourage cells to rake in more for use. However, such medications only improve symptoms in approximately two-thirds of patients, which indicates that the mechanism of the SERT protein is not fully understood (Fekadu et al.). In the past decade, evidence has accumulated that SERT expression and its functions may be regulated epigenetically through DNA methylation, histone (the ‘spool’ which thread-like DNA wraps around) modifications and microRNAs (RNA fragments that attach to messenger RNA to destroy them). In the case of DNA methylation, studies show the methylation of a nearby segment of DNA, which is the promoter for SERT transcription, is associated with a reduced level of SERT (Philibert et al.). With micro-RNA levels, evidence suggests that limiting an overabundance of the micro-RNA specific to SERT has the same effects of antidepressant medications, if there is an overabundance in cells that respond to serotonin (serotonergic neurons) (Baudry et al.). On the other hand, there could be not enough micro-RNAs, resulting in SERT expression in cells that are not required to react to serotonin

(noradrenergic cells). Under this assumption, too many cells would be harvesting serotonin from the extracellular environment, leading to a limited supply of serotonin for the cells that require it to function properly. Because determining which is the cause of depressive or anxiety symptoms would be highly difficult and invasive, it is understandable why the gold standard of prescriptions for either condition is not a solution for every patient.

The mechanism to which epigenetic changes occur on the SERT gene and other "stress genes" is widely studied, to provide a basis for gene-environment interactions that affect mental health. In a study where individuals were exposed to an acute psychosocial stressor, researchers found significantly more DNA methylation on stress-related genes for those in the experimental group. The authors also found that the younger the child, the more significant their DNA methylation response (Unternaehrer et al.). Thus, there is a clear connection between methylation of stress genes and a child's response to poverty. Poverty and its related consequences (food insecurity, homelessness, instability) are often considered chronic stressors to the affected individual. Thus, the epigenetic changes caused to these genes are not due to an acute stressor and reverted to their original state. Instead, a chronic stressor will cause the body's stress genes to remain in a constant state of methylation, which decreases the individual's ability to properly use neurochemicals and maintain a state of mental wellness. It is suggested that as many as 1 in 13 genes are modified in response to childhood poverty (McRae).

To understand how epigenetic changes alter the ways in which we think, let us think of an experiment in which the participant is asked to recall a negative situation, followed by a series of questions to determine their emotional appraisal of the situation. It is thought the person without stress-related marks on their genes would appraise the situation appropriately, understand their ability to cope, and find joyful or goal-building attitudes after the experience.

On the contrary, we would expect those who grew up in the face of chronic stressors with heavily methylated genes to experience more unpleasant emotions. In a study like the one described, the results were as expected – participants with more exposure to stressors "experience negative emotions as more unpleasant, more influential, disruptive on personal goals, and feel less able to cope with these emotions". Specifically, they were more likely to respond to negative situations with fear and sadness, which resulted in fewer problem-solving strategies (Szily et al.). Ultimately, a negative situation further down the line from previous stressors results in a more negative experience for the individual, which is highly linked to depression and anxiety disorders. Thus, the strictly *nurture* argument would suggest that it does not matter which genes you were born with in the genetic lottery, but rather the way these genes are changed throughout one's life would determine their susceptibility to depression and other mental ailments. Presumably, if someone is more prone to depression or poor decision-making, these individuals may also be more likely to be involved in situations that could negatively impact life outcomes (teen pregnancy, drug usage, crime).

Methodology

In the nature argument, Harden primarily relies on the work of social scientists like herself. In social sciences, most times, researchers look at humans at the behavioral level, looking at the outcome of psychological differences between people, rather than the psychological difference itself. Although Harden leans on the side of the nature debate, social scientists in general approach problems with a multiple-factoral mindset (i.e. even if there is a correlation, there may be many confounding variables), generally recognizing the multiple facets of personality in both our genetic makeup, but also in the effects of our environment. Because

social studies are often longitudinal or done outside a lab, they usually do not introduce a new variable like an experiment. Rather, social scientists seek to find correlations between behaviors and personality-types. This method is extremely useful in determining the physical outcomes of various diversities, but fails to perfectly control variables, since most cannot ethically experiment with changing a genome or life experience.

In current psychological-based studies, there also seems to be a lack of diverse representation in participants. “Both twin research and research with measured DNA, has focused almost entirely on understanding individual differences among people whose recent genetic ancestry is exclusively European and who are overwhelmingly likely to identify as White” (Harden). Certainly, the object of studies to find correlation is made much easier by eliminating a diverse set of confounding variables, but this also means the correlations that are found may not reasonably be applied to every person. Because we do not have this comparative data for most studies, we cannot meaningly inform our scientific understanding of social inequalities between racial and ethnic groups. Additionally, without this available data, correlational studies of genetic content and outcomes between racial groups may be limited. Although we may see differences in the specific sequences of DNA, with a limited sample, we may be unable to determine which of these differences in sequence is functionally significant. “Their analysis could be just picking up on population stratification, i.e., on biologically unimportant differences between people from different social classes” (Harden). Ultimately, there is much more work to be done to use the results of such studies as a truth that spans across all walks of humanity.

In the nurture side of this argument, there is a heavy reliance on the work of biochemists who seek to find causational relationships between molecules at the cellular or synaptic level.

While this is an excellent method on the micro-scale, it can be difficult to determine behavioral outcomes of molecular changes. Additionally, to ethically control variables, many researchers must resort to animal-models of human conditions. Because of this, there may be a gap in the data presented and its applications to humanity, especially in the likely event that the organism's behavior is different because of the unnatural setting of a laboratory. Unlike the social scientist, the biochemist has a clear and directed hypothesis before beginning an experiment, but as a result, they may miss alternate solutions to their proposed problem that are on a macro-level (research bias). For example, the studies presented in this paper focus mostly on the stress response's effect on epigenetic changes, however, these changes can also occur from many other aspects of the environment including diet, smoking habits, or air quality (Combs-Orme).

In both fields of study, we must attempt to gain perspective from cells or certain behaviors to its implications on an even broader, societal scale. Harden notes, "A study of what is correlated with succeeding in an education system doesn't tell you whether the system is good, or fair, or just". In this way, it is up to the audience to determine whether an arrangement is equitable and aligns with their values of human dignity and rights.

Analysis

At the core of this argument, my impulse was to make the nature versus nurture debate binary. In a world with so much uncertainty and doubt about matters of self-identity and biology, it is more easily understood when a question can be answered with a strict one or the other; however, I will argue that this debate cannot be settled with only one – perhaps, it is both nature *and* nurture. Regardless, the result of this literature is a call to action to start paying attention to

those that are disadvantaged at either the "genetic lottery" level or the level of having a stressful childhood that predisposes our bodies into harmful developmental patterns.

With the growth of modern technology, a plausible and fair conclusion may be drawn that perhaps we should just "fix" the genes of the people who are less advantaged genetically to make all matters equal. While genetic technologies are excellent in the production of children who will not live a life with a chronic condition or curing certain cancers, we are hesitant to believe in the full range of these technologies because they have previously been incompatible with social equality. In Nazi Germany, genetics was used against the world to advance racist and classist ideologies by eliminating those identified as "less than". In remembering a world that was paralyzed with fear from simply being born with one set of genes versus another, it would feel outlandish to suggest it is feasible (economically, ethically, or otherwise) to simply change the genes of those predisposed to negative mental health outcomes. Ultimately, our genetic differences and diversity is what keeps the human population growing; it is what advances our society cognitively; and it is at the core of enriching our lives with the differences of others. At the end of the day, if we were all the same, the world would be a boring place. Observable differences promote a sense of curiosity, of interconnectedness, and call us to contemplate the notions of fairness and empathy.

Another logical conclusion may be to take a less aggressive approach to *genetic perfectionism*, and instead of altering every "faulty" gene, perhaps we only modify a few. With this approach, we run into a fierce violation of human autonomy and basic principles of well-being. Who is to say which genes are the less desirable ones? Which features do these genes influence that are candidates for adjustment? While most hearing people believe they would not want to be deaf, the deaf community considers their deafness part of an indescribably beautiful

language and culture. The point is, what we consider *good* and *bad* not only differs between one person and another, but *every* person. Each person is a social construct in and of themselves. If we start changing one gene that we collectively decide is faulty, where do we draw the line? This approach also leads us down a path of eugenic beliefs that would ultimately end in a conflict of science and nature.

Although it is a risk to use genetics in the ways I have described, a larger risk is posed if we fail to understand the weight of genetics in our lives and assume the status quo that genetics do not play a role in social inequality. Genetics are a critical component of social inequality that is often swept under the rug because it all feels unfair and random. If one person hits the genetic lottery and scores the twelve most resilient genes in the genome, they will have advantages in navigating many of life's common challenges. On the contrary, if someone sorely loses this genetic lottery and is stuck with the twelve least reliant variants of genes in the genome, are they most definitely going to live a life filled with poverty and inequality? The clear answer here is no, but the question then becomes: how can this second person attain the resilience of the first when they were not born with it? If this theoretical second human being lived a life filled with happiness and abundance and encountered no stress at all, it is quite possible this person would never realize that if something stressful happened, this likely could have sent them down a dark rabbit hole of mental illness.

If we consider a situation somewhere in the middle of these two hypotheticals, we can see how our second model of epigenetics changes could play out in our world. Let us say that there are two people, person A and person B. Both were born with the exact same twelve resilience genes, six good, and six not-so-good. However, person A is fortunate enough to be born into a family and community that grants them every opportunity they need to succeed in life, but

person B was born into a tumultuous environment – family and/or community -- that is plagued with stress, danger, and misfortune. Person A will have few epigenetic changes on their genes to affect the way their brain perceives the world; however, person B's genes have become highly methylated, causing their transcription to change, so that their brain is constantly prepared for the worst to occur. Person B, even if they were given the chance, would likely struggle to change their pattern of thinking and continue to be a highly successful and motivated individual. Depressed brains "prefer" to stay depressed, and without copious mental health resources or a big enough break from the stressors and trauma of their life, this brain will continue processing neurotransmitters in this problematic, and often, destructive way.

Therefore, genetics not only need to be identified as a principal component of poverty but used as a further reason to combat the social inequalities that exist in our world. The inherent idea of everyone being born with a different "luck" of genetics calls us to see the differences in others and do our best to protect each different set of these genes. Rather than hone into the details of genetics and epigenetic changes to genes, we should focus on creating a world where any set of genes could lead to a successful and happy life – eliminate childhood poverty, expand access to mental health resources, and increase awareness of the neural differences that affect our perception of difficult life events. It is not nature *or* nurture, but an inextricable combination and interaction of the two. Ultimately, either approach should lead to the same conclusion – protect people's autonomies and well-beings, despite any genetic misfortune, and work to address the causes and consequences of poverty in the best ways we know how. Additionally, when those with nature on their side face hurdles, they deserve the same care, dignity, and assistance as those who are likely to face chronic challenges because of nature.

Ethics

> *"Knowing what we know now, we cannot pretend that genetics do not matter. Instead, we must carefully scrape away the eugenicists' scientific and ideological errors, and we must articulate how the science of heredity can be understood in an egalitarian framework" (Harden).*

To approach this argument from an ethical framework, I will use Martha Nussbaum's theory on human capabilities and rights. Nussbaum asserts that all humans are of equal dignity and worth and that "the primary source of this worth is a power of moral choice within them, a power that consists in the ability to plan a life in accordance with one's own evaluation of ends" (Nussbaum). Thus, Nussbaum would posit that a loss of this moral choice is a violation of human rights.

In the nature argument, it is difficult to claim an injustice upon people if they receive genes that would score lower in terms of the "genetic lottery" because the genetics we are born with are not due to outside influences such as social structure or government. Someone could certainly say that two people only meet one another through social boundaries and power dynamics, but even then, there are 70 trillion possible genetic outcomes between two people to create one child. Nonetheless, it is an easier argument to propose that an inequitable world is responsible for the epigenetic changes people experience throughout their lives due to their environment (the nurture argument). Although poverty requires a nuanced approach to understand its causes, we can understand that poverty itself causes a chronic level of stress people in higher classes are not usually exposed to. This chronic level of stress, whether it is due to a social institution or policy or fatherlessness (etc.), is the cause of many negative outcomes, including a negative appraisal on situations and a predisposition to anxiety and depression, which

may make it difficult, even much later in life, to sustain a fast-paced career with success. While many would argue there were many choices made along the way, I would argue that these choices are determined by the ways in which we perceive the world, which is directly caused by the regulation of our proteins and genes, made possible by epigenetic marks to our DNA. Thus, I believe Nussbaum would approach this situation and recognize a loss of human rights in the way we age the genes of youth without choice in the matter. The solution, in this case, would be to work to further address poverty to minimize this loss of choice and violation to human rights.

From a utilitarian perspective, there would be many costs associated with reducing poverty to a level of "fairness" by this standard, where no one is exposed to chronic stress because of poverty or homelessness. As a result, the utilitarian may posit this cost outweighs the benefit of eliminating this injustice. The contractualist, on the other hand, recognizes that a violation of a person's human rights and dignity is enough to object to poverty and work to combat it. I believe the contractualist would agree that, even if the cost is high, it would be worth it to diminish poverty to restore the moral choice of the human being.

Discussion

While many are hesitant to lean into the power of genetics to shape our lives, it is an incredibly pervasive part of what makes every human being the ways they are and plays a large role in determining the outcomes of one's life. Though I recognize factors outside of genetic material that determine one's life outcomes, I believe the field of genetics requires more attention from those that seek social equality and justice. The history of our world has taught humans to think of the words "genetics" and "eugenics" together; however, I would consider myself an anti-eugenic, despite my recognition in the power of DNA. We can learn more about

this field of study without conforming to racist and classist beliefs previously held by powerful leaders in our world. To be anti-eugenic, but thrive for a better understanding of genetic material requires us to:

> (1) understand the role that genetic luck plays in shaping our bodies and brains, (2) document how our current educational systems and labor markets and financial markets reward people with certain types of bodies and brains (but not other types of bodies and brains), and (3) reimagine how those systems could be transformed to the inclusion of everyone, regardless of the outcome of the genetic lottery. (Harden)

With these steps, we are pushed to continue to learn more, but use this knowledge as a tool to inform the ways in which we take action to demand justice from the institutions that surround us.

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