Intersection of Epigenetics and Poverty in America

by

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Abstract: With the current pace and progression of epigenetics research in line with the political climate in America, questions of how American politics affect the methylome of people who experience repercussions of politics is of great importance. The chronic stress on people experiencing generational poverty in an American capitalistic society should be questioned - what is its effect on individual and family genomes through epigenetic modifications, specifically methylation? This research explores the impact of chronic stress through poverty on epigenetic modifications and the related health outcomes in environments where American politics facilitates poverty. These research topics, when braided together, illustrate the gravity of the burden of living in poverty and the urgent need for change through research, reform and reconciliation.

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Introduction

In 2018, U.S. Census information showed that one in eight Americans are living below the poverty line (Semega 2019), 38.1million Americans surviving under high economic stress (Semega 2019). These 38.1million people are more than a statistic - they are brothers, sisters, mothers, fathers, community members, grandparents - real people who deserve to be seen and heard. While 38.1million is a disappointingly large number, it is still a conservative image of how many people are under high economic stress (Fisher 1992).

Financial stress even affects people who are living on incomes above the poverty line, as the poverty-line is too low to sustain a healthy life. For a family of four (two adults and two children) the annual income must be at or below $25,425 to be considered living in poverty conditions (Semega 2019). That leaves $2,118 a month to feed, clean, educate, transport, cloth, house and care for four people. Thus a family of four that is spending more than $635 per month on rent is cost-burdened to the point where they must make sacrificial decisions to survive (The State of the Nation's Housing 2019). For example, taking half a dose of medication to make the prescription last longer in order to have enough money left over to buy food. 38.1million bodies are taking the toll of living in high chronic stress that most often accompanies poverty (Semega 2019).

Additionally, these struggles in poverty are disproportionately experienced by people of color and females. In 2010, 26.09% of African American households and 25.9% of Latinx households lived in poverty while only 11.1% of White households experienced the same (Ajinkya 2012). Thus, White people experience poverty half as often as their African American and Latinx neighbors. Similar ratios are seen when women are in focus: in 2018, 22% of Native

American women and 20% of African American women lived in poverty, whereas 9% of White women lived in poverty (Fins 2019). In 2018, 46% of the 15.5 million women living in poverty lived in extreme poverty (at or below fifty percent of the federal poverty level) (Fins 2019). One in three African American children, one in four Latinx children and one in four Native American children lived in poverty in 2018 (Fins 2019). Whereas one in eleven White children lived in poverty (Fins 2019). These disparities show the reality of racism and sexism woven into the fabric of American economies and societies.

It is necessary to communicate that the experience of poverty is not fully reliant on the "bad choices" of individuals who find themselves at the bottom of the socioeconomic ladder in America. To illustrate, some politicians and economists still hold the false idea that national economic growth is the best anti-poverty program (Ryan 2014). This agenda serves to justify making people living in poverty wait around until the American economy grows for prosperity to trickle downward. However, this idea has not manifested since the 1980's (Bivens et al. 2014). From 1959 to 1973, increasing American economic growth predictably correlated with falling poverty rates. During his time, John F. Kennedy was justified in using his argument of using economic growth as a means to fight poverty (Hines et al. 2001). However, data analysis shows that this relationship decoupled in the mid 1970's (Bivens et al. 2014). If the advantageous relationship between economic growth and poverty decline had remained intact, projections show that poverty rates would have reached near zero by the mid 1980's (see Figure 1) (Bivens et al. 2014). The decoupling was and continues to be a result of stagnant wages despite the growth of business profits (Bivens et al. 2014). When a company experiences an increase in profits but chooses not to increase the wages of their workers, economic growth happens without

raising up people living on the least, disparities in wealth become a result of such a free market decision (Schmitt 2012). This data serves to dismantle the all too common assumption that the experience of poverty lies in the "faults" of those who are living the reality of poverty. In truth, people living in poverty work longer hours than most Americans (Bivens et al. 2014) and are seldom rewarded but in some cases are inadvertently punished, through a phenomenon called hustling backwards (Weisberg 2006). For example, when a worker being supplemented by government assistance programs gets a raise, welfare benefits are retracted disproportionately, resulting in a net loss of usable income. In fact, according to the 2005 Annual CEO Compensation Survey, "If the minimum wage had risen as fast as CEO pay since 1990, the lowest paid workers in our country would be earning $23.03 an hour today, not $5.15 an hour" (Anderson et al. 2005). The vast experience of poverty in America is a systemic problem - the result of corporate and policy decisions.

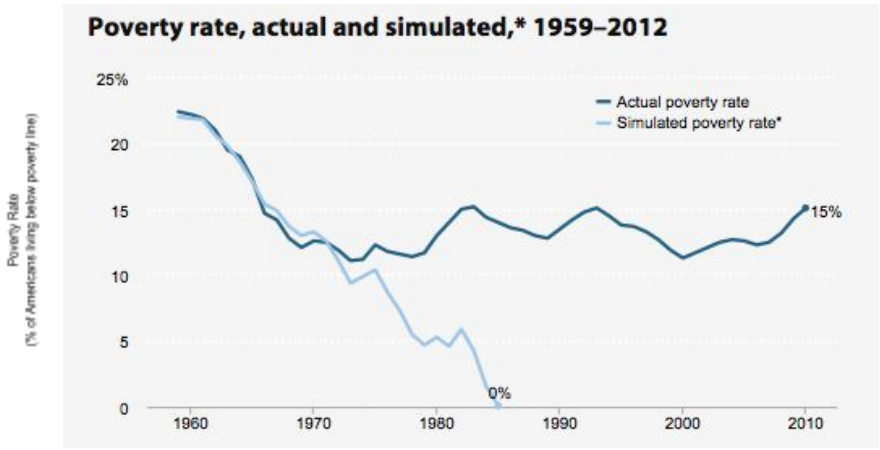

Figure 1. Actual versus simulated poverty rates. From : (Bivens et al. 2014)

Research has shown that living within the economic confines of poverty is often accompanied by poor health outcomes (Braveman et al. 2010; Shea et al. 2016). Low socioeconomic status is associated with higher occurrences of low infant birth weights

(Martinson and Reichman 2016; Larson 2007), intrauterine growth restriction (Larson 2007) and preterm births (Donoghue et al. 2013). In 2012, Dr. Hsu and company found an association between poverty and higher type two diabetes incidence (Hsu et al. 2012). Additionally, heart disease, obesity and lower life expectancy were also found to have the most grim statistics in people living under high financial stress (Braveman et al. 2010). This correlation between health and income shows that health outcomes improve with increasing income (Braveman et al. 2010). The evidence of damage done to people by systems of poverty also show up in individuals' genomes, specifically epigenomes (Murgatroyd et al. 2009). Environments of stress, specifically low household socioeconomic status early in life, have been shown to lead to methylation changes that link to multiple adverse health outcomes such as an increase in inflammation (McDade et al. 2017) and an increase in threat-related amygdala reactivity that is related to higher depression rates (Swartz 2017). Additionally, higher levels of methylation have been correlated with psychopathology, neuropathology and a higher risk of suicide during depression (Haghighi et al. 2014). With these findings considered, it is reasonable to ask what other effects come with poverty and if they can be healed. The remainder of this review will explore the question: how does the chronic stress on people experiencing generational poverty in an American capitalistic society affect individual and family genomes through epigenetic modifications?

Systemic Contributions to Poverty

It is a curious statement to say that homelessness is a choice when American cities have more families than homes (Dinsmore 2007). In the same way, the existence of poverty justified by attributing it to individual choice does not add up with the societal scaffolding that props up

economic unfairness. The landscape of economic policy leading to the abundance of poverty in America is a necessary starting point for research of this kind. Before introducing the heart of this paper, the epigenetic effects of poverty, it is vital to dispel the myth that people experiencing poverty are deservedly suffering due to their own destructive choices. Much of the creation of poverty can be attributed to systems (housing, job market, policy) that benefit from having people at the bottom of the economic ladders; living in poverty.

The plight of extreme financial burden, poverty, is experienced by all racial groups but people of color experience it at disproportionately higher rates. This reality has strong roots in the historical development of this country. African American groups experienced a heightened unequal history. Hanks articulates this reality that the beginning of the African American "experience began with slavery, which allowed whites to profit off of the bodies and blood of enslaved people, who by rule of law were unable to live freely, let alone build wealth to pass along to future generations" (Hanks et al. 2018). The economic hardship that disporportionately affects people of color in America can be traced all the way back to the collective genocide and maltreatment of groups of color.

Racialized Wealth Gap:

Income and wealth together indicate current and future economic security (Hanks et al. 2018). The relationship between wealth and income is very important. The effects of low income can be cushioned by a well of wealth because wealth can be sold for income. If a person or household is suffering from extreme low income with no wealth to fall back on the financial burden is even more impactful. According to Richard Florida with City Lab, more and more research is building to suggest that housing inequality may be the biggest players in America's

economic divide as much of generational wealth is harbored and passed down through home-ownership (Florida 2018). Let it be clear that the history surrounding racism in housing policy is in fact a major feeder of housing inequality. Florida states, “Economic inequality is one of the most significant issues facing cities and entire nations today” (Florida 2018).

The racialized wealth gap that feeds heavily into economic instability and thus epigenetic modifications, did not happen spontaneously, accidentally, or recently. For example, U.S. tax code prioritizes certain assets that statistically White people are more likely to have due to things like historical occupational segregation (Hanks et al. 2018). Additionally, due to systematic housing and mortgage discrimination African Americans are less likely to benefit from building wealth through homeownership (Hanks et al. 2018). Begining in 1865, after the abolition of slavery, exclusionary zoning prohibited the sale of property to African Americans (Lockwood 2020). Exclusionary zoning was ruled unconstitutional 50 years later, in 1917, but the ruling was quickly circumvented by the uprising of racially restrictive covenants that restricted property owners from selling to African Americans (Welsh 2018). Additionally, President Roosevelt’s New Deal included the Home Owners Loan Corporation (HOLC) which subtly laid out how the government would only ensure banks that did not give loans to people of color (Lockwood 2020). Sadly, even though these outright racist policies are not in play today, there still stand systemic obstacles that complicate African American’s pursuit to homeownership and wealth accrual. “The median black wealth in 2016 amounted to $13,460—less than 10 percent of the $142,180 median white wealth” (Hanks et al. 2018).

Construction of Poverty-line Definition:

The very definition of poverty, which undergirds the welfare system, contributes to the stronghold that poverty has over people. The poverty rate, or poverty definition, is a specific dollar amount that is considered to be the minimum amount of resources required to meet the most basic needs of a family unit - below which individuals are considered to be impoverished (Lee 2018). The U.S. definition of poverty is dangerously low which means an inadequate amount of people are getting the government assistance necessary since it is used to determine eligibility for financial benefit programs (Lee 2018).

The U.S. poverty thresholds were created by Mollie Orshansky of the 1963 and 1964 Social Security Administration (Fisher 1992). Orshansky used the 1955 Household Food Consumption Survey, that informed the emergency economy food plan, to determine that families spent about one third of their after-tax income on food (Lee 2018). The resulting welfare algorithm is thus food times three. In 1964, Mollie Orshansky herself reasoned that the poverty threshold would be a conservative underestimate of poverty (Fisher 1992).

The formula has since been shifted to account for inflation but not for increasing costs of living. The current algorithm is still food times three. However, if the 1963 logic were used today the multiplier would be food times seven. Together, this shows one example of how U.S. welfare policy is inadequate in uplifting people who have suffered in poverty for long periods of time, even for generations. What this means is that U.S. policy is compounding accelerated biological aging processes that are mediated by financial stress.

Generational Cycles in Poverty:

People emerging from families who have, for generations, experienced poverty are more likely to experience it themselves. When people first receive welfare before the age of 25 (a time

in life largely determined by one's family of origin) they are also more likely to be long-term welfare recipients (Pavetti 1996). In 1996, 42 percent of long-term recipients were receiving welfare aid before they were 25 years of age (Pavetti 1996). The pattern is that people who grow up, at least partially, in poverty are more likely to experience it when they are on their own and the cycle continues. It is evident then, that the U.S. welfare system does not do enough to help people who have been experiencing poverty for generations.

Biological Clock Method

Epigenetic research has uncovered profound correlations between the rate of biological aging and socioeconomic status of African American women (Simons et al. 2016). However, there is no foreseeable reason why the same correlations should not apply to all races. Methylation, an epigenetic modification, occurs when a methyl group bonds to a cytosine base to make 5-methylcytosine. The methyl group is added by methyl-transferase enzymes. The significance of the methyl group lies in its spatial capacity to block transcription factors and polymerases from transcribing the region they occupy. When maintenance machinery becomes overloaded, errors occur in the maintenance of methylomes which is strongly correlated to biological aging (Mendelsohn and Larrick 2017). The majority of methylation occurs at CpG sites where a cytosine base is adjacent to a guanine base and specifically in promoter regions (Moore et al. 2013; Simons et al. 2016 and Sleutels and Barlow 2002). These specific details of the human genome may seem far removed from the harsh realities of poverty in the outside world however, these CpG sites are stunningly relevant.

The potential for CpC sites to sustain methylation is what makes these seemingly small players into game changers. The attached methyl groups stick out into the DNA groove and

block transcription factors from riding the DNA strand and pulling amino acids together. Thus, protein synthesis is decreased in bodies that sustain areas of hypermethylation. It is important to note here that hypermethylation is not the only way methylomes can change, some sites experience hypomethylation depending on the methylation pattern in perfect homeostasis. Hypomethylation can be disruptive in proper protein synthesis as well, any dysregulation is harmful. For the sake of this argument excessive hypermethylation will be the phenomenon most often referred to for that sake of clarity and simplicity.

In order to measure the effect that high financial stress had on African American Women's bodies Simons et al. used Hannum et al.'s biological clock method which hinges on methylation of CpG sites to determine biological age. In order to produce a "biological clock" Hannum et al. started with 485,577 CpG markers. Using an association test Hannum et al. found 70,387 CpG markers to have significant associations between methylation status and age. On those 70,387 CpG markers they performed a multivariate regression method called Elastic Net to create a predictive model of aging (Hannum et al. 2013). The outcome was a group of 71 markers that were highly predictive of age due to their close proximity to age related genetic regions (this will be further unpacked below). This model is highly accurate with a correlation coefficient of 0.96 between age and predicted age by methylation markers. This means that the biological clock method is highly accurate in predicting one's age. The model developed by Hannum et al. does not only hold its value in adult populations, but in adolescence as well (Simpkin et al. 2017). These 71 CpG markers are the substance of the biological clock that Simons et al. uses to correlate socioeconomic status to methylation status. The methylation status

of each individual site is determined using the Illumina HumanMethylation450 Beadchip. Using that information they simply use the following equation to calculate predicted age.

$$Biological\ Age(i) = \sum_{j=1}^{71} (CpG(ij) \star Weighting(j)).$$

Where biological age is directly proportional to the fraction of CpG sites that are methylated normalized to a coefficient. The difference between biological age by methylation and chronological age is found to be larger in African American women experiencing chronic economic stress than those who are more financially secure (Simons et al. 2016).

Accelerated Biological Aging in Women of Color in Poverty

There are two ways that methylation patterns are thought to change thus affecting aging. First, environmental stress, especially from poverty, can play a major role in disrupting methylation patterns (Murgatroyd et al. 2009). Secondly, changes to a methylome can happen spontaneously by way of chemical agents or errors in copying methylation states in DNA replication (Murgatroyd et al. 2009).

A theory for suspected relationship between age and epigenetics has been developing since the 1960's when it was first investigated in mouse models (Simons et al. 2016). In 2016, Elliott et al. investigated the impact of chronic social defeat, an environmental stressor commonly experienced in situations of poverty, on DNMT3 regulation. It was demonstrated that the increased stress deregulated DNMT3 transcription (responsible for catalyzing methylation) which in turn deregulated global methylation patterns (Elliot et al. 2016). This serves as a proof of concept that stress holds the power to change methylation patterns.

Chronic low socioeconomic stress due to systemic issues facilitates shifting in methylation patterns often causing accelerated biological aging. In people over 20 years of age

there is a constant rate of methylation change to the 71 CpG sites that make up the "biological clock" identified by Hannum and colleagues. Notably nearly all 71 CpG markers are either close to genes or within genes whose functions are associated with conditions related to age such as Alzheimer's disease, cancer, tissue degradation, DNA damage and oxidative stress (Hannum et al. 2013). Changes in methylation patterns nearby or in age related genes can affect gene expression correlated with aging. This means that the more shifting of methylation patterns that deregulate transcription the more chaotic the system becomes thus, putting more stress on mechanisms at the molecular level. This is precisely why disruption to these CpG markers resulting from high economic stress is correlated with increased rates of aging and conditions associated with aging.

In order to measure the biological effect of poverty on methylation changes Simons and colleagues measured the extent to which participants experienced chronically low incomes. Participants then self reported their income on a 16 category scale where 0 was less than $10,000 and 15 was $200,000 or more annually. Their per capita incomes were averaged from 2002-2008. Additionally, they assessed financial hardship on a 4-item scale (Simons et al. 2016). Finally, hierarchical regression models were used to control for health-related behaviors (smoking cigarettes, consuming more than 3 drinks a day, exercise, a healthy diet, BMI) and childhood trauma, education and marital status to esure any resulting correlations were indeed due to income. (Simons et al. 2016).

The results show that biological aging (by methylation) most strongly depends on financial pressure (Simons et al. 2016). This association is maintained even after controlling for education, marital status and childhood trauma (Simons et al. 2016). The only variable that was

marginally significant to accelerated aging was lack of health insurance (Simons et al. 2016). Importantly, Simons and colleagues found that 68% of women in their sample with per capita incomes less than $3,900 exhibited accelerated aging, whereas 70% of those with per capita incomes around $15,000 experienced decelerate aging (Simons et al. 2016). This shows very clearly that African American women with incomes below the poverty rate disproportionately experience accelerated biological aging.

The Reversal of Biological Aging

There is hope in the resiliency of the human body to reverse these epigenetic modifications that accompany poverty. With the relief of enormous economic stress comes space for the built-in DNA repair mechanisms to bring order back to chaotic methylomes. It is known that the addition of a methyl group to a cytosine base (methylation) is a simple organic modification that is very well reversible. Demethylation (reversal of methylation) has been observed in plants, amphibians and mammals which means it does occur under physiological conditions (Chinnusamy and Zhu 2009; Rai et al., 2008; Bewick and Schmitz 2017). The endogenous mutation repair mechanism BER (Base Excision Repair) responsible for repairing lesions in DNA, (Dizdaroglu 2012) is a major player in repairing/reversing methylation modifications (Wu and Zhang 2013). BER functions out of a simple and predictable framework of 1) recognition 2) excision 3) DNA synthesis and 4) ligation (Jan and Hoeijmakers 2001). This pathway is employed in response to DNA missteps commonly known to be created by things like cigarette smoke, other carcinogens and UV radiation (La Maestra et al. 201; Barnes et al. 2018; Rastogi et al. 2010). There is special importance in the recognition step of BER for demethylation in mammals.

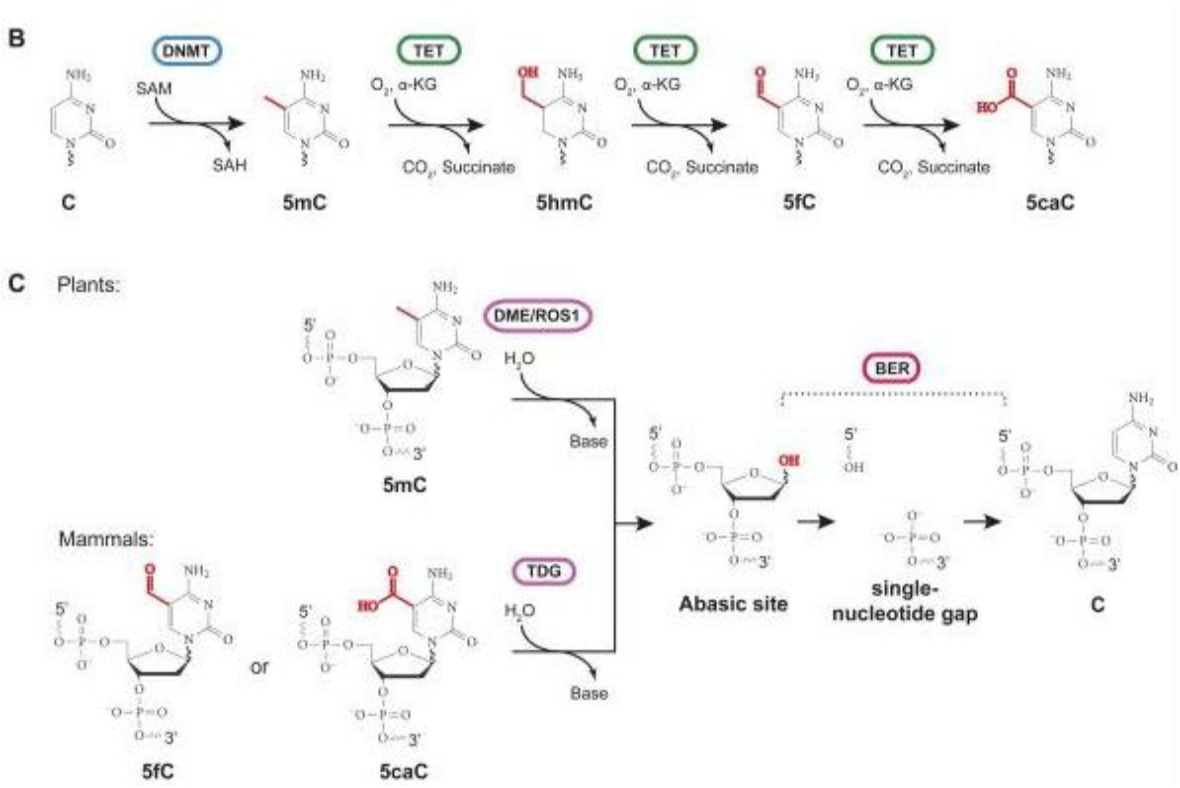

Figure 2. Constitutive mechanism of demethylation (Wu and Zhang 2013)

(*A*) Shows constitutive oxidation of 5-methylcytosine into 5-formylcytosine (5fC), and 5-carboxylcytosine (5caC).

(*B*) Shows entrance of 5fC and/or 5caC into BER by TDG.

To begin the demethylation mechanism in mammals 5-methylcytosine must go through a series of iterative oxidation steps catalyzed by a family of TET proteins (Wu and Zhang 2013). 5-methylcytosine goes through three oxidized states before being demethylated: 5-hydroxymethylcytosine (5hmC), 5-formylcytosine (5fC), and 5-carboxylcytosine (5caC) (Wu and Zhang 2013). In order to oxidize the methyl-group on the cytosine sites TET proteins flip the target methylated cytosine base out of the duplex DNA and into the catalytic site of the protein. The TET active sites contain Fe(II). An intermediate Fe(IV)-oxo reacts with 5mC/5hm/5fC to

generate 5hm/5fC/5caC. This regenerates the Fe(II) in the active site and creates more 5fC/5caC for Base Excision Repair. 5fC and 5caC modifications destabilize the *N*-glycosidic bond to be efficiently excised by DNA glycosylases (Wu and Zhang 2014).

The last three steps of demethylation more generically follow the BER four-step framework. Both 5fC and 5caC are recognized by TDG, a monofunctional DNA glycosylase, to initiate BER (Wu and Zhang 2013). Due to TDG's mono-functionality additional proteins are required to provide the AP lyase activity to remove the 5-carbon ring to make a single-nucleotide gap (Wu and Zhang 2013). This is the second step of BER: excision. The excision proteins will provide an active site that will most likely facilitate the nucleophilic attack of the 2' carbon thus creating an abasic site. The third step in BER, DNA synthesis, is carried out by DNA polymerases to fill in the abasic site with an unmethylated cytosine. Finally, DNA ligase seals the nicks by catalyzing the phosphodiester linkage between the 3' OH group and the 5' phosphate group of the adjacent base on both sides of the new cytosine (Bhagavan 2002). This results in an unmodified cytosine base. Thus, the 5-methylcytosine has been demethylated.

It is important to note that this endogenous repair system is built in. BER is an important constitutive housekeeping process that sustains and keeps the organism. BER is always running at some rate but there are rate limitations. BER can not go infinitely fast: nor can it be upregulated (Balliano and Hayes 2015). BER rates are determined by the enzymes that carry out the reaction. However, when the mechanism is overloaded with mutation rates that exceed BER rates the organism sustains more methylation modifications that have harmful effects such as cancer, increased aging rates, psychological disorders. Therefore, in order for the repair mechanisms to catch up, the cell needs to stop being assaulted. Once the trauma of poverty is

removed, normal endogenous repair mechanisms shift the balance back to demethylation to restore the genome or, in this case, the methylome. In the case of poverty this means the person needs to be relieved of the extreme environment of stress. Based on the biological mechanism eradication of poverty is the best way to protect methylomes. It cannot be expected that demethylation mechanisms will overcompensate for any group of people. Demethylation mechanisms do not eventually get, “used to" higher workloads. There is a long standing bias out there asserting that black women are biologically tougher or more resilient than other races. This is a biological refute to that bias.

Discussion

Research in biology should not be divorced from real people or real actions and implications. Neither can human biological research nor political decisions be divorced from the true human beings they affect. New research lends wisdom regarding how the body can be harmed due to stress giving insight to ways of caring for each other's bodies. This kind of research should not find permanent residence in academic circles but should be gifted to and engaged with by people and communities that could find healing and power from it. In conjunction, political decisions should take seriously the input offered by those who the decisions will directly affect. In the same way, when science pinpoints an agent that is harming people and policy has an avenue of alleviating the harm, action should be taken for the lives and wellbeing of fellow people depend on this action.

The motivation behind this research is to illuminate poverty as a public health crisis that should be treated with urgency and care by top performing teams. A secondary motivation is to spark conversations and imaginations to engage with how this public health crisis can be

addressed and averted. Research on epigenetics alone will never be enough to dismantle the poverty industry, or any other oppressive social structure. For tangible mobilization towards change, scientific research on the ill impacts of some social, political and economic systems, above all else must exist in partnership. It is a necessary next step, after acknowledgement of the need for change, to creatively imagine how and what new partnerships could usher healing in social structures and reconciliation across societal divides.

There is no proven procedure for how such partnerships with researchers need to look or who should constitute them. However, it is non-negotiable that partnership and consultation with those who have or are experiencing poverty must be top priority. Regardless of how well-intentioned a project is, teaming with those who directly experience the effects of a project holds efforts accountable to procedures that realistically parallel positive intentions. This protects people experiencing poverty from being exploited, misrepresented and from agreement with policy that has unforeseen negative consequences. Not only does partnership and consultation with people living in poverty protect from further harm but it would also dramatically increase the asset list of any team.

Imagine the ferocity that could emerge between expert survivalists and expert scientists collaborating to catalyze movement towards eradication of poverty. People experiencing poverty, including the disproportionately affected group of Black women, are not “lay down and die quietly” victims (Dinsmore 2007). Some of the most powerful and beautiful movements towards equality for all have been fronted by the people who are most marginalized. Powerfully, the world-wide movement to end violence against Black communities - Black Lives Matter - was radically co-founded by three black women Alicia Garza, Patrisse Cullors, and Opal Tometi

(HerStory 2013). Additionally, through personal narrative Julia K. Dinsmore graciously highlights some of the most robust skills that bloom from communities facing poverty: hospitality and pooling of talents and resources for the good of the community, coming out of places of poverty lends aspects of her story (Dinsmore 2007). Dinsmore shows how her community of welfare moms and dads forged a diverse network that upheld their survival. "Sharing was the unspoken bylaw, and practicing generosity, the dues for belonging to our unorganized, highly functioning patchwork quilt of mutual assistance" (Dinsmore 2007). Dinsmore shows the powerful creativity for problem solving that lives in spaces of economic unfairness. It is clear from these lived stories that people who are experiencing poverty know what they need and are capable of strategizing the most efficient ways to help themselves and each other. The most fundamental, essential and radical step to relieving the damaging stress from the epigenomes of those in poverty, is to listen and trust them when they say what they need. They know.

People experiencing poverty are not the problem with the system, rather they are the solution to a harmful system (Dinsmore 2007). Instead of trying to fix the people living in poverty, or using them as only research subjects, scientists should work to create spaces where skills from multiple experience sets can pool. Sciences bring to the table a specialized understanding and view of the natural world, skills in strategizing projects, and hold special standing as respected voices in the U.S. Each perspective brings distinct and vital sets of assets and skills. Scientists alongside any other desiring groups could seek out groups working for housing justice and reform and offer support they need. To ensure that these partnerships would steer clear of White-Saviorism the people who are typically ascribed less power need to hold the

majority of leadership roles to balance unequal power dynamics. Additionally, scientists are going to have to be willing to learn a new way, listen and believe in different ways than the scientific enterprise typically engages. In order for scientists to be good allies and partners they are going to have to be willing to do their own healing work to guard against any further exploitation of those experiencing poverty.

Research can be leveraged to tell a story, especially to people in positions of power such as political positions. The intersection between the experience of poverty and the biology of epigenetic regulations is a profound relationship that tells a tragic story in the most universal language that unites all living things: genetic code.

Due to the inheritability of epigenetic modification, liberation of one generation from the burden of epigenetic modifications by way of poverty, consequently liberates future generations that follow. The treatment plan for the inheritable epigenetic modifications is eradication of the environment that is causing them. Research can be leveraged to tell a story, especially to people in positions of power such as political positions. The intersection between the experience of poverty and the biology of epigenetic regulations is a profound relationship that tells a tragic story in the most universal language that unites all living things: genetic code.

Mobilization to fight for economic liberation is a direct predecessor to generations of genetic liberation. Here lies an unprecedented opportunity for scientists to live into their admirable code of ethics to disperse the narrative of epigenetic harm that poverty imprints on people and generations and to continue to fill out the narrative with hard evidence that could be used to lobby people in power for structural changes for liberation.

Further Research

Although this research does lay out one story of how poverty in a capitalist America negatively affects the bodies and of those experiencing it, there is still more work to be done. In order to make this research worthwhile action needs to be taken to create environments that facilitate reversal of harmful demethylation. In order to understand what that looks like, and how possible that really is, a more robust understanding of the initiation of reversal is necessary. To further fill out this research, it would be wise to do the same study on a white population to reiterate the results. Additionally, a study specifically on children would be beneficial. Endeavors to reform housing and economic policy are still necessary for the fruition of the goals presented in this paper. Additionally, a continuing exploration of the possible partnerships that could be brought together is fundamental to the dreams of this paper being reached.

Conclusion

I find it necessary to close by acknowledging that although the amount of academic work and effort put into these kinds of academic studies is admirable and gives those without experience in low-income environments some insight, it really is not new information. The real people living in the most economically dry places already know this. Those who have lived through these experiences can tell you how stressful and how hard it is on their bodies. They don’t need statistics and research to understand that surviving in poverty hurts their bodies and the bodies of their children. It is with this sentiment in mind that I headed cautiously into this research project to not use people living in poverty as subjects for my own intellectual entertainment and to portray them solely as victims. I hope I did as I intended. If this research is

only worth one thing, let it open minds to facilitate loving action to uplift our people from the clutches of poverty.

I will close with an expert from Julia K. Dinsmore's book, *My Name is Child of God… Not "Those People:" A first person look at poverty*. Dinsmore shares insight, from her own personal experience, into a collective mental framework that could help heal the tragic divisions, systems and ideologies that persist in America.

> "It's not useful for any of us to perpetuate the blame game. Doing so only causes more wounds to those who already bear the burdens of social and economic unfairness. I think it is useful, however, for us as a society to take responsibility for the harm done to people who have historically suffered or currently suffer under lack. We should address generational hurts that began in slavery and the colonization of North America. We need to acknowledge the fact that the labor of many, many people was ripped off to benefit a few.
>
> If we must place blame, why not start with looking at systems, public policy, and structures that control the flow of wealth and resources in our nation and world? If there is any use for shame, then let it move all of us whose basic needs are met toward truth, reconciliation, and rectifying resources to a new bottom line...the bottom lie spelled H-U-M-A-N-I-T-Y!
>
> Let shame - if it is possible - mobilize our collective desire to do better by ourselves by creating a land where every person can eat and live in a safe community" (Dinsmore 2007).

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