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Abstract

It is widely known that trauma is repeated throughout a victim's life, but the biological mechanisms of its recurrence (revictimization), even though understood biologically, are not accepted or discussed in all disciplines. A combination of socio-cultural and biological perspectives is needed to understand this cycle of revictimization and to offer help for sufferers and public health agencies. In order to better understand these issues, I conducted a synthesis of existing scientific research regarding the discrepancies between biological and sociological studies on revictimization. Within my review of sociological research it was revealed that initial trauma and revictimization are clearly understood as a positive feedback loop, with one increasing the other over a victim's life. In biology, however, this loop has been acknowledged but the study of the recurrence of trauma has not been integrated into these disciplines. In humans, biology and sociology are inseparable; a recognition in these disciplines that a positive feedback loop exists regarding revictimization is key. Recognizing the existence of this biological feedback loop has the potential to mitigate the damage of past, present, and future trauma. With a better understanding of the biological aspects of their recurring trauma, devastated sufferers can be empowered against damaging ideologies, such as biological determinism and victim blaming.

Keywords: revictimization, trauma, positive feedback loop

Introduction

The experience of a traumatic event is all encompassing. It forever alters an individual's genes, how those genes are expressed, and the synthesis of proteins and other molecules. Those alterations affect how organ systems, such as the nervous and cardiovascular, operate. The function of organ systems affects the body as a whole. The body affects the mental and emotional states of an individual. Mental and emotional functioning changes how societies and cultures perceive said individuals. Trauma affects everything that a human being is and everyone surrounding them. No person is untouched by trauma.

It is widely known that trauma is repeated throughout a victim's life, but the biological mechanisms of its recurrence, even though understood biologically, are not accepted or discussed in all disciplines. Within my review of existing biological research on trauma, there was severe compartmentalization, separating the work of different disciplines from the biological sciences, in addition to the removal of lived human experiences. The majority of the studies I reviewed couched their language toward trauma in blanket terms such as "stress". This term mutes survivors of trauma and equates their "stressful" experiences with the same "stress" that a student might endure during finals week. The depths of these studies are invaluable

to trauma research as a whole; however, the language used to describe trauma (or stress) is at best problematic and at worst an open door to biological determinism, scientific victim blaming, and an overall reductionist view of the lived human experience. Sociological studies of trauma do not fare much better on their own, either.

Within my review of sociological research it was revealed that initial trauma and revictimization are clearly understood as a positive feedback loop, with one increasing the other over a victim's life. Unfortunately, this was where most articles ended their studies of trauma and began to decry the biological sciences for not integrating the "material body" with the body embedded in time, space, and experience, as described by Margaret Lock in her synthesis of recent epigenetic research (Lock, 2015). There was also the occurrence of sociological studies that reference biological sciences (specifically genetics and epigenetics) without a nuts-and-bolts understanding of the topic. This does not aid the outcry for future cooperative, interdisciplinary research between the sociological and biological sciences. It has become apparent that more must be done to bridge the divide.

A combination of socio-cultural and biological perspectives is needed to understand this cycle of revictimization and to offer help for sufferers and public health agencies. In order to accomplish this goal, sociological sciences must become more genetically and epigenetically literate. Future research into trauma and revictimization should include fully integrated, holistic perspectives so that the experiences of survivors are recognized by all disciplines.

In humans, biology and sociology are inseparable, a foundational tenant of anthropology that is not acknowledged by various biological fields of study. A recognition in the biological sciences that a positive feedback loop exists regarding revictimization, is key towards addressing the complexities of the issue of trauma. Conversely, recognizing and integrating the existence of the biological facet of the feedback loop has the potential to mitigate the damage of past, present, and future trauma. With a better understanding of the multifaceted aspects of their recurring trauma, devastated sufferers can be empowered against damaging ideologies, such as biological determinism and victim blaming.

Methodology

For this project, I conducted a review of existing research, focused on salient findings between the years 2000-2019 regarding the discrepancies between biological and sociological studies on revictimization. In my analysis of this research, I attempted to integrate findings from studies within these disciplines. The goal was to unify some of the research of these fields into a holistic, biocultural trauma feedback loop that stretches from the genetic to the sociological causes of revictimization (the reoccurance of trauma). In addition to outlining an integrated feedback loop, I created an extremely tentative example of the feedback loop in action. One gene was isolated and followed from its epigenetic modifications, molecular alterations, organ system changes, psychosocial and emotional impacts, all the way to sociocultural responses.

Sociological Feedback Loop

There are a multitude of avenues in which trauma can be studied. This section will focus primarily on childhood trauma and mental health issues as the path toward revictimization, keeping intersectionality, resilience, and the lived human experience in mind.

It is no secret that the experience of trauma causes damage to the survivors. When those survivors are children, with yet fully developed brains, the damage can be permanent. In a review of neurobiology research on childhood trauma utilizing the Adverse Childhood Experience Study as a case example, Anda et. al found that: "...early life stress such as abuse and related adverse experiences cause enduring brain dysfunction that, in turn, affects health and quality of life throughout the lifespan" (Anda et. al, pg. 175). The study further explains that their findings support "the hypothesis of dysfunction", which states that early childhood trauma alters the functioning of particular regions

of the brain that are currently believed to be involved in anxiety and mood regulation. In another study, which conducted a statistical and systematic analysis of childhood trauma (or maltreatment) types and negative mental health outcomes, it was found that all the types of maltreatment under question had a statistically significant relationship with poor mental health outcomes (Cecil et. al, pg. 114). Childhood trauma, maltreatment, or adverse events all appear to have similar effects on the developing brain: dysfunction occurs, mental health issues ensue, and the quality of a child's life decreases. To further exacerbate the situation, children develop negative coping skills in response to these experiences.

In the large-scale, nationally-representative, epidemiological analysis of the United States conducted by Vaughn-Coaxum et. al, it was found that: "... exposure to nearly all forms of trauma was… strongly associated with increased negative emotion-focused coping" (Vaughn-Coaxum et. al, pg. 842). The use of the coping styles that were under observation, only increased as an individual experienced a greater number of traumatic events. Furthermore, their results suggested that forms of coping linked to poor mental health were related to the amount of traumatic events an individual had prior to participation in the study (Vaughn-Coaxum et. al, pg. 850). The World Health Organization, in a study on violence and mental health that utilized more than 160 experts from over 70 countries, also found that traumatic experiences with violence have a cumulative effect on the mental health of victims. The mental health of victims is greatly impacted by the severity, type, and number of experiences with violence that occur throughout their lifetimes. The greater the severity, type, and number of occurrences, the more negative mental health outcomes can ensue (Mercy et. al, pg. 22). The World Health Organization, noticed that poor coping behaviors, such as risk taking, were likely caused by childhood exposure to trauma or maltreatment (Mercy et. al, pg. 23). There is no singular cause that makes individuals more likely to become a victim of violence; however, there is a relationship between childhood mental health disorders, trauma, and negative life trajectories.

In a fifteen-year-long study of 3,804 schoolchildren in Hong Kong, conducted by Raine et. al, that was concerned with peer victimization and children with schizophrenia or schizotypal symptoms, it was noted that adult individuals with the disorder are: "...14 times more likely to be the victims of a violent crime" (Raine et. al, pg. 938). This is in addition to their discovery that the symptomatology of schizophrenia in school-aged youth causes other children to treat them poorly (Raine et. al, pg. 937). Schizophrenia and its spectrum are all found within a larger grouping of mental health disorders, specifically the "trauma spectrum". This spectrum includes: anxiety and panic disorders, depression, schizophrenia and other dissociative disorders. As previously discussed, the Anda et. al study goes into detail describing the comorbidity of these disorders. Hence the term "trauma spectrum disorders", which describes how they all occur together in different combinations and levels of severity (Anda et. al, pg.182). Mental health disorders, according to the World Health Organization study, are "both causes and consequences of interpersonal, collective, and self-directed violence" (Mercy et. al, pg. 20).

Copeland et. al found in their review of the Great Smoky Mountain Study, in which 1,420 children were interviewed through adolescence and young adulthood about their experiences with bullying, that this childhood event is much more serious than previously believed. To experience bullying in childhood is known to have negative impacts on development; however, recent research suggests that bullying can be as severe as maltreatment from family members (Copeland et. al, pg. 7573). A potential basis for bullying and other hierarchical behavior among children was also discovered through this study. Copeland et. al noticed that children who were purely bullies had significantly less low grade inflammation throughout their lives, while pure victims and bully-victims had the most low grade inflammation (compared to children who were not involved in bullying at all). Long term low grade inflammation is associated with high levels of depressive disorders and other health risks (Copeland et. al, pg. 7570). Victimizing others gives an individual biological ad-

vantages (Copeland et. al, pg. 7573). Populations that are already vulnerable are the most at risk, such as: minorities, refugees, those with disabilities, the elderly, women, the LGBTQA+ community, and many others (Mercy et. al, pg. 28).

The World Health Organization study, conducted by Mercy et. al, outlined a multiplicity of risk factors for experiencing violence on the individual level. "Individual-level risks identified include demographic factors such as age, sex, and race/ethnicity; psychological and personanlity disorders, alcohol and substance abuse, and a history of engaging in violent behavior or experiencing abuse" (Mercy et. al, pg. 28). Taken together, it becomes apparent that disenfranchised communities are hit the hardest and held the longest by the feedback loop of trauma. Ethnicity and socioeconomic status, in this particular example and social climate, cause a cascade of psychosocial and mental-emotional issues that are compounded by victimization and structural violence. This experience has been named "racial battle fatigue". In the chapter titled, "The Epigenetics of Being Black and Feeling Blue", in which a synthesis of preexsisting research is utilized, racial battle fatigue is described as: "...a state of keeping the bodies of stigmatized minorities hypervigilant in anticipation of the next white racial insult. Racial battle fatigue affects human biology and physiology at the cellular level, leaving the bodies of the poor, the impoverished, and the targeted more vulnerable to mental and physical health decline" (Smith, pg. 261). Smith goes on to explain how disenfranchised communities also struggle with lifestyle related disease. In order to cope with hypervigilance and the sensitivity that follows, risky lifestyles are often adopted by disenfranchised populations to relieve some of their burdens (Smith, pg. 262). Keeping in mind the Copeland et. al study on childhood bullying, there is a biological boost for those who wish to keep bullying minorities and other disenfranchised communities. There is, however, one defense that these communities have: resilience.

The chapter titled "Impact of Trauma: Vulnerability and Resilience", which relied on professional experience and a review of scientific literature, examined the recovery of victims of genocidal trauma using a perspective that recognizes the coexistence of strength and vulnerability inside each survivor (Giberovitch and Barry, pg. 68). Recovering from trauma is a difficult and ill-defined task. It never looks quite the same for any individual. The chapter outlined a few of the tasks that are necessary for recovery to begin. "Healing can be difficult because it requires accomplishing psychic tasks such as creating a sense of rootedness, belonging, and continuity with the past; working through losses, guilt, rage, and shame; and meaningfully integrating the experience into the totality of life" (Giberovitch and Barry, pg. 69). They go on to explain that the magnitude of the Holocaust may not be able to be fully integrated into an individual's life, that it will take more than a single generation or lifetime. The same can most certainly be said of the historic trauma of slavery. The key to assisting survivors toward whatever kind of recovery they need is empathy. One author observed that the types of therapy they utilized often mattered less than listening empathetically to survivors and creating honest, supportive relationships with them (Giberovitch and Barry, pg. 83). The chapter goes on to explain that survivors also fare better when they see actual biological proof that validates their experiences. When discussing experiences, such as intrusive memories, with their clients, one author noted the empowerment that survivors feel when their personal experiences are validated by research, as well as the comfort of knowing that other survivors are having similar experiences (Giberovitch and Barry, pg. 83). This validation can also help the families of survivors to be more sensitive and empathetic towards the symptomatology of trauma. Strong, caring, and empathetic relationships are the keys to recovery and are the foundations on which resilience, even in early childhood, is developed. In his article on early childhood development, Jack Shonkoff, from the Center on the Developing Child at Harvard University, discussed the source from which children develop their ability to cope with hardships through the synthesizing of preexisting research. It is through supportive relationships and chances to develop positive coping mechanisms, that children

acquire the ability to face adversity (Shonkoff, pg. 12). From childhood traumas to historical trauma, survivors need to be treated with empathy, on an individual basis, be involved in close, caring relationships, and have their experiences validated by science. This integrative view of survivor resilience requires a biocultural approach; however, trauma and revictimization are not solely the ailments of individuals, but of whole societies. To rephrase a quote from James Baldwin: Trauma is stuck in us, and we are stuck in trauma.

Epigenetics: Between Biology and Sociology

In this section, the primary focus will be on the epigenetic linkage of trauma and revictimization, including their sociological and biological aspects. This will be in addition to outlining how epigenetic modifications from trauma generally occur. Due to the nature and current capability of epigenetic research, a portion of cited research in this section can only theorize how their non-human based findings can potentially be applied to future human studies.

Genes are not static and unchanging. They wait for signals from the external world, such as the environment, food resources, and the social and physical location of our homes. Genes also receive signals from the sociological aspects of our lives, like socio-economic status and disenfranchisement (Smith, pg. 261). The experience of trauma leaves markers on the genes of victims, modifying how DNA is transcribed and replicated. This occurs through various processes such as histone modification, DNA methylation, mitochondrial modification, and many other avenues. A study in which mitochondrial functioning was selectively dysregulated in mice and then systematically characterized, it was found that mitochondrial defects accounted for 81 percent of the observed, hippocampal gene expression changes that were associated with alterations to the brain's stress response system (Picard et. al, pg. E6619). Their study sought to uncover if there was a relationship between defects in the stress response systems of the body and dysregulation of mitochondrial gene expression. Therefore, their research operated under the notion that: "...by supplying the majority of cellular energy, mitochondria contribute to the organism's overall adaptive capacity" (Picard et. al, pg. E6621). In another article that examined genome wide association studies in order to elucidate new genes involved in the development of PTSD, the role of mitochondria was also explored. Recent research, focused on the mitochondria, found that shifts in the membrane potential of the mitochondrian may be a potential cause of the reduction in hippocampal volume that is characteristic of PTSD sufferers (Feodorova and Sarafian, pg. 9). In other words, the mitochondria affected genetic and non-genetic signalling to glucocorticoid receptors (GR) within the hippocampus, which in turn affect various transcription factors that control the expression of thousands of genes (Feodorova and Sarafian, pg. 6). Taken together, the sociological causes of trauma cascade into the epigenetic precursors of revictimization. To again cite the World Health Organization study, the experience of violence, and its effects on one's mental health, are cumulative, with the incidence of one increasing the incidence of the other (Mercy et. al, pg. 22). It can then be suggested that, as an individual experiences more trauma, more epigenetic markers can be left on their genome, which causes more changes in gene expression to accumulate.

One mechanism in which epigenetic modifications are accumulated is through histone modification. Histone modifications are much more plastic and less stable than DNA methylation. They can be placed on a particular region of the genome, taken off, and placed back (Carey, pg. 72), as discussed by Nessa Cary in her book *The Epigenetics Revolution*. She is a professor at Imperial College in London and has specialized in epigenetics for eighteen years. This plasticity allows cells to experiment with various gene expression patterns. "If there is an advantage to the cell in those genes being switched off, the histone modifications may last long enough to lead to DNA methylation" (Carey, pg. 72). Once DNA becomes methylated, it tends to stay that way, making the modified gene expression heritable (Carey, pg. 72). There is, however, variation in how this expres-

sion plays out. This is due in part to the proteins that "read" histone modifications. They bring out other proteins, which combine with the "readers" into complex structures that can turn certain gene expressions on and off (Carey, pg.73). Depending on individual protein availability, some of the epigenetic markers of trauma can be either turned on or off. As Catherine Malabou discussed in her synthesis of existing research, the experience of trauma is different for everyone; some individuals feel their trauma more acutely than others, some trauma is inherited rather than experienced first hand, while others appear unaltered by it or do not remember (Malabou, pg. 187). Dr. Howe, a professor at Humboldt State University and the chair of the Psychology department, discussed the multifaceted nature of where these variations, in response to trauma, arise. The HPA axis, which regulates stress response, functions differently between individuals. This results in various reactions to trauma based on individual experiences, brain chemistry, genotypes, coping skills, all within the specific zeitgeist in which we live (Howe, pg. 472).

All of these factors are in addition to systems of power, privilege, and oppression, in which disenfranchised individuals and communities are left more vulnerable to negative environmental and genetic outcomes. Joseph Graves stated in his synthesis of preexisting research that: "Epigenetic programming from past experience (or inheritance) can enhance vulnerability to stress and place the brain at higher risk of neurodegenerative events. Stress, defined as a perturbation of homeostasis, can cause the onset of complex neural and endocrine responses characterized by activation of the HPA axis" (Graves, pg. 39). Activation of the HPA axis is directly associated with stress; dysregulation of the HPA axis is associated with trauma and trauma is a neurodegenerative event. The HPA axis influences the hippocampus and the amygdala, which are affected by trauma due to their regulation of memory and fear (Giberovitch and Barry, pg. 80). One particular gene, the ADRA2b gene, is associated with the dysregulation of brain regions associated with the HPA axis. When an individual carries a particular deletion mutation of the ADRA2b gene, said individual is significantly more reactive to emotional stimuli (Todd et. al, pg. 6514). The occurrence of trauma causes the hippocampus to shrink, due to hyperactivity in the amygdala. In a study that utilized magnetic resonance imaging on neurosurgical patients with ventromedial prefrontal cortex damage, it was found that hyperactivity in the amygdala is characterized by unregulated reactivity to negative, emotional stimuli (Motzkin et. al, pg. 276). A traumatic experience, therefore, is a prerequisite for future victimization and/or PTSD; however, not all individuals who experience trauma are genetically predisposed to these outcomes. Research on twins suggests that approximately 30 percent of the variation in PTSD symptomology is due to genetic variation (Feodorova and Sarafian, pg. 9). All of the gene expression changes from trauma are heritable; they are passed on through the generations regardless of whether or not they become activated (or deactivated) during any particular individual's lifetime (Carey, pg.73). This is emblematic of historical trauma like that of Holocaust survivors and African American communities, as well as intergenerational traumas of violence and incest. The legacy of trauma is remembered by the genes and the body, regardless of the passage of time. This causes the bodies of the disenfranchised to remain hypervigilant. It is the larger society, and it's views on trauma and victims, that interacts with susceptible genomes. As Darron Smith explained: "This dynamic interaction between our genes and environment should serve as a reminder that human beings thrive in the absence of war, famine, and other manufactured discord" (Smith pg. 262).

Biological Research

This section will discuss the biological mechanisms of trauma and revictimization. A multiplicity of research from various disciplines within the biological sciences will be utilized.

A promising new topic of inquiry, into the stress response systems of the body, is the role of mitochondria and its DNA. The conclusions of the Picard et. al study coincided with these recent revelations. "This result is consistent with recent

findings that mitochondrial dysfunction can exert robust and bidirectional regulation across the majority (~70%) of genes within the human genome" (Picard et. al, pg. E6622). In another study conducted by Picard et. al, mitochondrial functions were selectively manipulated in order to observe potential changes to stress response systems. Genetic and epigenetic modifications are all induced through the activation of metabolic pathways that are localized in the mitochondria. "Thus, both the addition and removal of epigenetic marks are metabolically—or mitochondrially—regulated" (Picard et. al, 73). The mutations and defects of the mitochondria and its DNA, due to its energetic and regulatory functions, can affect the transcription and replication of nuclear DNA. These defects are much more common in mitochondrial DNA than in nuclear DNA. There are approximately two to fifteen copies of mitochondrial DNA within a single mitochondrian that replicate once each month. This creates a higher mutation rate in mtDNA than in nuclear DNA, in addition to mitochondrial cell division which occurs independently of mitosis, as discussed in the Sas et. al review of existing scientific literature (Sas et. al, pg. 3). Thus, the vulnerability of the mitochondrial DNA (mtDNA) becomes the vulnerability of the nuclear DNA (nDNA). It has been theorized that oxidative stress is the culprit. This is due to the fact that mitochondria create the majority of reactive oxygen species. Due to the derived nature of mitochondria, each one still contains circular DNA, a hallmark of their bacterial origins. This is their individual, mitochondrial DNA in which key components of the oxidative phosphorylation process are contained, which Pinti et. al explained in their review of current cellular biology research (Pinti et. al, pg. 1). These studies point to the evolutionary history of the stress response system within multicellular life and the beneficial relationship between eukaryotes and mitochondrial ancestors.

Epigenetic modifications to gene expression, due to stress or trauma, occur in a multiplicity of regions within the body. Specifically, direct epigenetic changes occur in the expression of genes associated with the HPA axis, a particular region of the brain that regulates stress, fear, and emotion; it is the hub of the brain's stress response system. Some of the defects in the brain's stress response systems that are specifically associated with trauma are: a shrinkage in the hippocampus, an overactive amygdala, and cortisol levels becoming dysregulated. These factors were noted in the Feodorova and Sarafian study. "Decreased activity and neuronal atrophy in the hippocampus and in the PFC, as well as increased activity and neuronal growth in the amygdala have been confirmed" (Feodorova and Sarafian, pg. 6). They also noted the relationship between changes to the hippocampus and cortisol. The vulnerability of the hippocampus to stress is due to its high number of corticosteriod receptors (Feodorova and Sarafian, pg. 6). These receptors are responsible for the secretion and the reuptake of these cortisol-based hormones. Too much secretion of cortisol can lead to immunosuppression, while too little reuptake can lead to autoimmune disorders; both outcomes lead to poor mental health. In a study examining the outcome of forced swim (FS) in mice, which later extrapolates to future human research, it was found and hypothesized that: dysregulation of glucocorticoid secretion, due to trauma and/or chronic stress, increases an individual's vulnerability to mental health disorders (Mifsud and Reul, pg. 11336). The study goes on to explain that: "Glucocorticoids act via mineralocorticoid receptors (MRs) and glucocorticoid receptors (GRs) in the hippocampus, resulting in altered transcription of target genes" (Mifsud and Reul, pg. 11336). With defects in the transcription of its genes, the hippocampus cannot go through neurogenesis properly, hence the neuronal atrophy. This has the potential to explain the difficulties with memory that are associated with traumatic events, since the hippocampus plays a vital role in the formation of long term memories.

The amygdala, on the other hand, is associated with the processing of memories, as well as emotional responses to stressful stimuli. When working properly, the amygdala assists in limiting responses to stress. After trauma, the amygdala becomes dysregulated and intense, emotional reactions to stimuli ensue. This is due to the previously discussed gene, ADRA2b. A study,

in which fMRIs were taken in order to conduct neural pathway analyses, found that "...emotionally salient stimuli are subjectively experienced with greater perceptual vividness, a phenomenon we call emotionally enhanced vividness (EEV). EEV has been linked to greater activation of object-sensitive regions of the visual cortex, an effect mediated by amygdala activity" (Todd et. al, pg. 6506). The effects of a smaller hippocampus and an overactive amygdala can also cause an increase in the likelihood of developing poor coping behaviors, due to the shrinkage of the hippocampus and the anterior cingulate cortex (ACC), which regulates rational decision making. The regulation of rational decision making by the ACC and hippocampus, is itself regulated by hormone secretion, namely cortisol. When cortisol levels are dysregulated, inflammation occurs due to cortisol's (usually) anti-inflammatory properties. Increased and prolonged inflammation is associated with the development of mental health disorders (Copeland et. al, pg. 7570). HPA axis functioning and cortisol regulation reported in victims of bullying, were consistent with the researchers' data illustrating increases in CRP (C-reactive protein, which is a marker of low-grade systemic inflammation) levels of said victims (Copeland et. al, pg. 7572). The hormonal, neurological, and inflammatory responses to stress exist in a positive feedback loop for those susceptible. Susceptibility and resilience are two sides of the same proverbial coin.

In one study, titled "Blood-Brain Biomarkers for Stress Susceptibility", Chattarji and Rao reviewed human and non-human neurobiology research and discovered several genes that may play into individual variation of stress susceptibility. "Among these nine factors, the glucocorticoid receptor (NR3C1) is of particular interest as the development of PTSD-like symptoms can be blocked by the administration of glucocorticoids shortly before or after stress" (Chattarji and Rao, pg. 13254). Their study also found that the particular genes under question were associated with both the amygdala and the hippocampus. These regions of the brain, and their dysregulation, are clearly understood to be involved in the development of PTSD (Chattarji and Rao, pg. 13254). Susceptibility to the negative effects of trauma stem from genetic variations downstream and the dysregulation of specific brain regions upstream.

This susceptibility is due to a bias against long-term neurological needs, in favor of short term protection. Recent research has shown that an evolutionarily salient mechanism favors habitual, stress reduction behaviors over executive functioning during heightened stress in order to promote short term survival (Sinha et. al, pg. 8837). This may explain why the hippocampus and the amygdala become dysregulated, due to their association with the prefrontal cortex, which controls executive functioning.

In their study on resilient coping, utilizing an exposure paradigm and fMRI, Sinha et. al discovered that "...greater neural flexibility signals in the ventromedial prefrontal cortex during stress correlated with active coping ratings whereas lower dynamic activity in the VmPFC also predicted a higher level of maladaptive coping behaviors in real life" (Sinha et. al, pg. 8837). Due to trauma's effect on the prefrontal cortex, decision making and stress-response regulation are much more difficult for those affected by a multitude of traumatic events over their lifetimes, hence the maladaptive coping behaviors. This is in addition to the increase in vulnerability to mental health disorders (Vaughn-Coaxum et. al, pg. 843). Long term, low-grade inflammation, a marker of chronic stress, is another factor that increases one's vulnerability to mental health disorders, such as PTSD (Copeland et. al, pg. 7570).

In another study on resilience, in which mice were repeatedly inoculated with M. vaccae against stress related inflammation, a bacterial connection to the stress response system was uncovered. A reduction in T cells, increased IBD, elevated proinflamatory responses, and autoimmunity issues are all positively correlated to PTSD (Reber et. al, pg. E3130). The close relationship between the brain, stress, and the bowels of humans is well understood, as discussed by Moloney et. al in their review of current research on the microbiota-gut-brain axis (Moloney et. al, pg. 104); however, this particular study focused on animal models and bacterial treatments for gastrointestinal sources of stress.

Reber et. al, found that "...immunization with M. vaccae induced a long lasting shift toward a more proactive coping response, characterized by decreased submissive, flight, and avoiding behaviors, during chronic psychosocial stress that, based on previous studies in rodents and humans, may decrease vulnerability to the development of more persistent anxiety- and depressive-like symptoms" (Reber et. al, pg. E3131). There is undoubtedly a connection between the biome in the gut and the functioning of the mind. When synthesized, these studies suggest that genetic differences in the neural structuring of particular regions of the brain, act together with the microbiota of the gastrointestinal tract to cause susceptibility (or resilience) to stress.

Tentative Findings of a Biocultural Feedback Loop

In this section, all the previously discussed studies will be synthesized into a tentative biocultural trauma feedback loop. In particular, the relationships between trauma and revictimization will be elucidated through: childhood trauma, interpersonal violence, and mental health disorders.

At the genetic level, a deletion mutation occurs on the ADRA2b gene. This mutation results in carriers experiencing emotional stimuli in a more intense manner than non-carriers. Carriers of this deletion have more vivid and emotional perceptions of the world due to increased activity in regions of the brain associated with evaluation. Differences in serotonin and norepnephrine availability, as well as personal experiences, dictate what our brains view as emotionally valuable input (Todd et. al, pg. 6515).

On the epigenetic level, this modification could have occurred through inheritance or through a mutation in mitochondrial DNA, which lead to improper nuclear DNA replication or transcription. It could possibly be an inherited mitochondrial or nuclear DNA mutation as well. In order for any particular mutation or modification to become inheritable, the modified gene must be located in an individual's gamete cells (Carey, pg. 55). Also, it could have occurred through either DNA methylation or acetylation, or through a histone modification that later became methylated (Carey, pg. 73). The avenues are essentially endless as to how this particular deletion mutation occurred. The Todd et. al study did not specify the source of this mutation and so speculation is all that can be done here; until, of course, more research on the ADRA2b deletion mutation is conducted.

In the molecular level, mitochondria play a leading role. This is due to the fact that these organelles are the central hub of cortisol production (Picard et. al, pg. 76). Chronic stress and trauma cause cortisol to become dysregulated. When this occurs, a cascade of chemical reactions occur in the stress response systems of the brain, particularly in the hippocampus and its associated genes (Misfud and Reul, pg. 11337). The mitochondria are also responsible for the atrophy of the hippocampus in PTSD patients. This is due to genetic and non-genetic signalling of glucocorticoid receptors, in which modifications are made to the mitochondrial membrane (Feodorova and Sarafian, pg. 9). Cortisol is created by, and also modifies, the mitochondria, in ways that trickle down to the level of organs and organ systems.

Within the organ system level, modification to the ADRA2b gene is correlated to higher activity in the ventromedial prefrontal cortex, which in turn causes hyperactivity in the amygdala, and a reduction in the size of the hippocampus (Todd et. al, pg. 6514).

These same effects can be seen in these regions of the brain after the occurrence of trauma and are necessary for PTSD and other trauma spectrum disorders to occur (Anda et. al, pg. 182). This relates back to the dysregulation of cortisol that occurs during chronic stress. Secretion of cortisol is diminished due to changes in the HPA axis of the brain. Without cortisol, inflammation ensues, causing psychological disorders (Copeland et. al, pg. 7572).

The psychosocial and emotional level is characterized by hyperactivity in the amygdala, which is associated with intense mood and anxiety disorders, along with poor coping skills, and dysregulated cortisol secretion, which impacts behavioral

responses to stressful stimuli. The Vaughn-Coaxum et. al study explains that the HPA axis, the prefrontal cortex, and the effect that trauma has on these regions of the brain, are the likely sources from which stress response and decision making are altered (Vaughn-Coaxum et. al, pg. 843). The study goes on to describe how the amygdala, in individuals with PTSD, has abnormal volume and thus affects an individual's processing of anxiety, fear, and mood. The prefrontal cortex itself is influenced by cortisol and other glucocorticoids that have a mediating effect on behavioral responses to sustained stress and trauma. Failure of the prefrontal cortex to remain plastic during stress was associated with failure to regulate emotions and behavior (Sinha et. al, pg. 8840). A lack of self-regulation is a hallmark of poor coping skills and poor mental health.

On the societal level, mental health issues are still a stigmatized topic of discussion due to a lack of understanding. Those with mental health disorders are more likely than the rest of the population to be repeated victims of violence because of the disenfranchisement that comes along with stigma. To reiterate the World Health Organization study: "Mental health issues are addressed in the report as both potential causes and consequences of these different types of violence" (Mercy et. al, pg. 21). The external displays of mental health disorders seem to elicit negative responses from others (Raine et. al, pg. 937). This occurs due to power plays of social stratification; an individual increases their social status by diminishing the already disenfranchised other (Copeland et. al, pg. 7572).

After an initial traumatic experience, an epigenetic tag alters the expression of a particular gene. The change in gene expression leads to dysfunctional production and secretion of molecules. The aberrant molecules then affect the functioning of organs and organ systems, such as the brain. When the molecular functioning of the brain is abnormal, the regulation of psychosocial and emotional states becomes abnormal as well. The individual, then, behaves abnormally, which elicits toxic responses from their society. These toxic responses lead to another traumatic experience and the cycle of revictimization begins again.

Discussion of Future Integrative Research

A biocultural view is paramount for all future research. Just because stressors can be reduced to the reactions of molecules does not mean that science is gaining a better understanding of how trauma works. As stated in the Graves article: "... the growing sophistication of modern biological techniques has not always allowed for the adoption of improved philosophical methodologies or ethical understanding regarding the applications of this new knowledge" (Graves, pg. 42). The body cannot be separated from the experiences of the individual within the body. Reducing human experience to molecules has the potential to disempower survivors' voices, to claim that biology, instead of personal perseverance, is the sole cause of their successes; to understand why revictimization occurs for some people and not for others, we cannot reduce everything to a genetic level because it takes away the autonomy and resilience of human beings to shape their own lives. Genetics can only do so much without the social environment to influence gene expression, behavior, health, biology, and sociocultural outcomes, as discussed by Harris and McDade in their synthesis of biological and sociological studies of human development (Harris and McDade, pg. 16). Simultaneously, we cannot blame the victims for their inability to prevent revictimization in their lives; the more exposure an individual has to trauma, the more likely negative outcomes will continue to occur (Vaughn-Coaxum et. al, pg. 854). In addition, factors such as structural inequality and biological damage are difficult to overcome, with or without traumatic experiences. This does not mean that some people are biologically determined to be trapped in cycles of trauma and revictimization; it means that different combinations of environmental and genetic factors have the potential to produce different results given the degree of attention that is known and paid to trauma and revictimization triggers. It is difficult to determine if an effective "treatment" for trauma and revictimization will ever be discovered, although there are promising avenues. There are, however, some issues with a "treatment" or "immuniza-

tion" to stress from trauma. There is the potential for treatment of only the symptoms (Anda et. al, pg. 182) within individual bodies, while the focus is shifted away from treating adverse childhood experiences or society's relationship with violence (Lock, pg. 163). The tentative hypothesis of the biocultural trauma feedback loop, posited by this research, could be of use as a holistic avenue for future trauma research. A wide array of biological and sociological factors of trauma can be understood and addressed comprehensively, while keeping the lived experiences of survivors front and center. Utilizing this method, can lead to fuller, holistic, and more nuanced research on trauma and revictimization, as well as the myriad of avenues through which these occurrences are created and recreated.

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