The Link Between Posttraumatic Stress Disorder and Youth Suicide in Native American Populations: A Literature Review

Taylor South
Carroll College, Helena, MT

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The Link Between Posttraumatic Stress Disorder and Youth Suicide in Native American Populations: A Literature Review

Taylor South

Carroll College
Abstract

Recent reports have illustrated the abnormally high, and growing, epidemic of youth suicide amongst the American Indian and Alaskan Native (AI/AN) population. This growing epidemic has led to many tribes calling for a state of emergency. Given that the suicide rate is much higher than the population average, research was conducted to find a correlational link. Based upon high rates of trauma within the AI/AN population and a history of trauma in the recent past, research was conducted to establish a link between posttraumatic stress disorder (PTSD) and suicide in this population. To establish a correlation between PTSD and the suicide epidemic, 132 sources were analyzed and possible connections were established. It was discovered that PTSD can arise via several routes: biological, psychological, sociological, and through cultural narrative. Furthermore, evidence suggests that PTSD can be transmitted intergenerationally by way of all previously stated routes. Overall, those exposed to multiple traumas earlier in life are more likely to develop PTSD, and thus, be at risk for suicide. On the contrary, it was found that a sense of culture and identity can be protective factors against both PTSD and suicide. If a causative link can be established between PTSD and suicide than appropriate therapies can be developed to reverse a growing epidemic.

Keywords: Historical Trauma, Posttraumatic Stress Disorder, Suicide, Native American, Alaskan Native
The United States of America is often referred to as the great melting pot. What makes this great country unique from many others is its diversity in cultural and ethnic backgrounds (Fearon, 2003). Coming from numerous areas around the world, the settlers of this country brought with them traditions and customs from home. When it comes to working in the healthcare field, working with such a diverse population requires a willingness and openness to serve human beings that may be different than one’s self.

America’s first people, comprising 567 federally recognized tribes (Bureau of Indian Affairs, 2017; Indian Health Service, 2016a), make up 2% of the population which equates to 5.4 million people ("FFF: American," 2015). Experiencing a lower life expectancy and a disproportionate burden of disease, this population has long experienced an overall lower health status when compared to other Americans (Indian Health Service, 2016a). Though efforts are being made, an underfunded Indian Health Service and a cycle of poverty plague Native Americans in their pursuit of culturally sensitive healthcare and increased health status. Perhaps one of the most alarming statistics referring to disparities in Native American health is that of suicide rates among Native American youth. The suicide rate for American Indian or Alaskan Natives (AI/AN), aged 15-19 and 20-24, is 15.2 and 23.6 per 1,000 people, respectively (Heron, 2016).
This is about 2 and 1.5 times the national rate for each age group. Major depressive disorder, historical trauma, and family violence have been cited as risk factors for suicide in this vulnerable population (Rosston, n.d.).

Though mental illness is often considered a risk factor for suicide, it is typically linked with major depression and is not considered in posttraumatic stress disorder (PTSD). Posttraumatic stress disorder is defined as an inability to properly cope with a traumatic life event (Mayo Clinic Staff, 2017). PTSD often manifests itself in anxiety, fear, and behavioral changes revolving around the traumatic event. Research has found that the prevalence of PTSD is higher than the national average in several tribes (Beals et al, 2005a). A traumatic event is defined as, “a single, enduring or repeating experience that seriously threatens the physical or psychic integrity of the concerned person” (Schmidt et al, 2011). A higher prevalence of PTSD, both in parents and in youth, may create a generational cycle of the disorder that is hard to escape. Such a cycle may lower the quality of life, which in turn may lead to suicide ideation and possibly, attempt. On the contrary, the experience of a traumatic or seriously traumatic life event does not necessarily lead to the development of PTSD (National Collaborating Centre for Mental Health, 2005), as there are many protective factors. Though the growing youth suicide epidemic is multifaceted, I will examine the relationship between PTSD and the growing suicide epidemic in AI/AN youth. If a connection between PTSD and youth suicide can be established, such information could offer valuable insight to the prevention and treatment provided to this vulnerable population.

Unrest in Indian Country: Suicide Among Youth
Calling for a State of Emergency. Over the past couple of decades, suicide has risen in prevalence on many AI/AN reservations. Numerous recent news reports have detailed the haunting, heart breaking tales of this population’s battle with high suicide rates. The epidemic is detailed by stories such as the state of emergency called after a small Cree community experienced 101 suicide attempts in a period of 8 months (Domonoske, 2016); the Sioux in South Dakota grieving the loss of over 150 of their beloved youth in a year’s time (Nieves, 2007); or a Lakota tribe who had to deal with the loss of 9 young people amidst approximately 103 suicide attempts in a span of only 4 months (Bosman, 2015). The problem of youth suicide is not an outbreak on a local scale. The problem is an epidemic that has spread throughout several tribes across North America. Given that numerous tribes are seeing similar suicide rates, there may be a commonality in factors eliciting such behavior: an underlying cause to an epidemic. Historically, suicide was very rare in Native American culture, and it continues to be stigmatized to present day (Meyers, 2007). Prior to the Indian wars, assimilation, and boarding schools, suicide was not a problem within the Native American population. A history of trauma, combined with current hardship, may contribute to psychological instability in AI/AN youth, and thus be a predecessor of suicide.

The Youth Suicide Epidemic. Recently, epidemiological data on national suicide rates has brought the most attention to a specific minority group: AI/AN youth. With a combined gender suicide rate that is almost double the national average (Fig. 1), it is evident that suicide has become a serious problem for teens and young adults of Native descent. In addition, studies have found that young AI/AN men are twice as likely as any
other racial or gender group to commit suicide (Sifferlin, 2015b; Fig. 1). Staggering as they are, the numbers for AI/AN youth may be a low representation as Natives are often racially misclassified in medical and mortality records (Arias et al, 2008; Haozous et al, 2014). Extremely high numbers, mixed with a high rate of misclassification, embodies the severity of the problem that America’s Native population currently faces. Tragic stories mixed with staggering numbers detail the reality of the problem faced by youth of AI/AN descent.

**Figure 1.** A comparison of youth suicide rates in the United States for the year of 2012-2013. This graph was adapted from data presented by (Santhanam & Crigger, 2015) and (Sifferlin, 2015a).

**Suicide Risk Factors.** Arising from a mental or physical ailment, suicide is not a spontaneous act, but something that is premeditated (Smith et al, 2008). Given the contemplation needed to consider the act of taking one’s own life, there are certain
factors that influence a human being to attempt suicide. From a broad perspective, the CDC reports that the risk factors for suicide include: family history of child maltreatment, history of mental disorder, local epidemics of suicide, a feeling of isolation, and barriers to receiving mental health treatment (“Suicide: Risk”, 2016). A study conducted on AI/AN youth found that having a friend who attempted or completed suicide, a family history of suicide, drug and alcohol use, and sexual or physical abuse pose the greatest risk for suicide attempt. Of these risk factors, having a friend attempt or complete the act of suicide was found to be the most influential (Borowsky et al, 1999). Given that several of the risk factors for suicide exist within AI/AN youth populations, there is reason to believe that a higher prevalence of suicide either means that there exists a higher prevalence of the determined causative factors within this population or there exists factors that have yet to be determined. If the latter were to be true, it would suggest that these children may face greater amounts of various traumas, from mourning the deaths of friends and family, to physical and sexual abuse. Several of the same risk factors for suicide, especially loss of a loved one or physical or sexual abuse, are also risk factors for posttraumatic stress disorder (Mayo Clinic Staff, 2017). Given similar origins, these two mental disorders may exist as comorbidities or it may be possible that one disease may increase the incidence rate of the other.

The Characteristics of Posttraumatic Stress Disorder (PTSD)

*The Human Stress Response.* When exposed to a stressful stimulus, an innate stress response is activated to return the bodily system back to homeostasis. A stressful
event is characterized by either a biogenic or psychosocial stressor (Girdano, Dusek, & Everly, 2009). Biogenic stressors exist as actual molecules that initiate the stress response. In contrast, psychosocial stressors are much more difficult to characterize. These stressors, existing as real, imagined, anticipated, or recalled, are subjective in nature, and exist due to an individual’s interpretation of an event (Everly & Lating, 2013). Despite the origin or characteristics of the stressor, a similar “fight or flight” response is elicited. The fight or flight response is believed to be an adaptation that gave early humans and other organisms a response to promote survival when faced with environmental stressors. Once the response was elicited, they would have the biochemical tools to either fight or escape (flight) from the stimulus that was causing the stress. The physiological response is where this biochemical pathway derives its name.

Though much is still unknown, research has shown that environmental stimuli trigger the hypothalamus to release both corticotropin-releasing hormone (CRH) and arginine-vasopressin (AVP) (Randall, 2011; Schmidt et al, 2011), as well as to signal the adrenal glands to release epinephrine ("Understanding the Stress," 2016). Epinephrine has been shown to cause effects such as increased blood pressure, increased heart rate, and increased stimulation of skeletal muscles (Everly & Lating, 2013). CRH is a signaling molecule that activates the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary, which in turn leads to the production and secretion of glucocorticoids from the adrenal cortex (Smith & Vale, 2006). Acting on the hypothalamic-pituitary-adrenal (HPA) axis, the molecules in the glucocorticoid family are those that elicit, and
control, a physiological stress response. The HPA axis serves as a region of the brain linked to several systems of vital functioning. Along with the stress response system, some others include biological processes related to mood, growth, immune function, metabolism, and the circadian rhythm (Timmermans et al, 2013). Though this process is often quite flexible, over or under expression of glucocorticoids has been linked to the development of pathologies (Flandreau et al, 2012; Schmidt et al, 2011; Varghese & Brown, 2001).

Cortisol, a glucocorticoid, is the primary actor in the human stress response. Modulating several pathways, cortisol has been discovered to play a role in metabolism regulation, suppression of the immune response, and the regulation of ion balances (Randall, 2011; Sapolsky, Romero, & Munck, 2000). Being a signal that has a systemic effect, cortisol’s main role is to return a body affected by stress back to homeostasis. Cortisol acts via a negative feedback effect on the hypothalamus, pituitary, and hippocampus (Bremner, 2006). Balancing the stress response, the negative feedback pathway of cortisol helps to maintain a safe level of blood cortisol, and shuts down the stress response once homeostasis is obtained.

Simultaneous to the actions of cortisol, AVP signals for the kidneys to uptake water, producing less urea, as well as to cause vasoconstriction, causing hypertension (Randall, 2011). In addition, the hypothalamus signals for the release of epinephrine from the adrenal glands. Epinephrine is a systemic acting signaling molecule that increases heart rate, spikes blood pressure, increases respiratory rate, and causes the body to release glucose in the bloodstream (“Understanding the stress”, 2016). Taken
together, these various chemicals cause an overall increase in awareness, energy, and sensual perception. Signals released from the brain ultimately trigger a complex physiological response that allows the organism to respond to the stressful stimulus (Fig. 2). This response shifts all bodily functions to coordinate for one common goal: the survival of the organism.

**Figure 2.** A depiction of the human stress response. A stressor - real or perceived - causes a cascade of neurological events that ultimately leads to the release of systemic signaling molecules. Of the chemicals utilized in the stress response, cortisol plays the most important role. CRH is an abbreviation for corticotropin-releasing hormone, ACTH an abbreviation for adrenocorticotropic hormone.

**Defining PTSD.** Post-traumatic stress disorder (PTSD) is defined as a continuing stress response, one that lasts far beyond the normal recovery time to a traumatic event. Symptoms of this disorder often include disturbing memories, avoidance of
anything associated with the event, intrusive thoughts, nervous hyperarousal, negative mood, changes in memory and concentration, and changes in emotional reactions (Bremner, 2006; Mayo Clinic Staff, 2017; Schmidt et al, 2011). Often not standing alone in a clinical diagnosis, PTSD is linked with frequent comorbid psychological disorders (Panagioti et al, 2009). Unable to regulate the fight or flight response, PTSD patients are plagued by the event in which the PTSD originated. It is believed that the malfunctioning stress response is created by structural or chemical changes to the structures in the brain that control it. Biologically, this leads to an under or over stimulation of the stress response, and thus, a dysfunctional system. A system that is incapable of responding properly to stressful stimuli leads to the physiologic characteristics associated with PTSD.

**Psychological Effects.** Arising from an individual’s interpretation of a potential stressor, psychosocial stimuli can cause a merely identical response to that of a stressor that is biological in origin. As the psychosocial stimuli are not actually causing a biochemical reaction themselves, the stress response is completely subject to an individual’s interpretation of that stimuli. In fact, it has been shown that the physiological reactions of the stress response are not caused by non-biogenic environmental stimuli, but the cognitive interpretation and emotional response to them (Everly & Lating, 2013). What differentiates trauma survivors, those who develop PTSD and those who do not, depends on the individual’s interpretation of the event. For example, a soldier who feels that he is responsible for his comrade’s death may be more likely to develop PTSD than another traumatized soldier who does not have this.
perception. Though the biochemical response is the same among human beings, the subjective response to the stimuli may have lasting effects on the genome, and thus, one’s ability to cope with stress later in life (Yehuda, 2002). Therefore, the psychological interpretation of an event may lead to biological problems down the road. In such a model, cognition, especially that of perception, alters biology. Resulting from an individual’s unique response to a stressor, PTSD is hard to predict, though it is possible to identify people who are at a greater risk.

**Biological Effects.** The biology behind PTSD is a complex process that works across numerous neurological systems. When studying the biology of PTSD, one must first know how the stress response works, and then he or she will understand how variations in this response can lead to PTSD symptomatology. Causing dysfunction to the stress response, PTSD works within the neurochemical and neurological systems (Fig. 2). Currently, it is believed that those at higher risk for developing PTSD do so because of a weakened stress response at the time of a trauma (Yehuda, 2002; Sherin & Nemeroff, 2011) or due to a weakened coping ability resulting from prolonged exposure to trauma. Therefore, those who experience multiple or prolonged trauma are at risk. A dysfunction somewhere in the complex stress response system may be responsible for the inability to properly cope with a traumatic stressor and ultimately the development of PTSD symptomatology.

**Neurochemical Factors.** The two chemicals that have been shown to be systemic actors of the stress response are cortisol, also known as glucocorticoid, and
norepinephrine (Bremner, 2006). Acting as the primary signaling molecule in the stress response, glucocorticoids elicit effects on metabolism, brain, and immune function (Sherin & Nemeroff, 2011; Randall, 2011). Currently, glucocorticoids are understood to affect the potential development of PTSD via two different routes. Studies have found that extended exposure to glucocorticoids, which would be brought forth from prolonged trauma exposure, leads to death of the neurons in the hippocampus (Sapolsky, Krey, & McEwen, 1985). Therefore, persons exposed to trauma for an extended period would slowly lose the ability to cope if no improvement was made against the cause of the stress.

Furthermore, though it is not conclusive, some studies have found a correlation between a pre-trauma state of hypocortisolism and the development of PTSD. Studies conducted on survivors of the holocaust, 9/11 victims, and war veterans have all found this phenomenon (Yehuda et al, 2005). Discovering low levels of either blood or urinary cortisol in patients with PTSD, these studies suggest that hypocortisolism may be a factor associated with the disease. What is most surprising is the discoveries suggesting that the hypocortisolism may be passed on generationally (Yehuda & Bierer, 2008). Transmission of low cortisol levels would immediately put offspring at risk for dysfunctional stress coping ability, and possibly the development of PTSD. Explaining two different routes of development, evidence suggests that PTSD symptomatology arises via both an over and under expression of glucocorticoids.

In addition, those diagnosed with PTSD have also shown an inability to regulate
norepinephrine (NE) levels. NE is known to work alongside both CRH and vasopressin to affect fear conditioning, hyperarousal, and memory encoding (Schmidt et al, 2011; Sherin & Nemeroff, 2011). One study found elevated residual levels of norepinephrine in the cerebrospinal fluid of war veterans with PTSD (Geracioti et al, 2001).

Norepinephrine is released from the adrenal medulla and works as both a neurotransmitter and a hormone (Rice University, n.d.). In the stress response it acts to increase heart rate, to increase blood flow to skeletal muscles, and to release glucose from its stores as glycogen. Increased residual levels of this hormone would elicit a state of hyperarousal.

**Neurological Factors.** The bodily structures associated with the control of the stress response are the amygdala, hippocampus, prefrontal cortex, and HPA axis (Bremner, 2006; Randall, 2011; Siddiqui et al, 2008; Smith & Vale, 2006; “What is Post”, 2016). The functions, and dysfunctions, can be viewed in table 1. Post-traumatic stress disorder symptomatology arises through the biochemical malfunctioning or structural impairment of these structures.

**Table 1.** The different brain structures that play a role in the symptomatology of post-traumatic stress disorder. Each structure plays a unique role in the human stress response. Normal functions and dysfunctions are described (Bremner, 2006; Siddiqui et al, 2008; Smith & Vale, 2006; “What is Post”, 2016).

<table>
<thead>
<tr>
<th>Brain Structure</th>
<th>Normal Function</th>
<th>Impaired Function in PTSD</th>
</tr>
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<tbody>
<tr>
<td>Amygdala</td>
<td>Functions in the processing of emotions. Triggers the fight or flight response.</td>
<td>Increased activation triggers fight or flight in response to memories or perceived...</td>
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</tbody>
</table>
**Hippocampus**  
Responsible for the formation of long term memories.  
Damage to neurons causes dysfunction in memory formation and retrieval.

**Prefrontal cortex**  
Functions in complex decision making, memory recovery, intelligence, language, and behavior.  
Damage causes inappropriate behavioral responses and poor decision making.

**Hypothalamus-pituitary-adrenal (HPA) axis**  
The control center for various endocrine responses. In the stress response, the HPA signals the release of cortisol.  
Increased activation causes overproduction of stress hormones. Thus, stimulating the stress response at inappropriate times.

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**Risk Factors for American Indians and Alaskan Natives.** Given the various routes of arousal, PTSD can manifest itself in numerous different ways. Ultimately the manifestation of PTSD results from a biologic change in the stress response system created by the psychological perception of stress. Such perceptions arise due to actual, remembered, or imagined trauma. What puts AI/AN at a higher risk for this disease is the incorporation of trauma into their daily life experience via historical trauma, childhood maltreatment, and trauma due to accidents (BigFoot, 2007; Bombay et al, 2009; Borse et al, 2008; Brockie et al, 2015; Fig. 4; Fig. 5; Manson et al, 2005 Rosston, n.d.; Sotero, 2006). All three factors have been shown to have a correlation in the manifestation of PTSD (Sandica & Pop, 2014). Furthermore, recent studies have revealed a connection between the development of PTSD in youth and their pre-traumatic risk factors (DiGangi et al, 2013) and their post-traumatic recovery environment (Trickey et al, 2012) rather than the trauma itself. Both theories suggest
the importance of dysfunctional coping factors, psychosocial impairment, and unhealthy system of support as risk factors for the development of PTSD. High incidences of child maltreatment (U.S. Department of Health and Human Services, 2016) suggest that Native children are more likely to be exposed to pre trauma factors. In addition, they also may not receive the proper support to combat the trauma that may arise in their daily life experience. The stressors on reservations are most often chronic, which results in hyperactivity of the stress response system and thus impacts its ability to properly function (Brockie et al, 2013). A high incidence rate of trauma exposure mixed with pre-exposure risks and a toxic coping environment puts AI/AN youth at a proposed higher risk for developing PTSD.

Along with these risk factors, Native youth may also be at a genetic disadvantage for developing the disease. Studies within the field of epigenetics suggest that reduced stress coping ability may be passed onto offspring (Davidson et al, 1998; McFarlane, 1988; Skelton et al, 2011; Reich et al, 1996). Therefore, the occurrence of PTSD in Native populations may be in cyclic form. The high rates of trauma, toxic living environment, biological inability to cope, and a possible genetic component put Native youth at a severe disadvantage for preventing the disease after trauma has occurred. Such findings are pivotal in explaining the recurring phenomenon of youth suicide amongst the AI/AN people, for PTSD is a risk factor for suicide attempt (Brockie et al, 2015; Gradus et al, 2010; Nepon et al, 2010; Sareen et al, 2005). A reduced stress coping ability in combination with the influences of historical trauma, childhood maltreatment, and high accidental trauma rates may be what is eliciting the development of PTSD. Struggles
with PTSD symptomatology mixed with inadequate support systems may be the combination fostering suicide attempt in these young PTSD patients.

**A History of Trauma**

Recent research has shown that family history may have a bigger impact on the present generation than previously expected (Brown-Rice, 2013; Doucet & Rovers, 2010; Sotero, 2006). From the Trail of Tears to boarding schools for Native youth, the AI/AN populations have faced many severely traumatic events that may have led to the current crisis facing the culture. The systematic destruction of the AI/AN way of life brought about losses of people, land, culture, and family that may still influence the present-day generation (Brown-Rice, 2013). Trauma that occurred a few centuries ago may have created transgenerational trauma that has contributed to poor mental health status of many present day Native Americans (American Psychiatric Association, 2010; Manson & Altshclul, 2004). Research suggests that trauma can be transferred to successive generations via: children identifying with their parents suffering, children being influenced by the way the trauma is talked about in the household (Mohatt et al, 2014), children being affected by certain parenting styles (Doucet & Rovers, 2010), and children inheriting certain epigenetic markers (Yehuda & Bierer, 2008; Kellermann, 2013). This generational line of trauma, whether biological or psychosocial, may subsequently be putting AI/AN youth at a greater risk for developing PTSD. Many of the above factors are perceptions of a historical or current day situation. As discussed earlier, perception of stress breeds the physiological activity of the stress response system. Being exposed to a negative perception of family or cultural history early on in
life could have serious ramifications in the psychosocial development of AI/AN children. Low sense of self worth has been connected to a multitude of mental illnesses, specifically depression and anxiety disorders (Mann et al, 2004). Conversely, the positive promotion of culture has been shown as a protective factor against suicide (Chandler et al, 2003).

Along with the psychosocial factors related to the home life, AI/AN children may also be biologically predisposed to developing a reduced stress coping ability. Evidence suggests that epigenetics, which will be discussed in greater detail later, may play a role in a weakened stress response (Skelton et al, 2011; Kellermann, 2009). Couple a genetic susceptibility with psychosocial risk factors, and the child becomes much more at risk for developing symptomatology. A suppressed ability to respond to stressful stimuli coupled with a home environment that is either hopeless, violent, or a combination of the two puts children in an environment that may foster the development of PTSD.

The Theory of Historical Trauma. Similar to what is known as intergenerational trauma, Historical Trauma (HT) affects not just a family, but an entire population of people. According to Gagne and Duran, HT can be defined as, “a combination of acculturative stress, cultural bereavement, genocide, and racism that has been generalized, internalized, and institutionalized” (as cited in Wexler, 2009, p. 267). Furthermore, HT acts as a culmination of traumatic events that affect an individual and his or her subsequent generations (BigFoot, 2007). Sotero (2006) explains that HT is comprised of four main characteristics: (1) mass trauma is inflicted upon a target population in a domineering and systemic way, (2) the trauma is not limited to a single
event, but is stretched out over a period of time, (3) a cycle of trauma is created within
the population, (4) the trauma becomes so systemic that it spreads throughout all
aspects of the life course, and cripples the population in physical, psychological, social,
and economic aspects (Sotero, 2006). HT proposes an event that affects more than just
a few individuals, creates a cycle of trauma within the population that outlasts the initial
event, and ultimately leads to a dismantling shift in the culture. Recounting the
devastating experiences of North America’s Native peoples over the past 300 years may
help explain the struggles that these populations currently face. HT that is specific to the
Native populations of North America can be defined as a “collective experience of
violence perpetrated against Indigenous Peoples in the process of colonizing the
Americas resulting in an unresolved humanitarian crisis for reservation communities”
(Brockie et al, 2013, p. 2). This trauma occurred through the processes of forced
relocations, sending of children to boarding schools, prohibition of language and cultural
practices, restriction from sacred places, and outlawing of traditional religious practices
(BigFoot, 2007; Rosston, n.d).

Connected to a lost sense of identity and a feeling of despair, HT is being
discovered to have large emotional and psychological impacts on Native people. Further
research suggests that HT, although not directly experienced, can manifest itself in
similar ways as experienced trauma. Current studies are suggesting that the negative
narrative surrounding AI/AN cultural history and the negative perception associated
with being part of this ethnic group is the true manifestation of HT (Mohatt et al, 2014;
Wexler, 2009). Such research states that a negative historical narrative, when in
connection with modern issues, breeds a narrative that becomes entwined with the contemporary culture. Each human being’s personal narrative, and thus one’s identity, stems from social interactions within a larger cultural context (Hammack, 2008). The beliefs surrounding HT, and its impact on identity, ultimately has an effect on what it means to be a part of the AI/AN ethnic group.

A strong connection to culture has been found to be a protective agent against mental illness (Walters & Simoni, 2002), and to further promote healthy outcomes (Zimmerman et al, 1996). Consider, for example, the Native American reservation. Once serving as the residential confinement area for relocated AI/AN, these areas are now essential for cultural indoctrination. Reservations serve as a both a reminder of heartbreak, as well as a base for the continuity of culture (Mohatt et al, 2014). One’s perception of such an area, whether it be a sense of loss or one of resilience, may have an impact on psychological wellbeing. The loss and suffering experienced by AI/AN may be communicated at a much greater frequency than the positive aspects of cultural history. A negative cultural narrative paints a negative light on what it means to be Native American. When presented to children, such a narrative breeds avoidance of one’s cultural history and low self-esteem (Jackson & NCTSN Culture Consortium, 2014). Though the efforts of assimilation may seem to be in America’s ancient past, such efforts ended in the second half of the twentieth century (Native American Public Telecommunications, 2006). The people who experienced this humanitarian crisis are the parents of today’s middle-aged adults and the grandparents of today’s youth. Given that the assimilation process stretched to all tribes and reservations, such a large-scale
transmission of transgenerational trauma suggests the possible existence, and large-scale impact of Historical Trauma.

**Intergenerational Trauma.** Though the massive trauma faced by Native Americans occurred in the late nineteenth century, its effects still appear to be plaguing Native peoples to this day. The failure to properly cope with the effects of HT have resulted in the transgenerational transmission of what has come to be known as Intergenerational Trauma (IT). Occurring when trauma is not resolved, but internalized, IT is the transmission of trauma from one generation to the next (BigFoot, 2007). Such an ailment arises from impaired parenting or lack of effective intervention and support from the community. Brokenness within support systems, such as the family unit or community, may result in a youth’s inability to successfully draw from these resources when faced with a stressor (BigFoot, 2007). The development of poor stress coping, greater exposure to stressors, or mental illness may then be generated within these children. As these factors often continue to exist into adulthood, they may thus in turn alter their own parenting styles (Bombay et al, 2009; Fig. 3). Poor parenting may lead to childhood trauma exposure, and thus, contribute to similar ailments into the subsequent generation. For example, a mother’s PTSD may contribute to PTSD in her child due to affected parenting styles because of the disease. In this sense, trauma is passed generationally via psychosocial routes. Therefore, it is not solely biological in nature, but based upon human social behavior as well.
Figure 3. The proposed cycle of the intergenerational transmission of trauma. A parent (generation 1) is exposed to adverse childhood experiences (ACEs). This parent later develops reduced stress coping ability, increased stress exposure (due to environment or behavior), and/or mental illness. These factors are interconnected, but can function independently. Poor parenting styles then result from the previous factors. Poor parenting exposed generation 2 to ACEs. This figure was adapted from the data presented in (Bombay et al, 2009).

Theories such as Historical Trauma and intergenerational trauma help to explain the generational inheritance of a dysfunctional stress coping ability via psychosocial methods. Such theories suggest that trauma is passed onto the next generation because it is either created or not properly managed by parents and elders. In the case of PTSD, the prevalence in AI/AN is high compared to other populations. AI/AN make up 1.7% of
the American population but 3.2% of America’s PTSD patients—almost twice the
expected rate (Bassett et al, 2014). A high prevalence rate of PTSD may create poor
parenting and debilitated support systems, which may amount to the cyclic
manifestation of trauma throughout generations.

The Genetic Component of PTSD

Various studies have shown a link between traumatic events and their lasting
effect on the genome. Such alterations have been known to arise due to epigenetic
biochemical processes (Brockie et al, 2013; Murgatroyd et al, 2010; Oberlander et al,
2008). Epigenetics is defined as the expression of certain physiological traits through the
regulation of genes (Chiang & Su, 2015). The epigenome is formally defined as, “the
entirety of all molecular control elements programming the genome respectively
regulating gene activities” (Schmidt et al, 2011). Epigenetics considers the organism’s
interaction with its environment, and hypothesizes about gene alteration in response to
environmental cues. In the scope of epigenetics, genes are internally regulated in
response to external stimuli. If the epigenetic hypothesis is true, then human beings can
genetically respond to both real and perceived environmental stimuli. Such a possibility
gives greater importance to the experience of humankind in one’s growth and
development. Recent literature states that genetic factors and environmental influences
are, in fact, dependent on each other and acquired environmental information directs
the usage of the genetic material (Murgatroyd et al, 2010). Eva Jablonka, Israeli theorist
and geneticist, explains the epigenetic phenomenon well when she states, “What we
know about the epigenetic marks is that they can dispose one towards developing some
behaviors, but the specific behavior depends on specific inputs the person gets in its own lifetime” (Kellermann, 2013).

**How epigenetics works.** To fully understand the impact of epigenetics, one must first establish a foundational knowledge of the cellular genome and how it works. As is taught in the central dogma of molecular biology, deoxyribonucleic acid (DNA) is transcribed into ribonucleic acid (RNA), which is then translated into protein (Crick, 1970). From enzymes in the stomach to neurons in the brain, protein molecules carry out numerous functions that are vital to life. In this model, DNA serves as the instructions for biological activity within a cell. The building blocks of DNA are the nitrogenous bases known as cytosine, guanine, adenine, and thymine. What is peculiar is how multicellular organisms, who contain nominally identical genomes, maintain different phenotypes from each other (Riddihough & Zahn, 2010). The answer to this mystery is believed to be found in epigenetics. Epigenetics is believed to be a genetic code that is influenced by the environment, and carries out the ability to regulate the genome and to develop stable alterations in phenotypes within one or two generations (Chiang & Su, 2015). Eliciting alterations in the genetic code leads to the production of different kinds of protein, which in turn, affect a different function on the cell. Epigenetic control is therefore the switch that turns genes on or off based upon environmental cues.

**The importance of methylation.** Our life story as humans is a compilation of both biology and experience. Current scientific evidence suggests that human beings are concurrently genetically predestined, and at the same time, highly malleable
(Kellermann, 2013). Though human beings are born with approximately 20,000 genes, these genes are constantly being modified throughout the course of daily life. Current knowledge suggests that the human genome is commonly modified through the processes of DNA methylation and histone deacetylation, which alter chromatin structure, and ultimately, the degree to which genes are transcribed (Schmidt et al, 2011; Nan et al, 1998). The plasticity of human gene expression allows the species to actively adapt to a changing environment. Such genetic modifications are not always positive though. A recent study conducted on mice found that early life stress leads to the phosphorylation of a repressor protein on the arginine vasopressin (AVP) gene (Murgatroyd, 2009). While phosphorylated, the repressor cannot bind the genetic elements, and thus, the gene is transcribed. During transcription, the methylation of the DNA is altered, affecting the binding site of the repressor. Rendered dysfunctional, the repressor does not bind correctly and the gene continues to be transcribed. Such an event leads to HPA hyperactivity as well as reduced stress coping ability. Altering the level of methylation directly affects the binding ability of genetic elements, such as the repressor.

Studies in humans have linked the degree of DNA methylation, specifically in neurotransmitter genes, with the development of PTSD (Brockie et al, 2013). A study of methylation in suicide completers found that both hypo and hypermethylation cause permanent transcriptional change across the genome, especially in the neuroplasticity of cells in the hippocampus (Labonte et al, 2012). Furthermore, researchers studying the genetics of those who completed suicide, found a positive relationship between DNA
methylation and abuse experienced at an earlier age (McGowan et al, 2009). Such evidence suggests that methylation of DNA, particularly in human neuronal cells, ultimately leads to their dysfunction, and thus, the risk for psychopathological disease. More specifically, studies have elucidated that the gene most often associated with psychopathology is the human glucocorticoid receptor (GR) gene (Klengel et al, 2013; Radtke et al, 2015; Tyrka et al, 2012). Hypo or hyper expression of this gene alters the cortisol response received by the HPA axis, via a negative feedback loop (Randall, 2011), and alters the body’s innate stress coping system. It is proposed that alterations to the GR gene are the genetic changes that result in the physiologic manifestations of altered stress coping and psychological hardship.

Though methylation does not cause structural change to the gene, it does change how the gene is expressed through transcription. Changing gene expression changes gene products, and thus, phenotypes of the organism. Causing permanent change, trauma affects not only psychological aspects of daily life, but the actual structure of genetic makeup. The degree of change that occurs during or before adolescence can leave a lasting impact on adult life.

**Are Nightmares Inheritable?** Given that exposure to trauma has the potential to epigenetically alter gene expression, the next question is whether these genes, or rather gene alterations, can be inherited. If epigenetic modifications can alter a parent’s stress coping ability, could this same trait manifest in their children? In Indian country, where violence and trauma are particularly high (American Psychiatric Association, 2010; Fig. 1; Fig. 4; Fig. 5), a reduced ability to cope with stress may reinforce illnesses such as PTSD
and behaviors such as suicide attempt.

In fact, family and twin studies have shown that a moderate risk factor for PTSD is genetic in origin; PTSD symptomatology and the experience of assaultive trauma have shown to be inherited at a variance of 30-50% (Ehlers et al, 2013; Skelton et al, 2011). Furthermore, studies within the last couple of decades have shown that those with PTSD, as compared to trauma survivors without PTSD, are three times more likely to report familial struggles with anxiety, depression, psychosis, and antisocial behavior (Davidson et al, 1998; McFarlane, 1988; Reich et al, 1996). Such a finding is suggestive of these mental behaviors being connected through family lineage. Though inheritance of PTSD has been physiologically modeled, there is not much concrete biological evidence currently. The transgenerational transmission of epigenetic changes has been well studied and elaborated in plants and some less complex animal species (Heard & Martienssen, 2014), and there exists some indirect evidence for transmission in humans (Sen et al, 2015). As research continues to uncover the biological component of PTSD, an inheritable trait may one day be discovered. However, current lack of biological evidence does not mean that some forms of trauma cannot be passed down socially, or even culturally.

Though trauma is still important in the causation of PTSD, some may be at a greater disadvantage than others. Being genetically less able to cope with stress is a causative factor of PTSD development in offspring (DiGangi et al, 2013). Though this type of behavior may be an outcome of PTSD, it is not the rule. In fact, the flexibility of epigenetics suggests that, depending on environmental factors, the same individual
could express both behaviors of overwhelming anxiety as well as rock solid stress coping ability at different times throughout their life (Kellermann, 2009). This evidence suggests that if epigenetic markers for PTSD can be inherited, they are not set in stone, and thus, are susceptible to medical, psychosocial, or occupational intervention.

If PTSD is genetically inherited, then evidence should lie in past and present victims of trauma. Looking at two events that caused large-scale trauma on numerous victims, the Holocaust and September 11, one can see that there exists a common thread between the two: the passing of genetic susceptibility to PTSD. Rachel Yehuda—the Director of the Traumatic Stress Studies Division at the Mount Sinai School of Medicine—has attempted to examine the effects and transmission of PTSD in Holocaust survivors (2002, 2008). One study conducted by Yehuda’s team found that the amount of urinary, salivary, and plasma cortisol was lower in the offspring of survivors with PTSD as opposed to the offspring of those without (Yehuda & Bierer, 2008). As discussed earlier, cortisol, a signaling molecule released by the hypothalamic pituitary adrenal (HPA) axis, is the main hormone responsible for the stress response. If cortisol is underproduced by the body, then the stress response is underutilized, and the body cannot respond to stress as well. Thus one’s stress coping ability is altered. The low amounts of cortisol found in the offspring of PTSD victims suggests these children may have inherited a weakened stress response from their parents. Yehuda and her colleagues found a strong correlation relating maternal PTSD and infant salivary cortisol levels to parental PTSD and urinary cortisol levels in adult offspring (Yehuda et al, 2002). Observing the presence of the same disease across generations suggests that the
The link between posttraumatic stress genetic information for PTSD is heritable. As discussed earlier, PTSD is not a specific gene, but the way in which a set of genes involved with the stress response is regulated.

As PTSD may epigenetically modify the stress response, the production of cortisol could be down regulated as an effect of this disease. The presence of this same deficiency in the stress response of offspring, even though they were not exposed to the traumatic event, suggests that the weakened stress response came about from one of two ways: trauma in early childhood or genetic inheritance.

The Trauma Experienced by Youth

The Effects of Early Trauma. In line with the theory of transgenerational transmission of PTSD, the effects of trauma early in one’s life experience can have life altering repercussions. Parents who struggle with symptomatology of PTSD may pass on the disease, not biologically, but through the way in which they treat their children. In this model, social transmission of trauma is the key component. Babies who were raised under situations of neglect or abusive care showed lower plasma cortisol levels (Gunnar & Bazquez, 2001). Biologically, tests in animals have found that trauma to infant stage offspring results in sustained plasma cortisol levels (Levine et al, 1993). Given the early developmental stage of these animals, it may be likely that underdevelopment of both the HPA axis and the negative feedback pathway of cortisol may be responsible for maintained blood cortisol levels.

More recent evidence has found that early life trauma also has the power to elicit epigenetic changes. Studying infantile mice, who have been separated from their
mothers, scientists have discovered a biochemical process that creates a genetic scar on the mammals (Murgatroyd et al, 2010). Stress early in life has been linked to the phosphorylation of MeCP2, a protein that is believed to function in gene expression by modifying chromatin (U.S. National Library of Medicine, 2017). When phosphorylated, the MeCP2 protein is unable to bind the AVP enhancer site and repress transcription. Though the MeCP2 protein does not remain in the phosphorylated state, when it regains its function DNA methylation has been reduced and thus its binding ability becomes substantially impaired. This genetic change leads to high levels of AVP transcription, which along with CRH, has been linked to reduced stress coping ability and HPA hyperactivity (Beurel, & Nemeroff, 2014; Flandreau et al, 2012).

Figure 3. The epigenetic changes related to the dysregulation of the AVP gene. 1. The MeCP2 protein acts as a repressor of the AVP gene. 2. Due to high levels of early life stress, the MeCP2 is phosphorylated and removed from its location on the enhancer site of the AVP gene. 3. Due to prolonged absence of the
repressor, the enhancer becomes demethylated. 4. Due to the demethylated sites on the AVP enhancer, MeCP2 is unable to bind upon dephosphorylation. Figure was adapted from information retrieved from (Murgatroyd et al, 2010).

Not only does the incidence of trauma affect offspring, but the duration of the trauma matters as well. Research shows that prolonged trauma can cause the bodily system to override factors that would normally be inhibiting (Stanton & Levine, 1988). This can lead to both hyperactivity of the HPA axis as well as an inefficient cortisol negative feedback pathway. Such evidence suggests that trauma early in life can elicit the same genetic modifications seen in adults. It is believed that mothers suffering from PTSD may exhibit different or inconsistent behaviors toward their offspring, which in turn may cause development of PTSD in the subsequent generation (Yehuda et al, 2005).

In fact, a study conducted among two separate Native American tribes, encompassing 3086 members, found that the highest lifetime rate of disorder in American Indian women was PTSD (Sarche & Spicer, 2008). Though this data is only descriptive of two tribal groups, it is suggestive that children of at least some tribes may be at a higher risk of acquiring PTSD via trauma early in life. Mothers who are struggling with PTSD, and without proper treatment, may submit to parenting styles that are harmful to youth. A study on mice who experienced maltreatment as pups found increased methylation BDNF gene and abnormal child rearing behaviors later in life (Roth et al, 2009). Upon testing the pups who were raised by maltreated mothers, researchers found similar methylation patterns on the BDNF gene. Scientists then cross-
fostered pups between control group and previously maltreated mothers. The analysis of this nature versus nurture environment found that both prenatal and postnatal environments play a factor in youth development and DNA methylation patterns. In humans, third trimester exposure to depressed/anxious maternal mood has been linked to increased salivary cortisol levels (Oberlander et al, 2008). Therefore mothers who are affected by mental illness may pass on those traits through traumatizing parenting styles, or through *in utero* biochemical transmission. Both routes appear to have an effect on the developing brain of youth.

Research has shown that trauma can be transmitted both biologically and psychosocially. Both pre and postnatal trauma have been shown to affect DNA methylation patterns, subsequently passing on these epigenetic changes to the next generation.

**Trauma on Reservations.** Ranging from emotional and physical abuse, to various forms of neglect, childhood trauma takes many forms. AI/AN youth are exposed to violence at much higher rates than their peers. Compared to other racial groups, AI/AN were anywhere from 1.5 to 3 times more likely to experience violent victimization than any other group (American Psychiatric Association, 2010; Truman et al, 2013). A study conducted across two separate reservations found that 62-70% of the residents experience at least one traumatic event during their lifetime (Manson et al, 2005). Another study, conducted amongst 309 participants who reside on a reservation, found that 94% of them had experienced a traumatic event (Ehlers et al, 2013). Though the latter is slightly higher, similar statistics have been found in the general population.
Further evidence suggests that AI/AN children are often exposed to multiple, continuous traumas throughout their childhood (Kenney & Singh, 2016). Higher incidence of repeated trauma exposure may explain why AI/AN populations currently face a higher rate of PTSD (Bassett, 2014). Given its high prevalence and continuity, trauma appears to be woven into the developmental years of those who grow up on reservations. The exposure to multiple traumatic experiences in childhood has been linked to the development of PTSD in AI/AN children (Deters et al., 2006).

Furthermore, not only is the trauma rate high for AI/AN youth, but this same population of children is at a serious risk of being bystanders. For example, AI/AN children are more likely than children of any other racial background to witness their mother being abused (Wahab & Olson, 2004). Given the interconnectedness of AI/AN communities, studies also suggest that AI/AN children may be indirectly affected by trauma that is directed towards friends or family. In this instance, the youth are not the victims themselves, but bystanders (Sarche & Spicer, 2008). Being in the position of a bystander may have a more significant impact on those in a close-knit AI/AN community than it would for the general population. According to Robin, Chester, and Goldman, the interconnectedness of Native American communities may lead to a serious trauma impacting far beyond one’s immediate family and friends (as cited Sarche & Spicer, 2008).

Higher rates of death due to serious trauma leave youth at a greater risk for becoming a bystander, especially in a close-knit community structure. Given the close-
knit relationships among relatives and non-relatives in Native American culture and the bystander effect, trauma to an individual in an AI/AN community stretches far beyond that of just family members. Taking into consideration the impact of serious trauma on the AI/AN community, the rate of death due to unintentional injury, such as a car crash or accidental drug overdose, among this group is two times the national average and higher than all other racial groups (Fig 4). In youth specifically, the death rate from unintentional injury is 200% greater in AI/AN males and 20% greater in AI/AN females than the national average (Fig 5). Unintentional injury is commonly defined as injuries resulting from motor vehicle crashes, falls, fires and burns, drowning, poisoning, and aspirations ("Unintentional Injury," 2013). As defined, unintentional injury can be a source of traumatic death. As can be seen figures 4 and 5, AI/AN populations witness deaths due to unintentional injury, both in members of the youth age group and in those outside of it, at a rate that is higher than the national average. Both experienced and witnessed trauma have links to PTSD and suicide (Bombay et al, 2009; Miller et al, 2013; Patki et al, 2014). Therefore, merely being a witness to a peer or family member death, due to unintentional injury, may put the bystander at greater risk for PTSD development and suicide. Given that both PTSD and suicide attempt arise from this familiar source, they may correlate in their effects. A child who is exposed to an environment with such a high death rate, especially among their peers, becomes more susceptible for both PTSD and suicide attempt because they have a higher instance of becoming a bystander.
The link between posttraumatic stress

Figure 4. A comparison of the deaths due to unintentional injury as a percentage of total death across racial groups in the United States. Data was obtained from (National Center for Health Statistics, 2016).

Figure 5. A comparison of the deaths due to unintentional injury as a rate per population density across racial groups in the United States. Graph was adapted from information obtained from (Borse et al., 2008).

Child Maltreatment. Biologically, child abuse has been reported to have a significant impact on epigenetic modification, and thus, vulnerability to health.
disparities (Brockie et al, 2013). As stated previously, chronic stress has been identified as a risk factor for the development of PTSD. It has been reported that the most common stressor encountered by children living on reservations is in fact chronic, not acute (Brockie et al, 2013). Such an exposure puts children at risk for hyperactivation of the stress response, which in turn can lead to dysfunction of the system.

Literature suggests that although childhood maltreatment and PTSD work independently of one another for risk of suicide attempt, in combination, these factors significantly increase the likelihood of suicide attempt (Panagioti et al, 2009). Not only is childhood maltreatment a risk factor for the development of PTSD, but when combined with a previous diagnosis of PTSD, it is a risk for developing further morbidity. Therefore, children who are exposed to continual traumas may not only be at an increased risk to develop PTSD, but to also develop symptoms associated with suicide ideation.

**Maltreatment & PTSD.** A study conducted within a southwestern Native American population found that one form of maltreatment, sexual abuse, is positively correlated with the development of PTSD in female children (Robin et al, 1997). This report found that females were three times more likely to be the recipients of sexual abuse than males, and that family or close friends are most often the perpetrators. Furthermore, a separate study on two separate Native American tribes found that both childhood physical and sexual abuse increased the odds of the development of PTSD (Libby et al, 2005). Multiple traumas and sexual abuse have been linked to PTSD (Ehlers et al, 2013). A study conducted by the National Institute of Justice found that 56.1% of
AI/AN women and 27.5% of AI/AN men have been victims of sexual violence at some point in their life (Rosay, 2016). Given the increased rates of childhood trauma, mixed with a greater susceptibility for the development of PTSD, AI/AN children may be at a significantly higher risk of developing the disorder.

**Maltreatment & Suicide.** The U.S. Department of Health and Human Services defines child maltreatment as the following, “Any recent act or failure to act on the part of a parent or caretaker which results in death, serious physical or emotional harm, sexual abuse or exploitation; or an act or failure to act, which presents an imminent risk of serious harm” (U.S. Department of Health and Human Services, 2016). A national survey on child maltreatment in the United States found that AI/AN children were victims at a rate of 13.4 per 1,000 children (U.S. Department of Health and Human Services, 2016). This is the second highest rate in the nation, behind African American children, and about 1.5 times greater than the national average. In Montana, a state that contains seven Indian reservations and one additional state recognized tribe, child maltreatment in Native American populations exists at a rate of 10.5 victims per 1,000 children. This compares to a statewide rate of 5.3 children per 1,000 in that same year (U.S. Department of Health and Human Services, 2016).

Given that maltreatment of Native youth in Montana is almost double that of the state average, it could be expected that the suicide rate would also be higher in that population. A Youth Risk Behavior survey of Montana students found that 16.2% of Native students had attempted suicide at least once before the survey. Only 6.5% of all Montana students had made an attempt (Rosston, 2012). Though there exists no causal
evidence, Native American youth throughout the state of Montana show some of the highest rates of both child maltreatment and suicide attempt (Rosston, n.d.; U.S. Department of Health and Human Services, 2016). To back both of these rates, studies have shown that all forms of child maltreatment are connected to suicide attempt in adolescents, with sexual and emotional abuse being the most important forms (Miller et al, 2013). If such a connection were made, efforts to prevent maltreatment as well as efforts to treat victims would result in the lowering of youth suicide rates. If established, such an intervention would be one of many routes toward suicide prevention.

**Discussion**

*The Distinction Between PTSD and Major Depression as a Risk Factor.* Current research on the connectedness of suicide with other psychological disorders has found both PTSD and major depressive disorder to have a significant impact on suicidal ideation (Gradus et al, 2010; Panagioti et al, 2009). Furthermore, these two morbidities are commonly found together, and, operating simultaneously, cause a severe increase in the risk of suicide ideation and attempt. As with PTSD, one route of the development of depression is from traumatic experience (U.S. Department of Veterans Affairs, 2015). What separates the illnesses is the manifested symptoms. Whereas PTSD is commonly related to an inability to properly move on from a traumatic event, depression is manifested in a loss of interest in the activities of daily life. It is easy to see how the persistent psychological struggle with PTSD symptomatology may give rise to a depressed mental state. Numerous studies have revealed the significant comorbidity between PTSD and major depression (Breslau et al, 2000; Kilpatrick et al, 2003; National
Collaborating Centre for Mental Health, 2005). Breslau et al (2000) found that there exists no significantly distinct vulnerabilities or exposures for PTSD as compared to major depression. Instead, the study found that a patient with pre-existing major depression was two times more likely to have been exposed to traumatic events, and were three times more likely to develop PTSD. Similarly, patients with a prior diagnosis of PTSD were about three times more likely to develop major depression. A study conducted on adolescents by Kilpatrick et al (2003) found that 75% of adolescent PTSD cases also had a comorbidity. Of this group of adolescents, 29% of major depression cases met criteria for PTSD, and 62% of PTSD cases met criteria for major depression. Evidence suggests that PTSD and major depressive disorder are very likely to develop alongside and influence each other. This phenomenon may result due to similar risk factors for both diseases.

Given the relatedness between PTSD and depression, it is of significant importance to discover how their interactions affect suicidal behavior. Suicide may not be a direct effect of PTSD, but rather of its comorbidity with major depression. As both illnesses arise via interpersonal trauma (Kilpatrick et al, 2003), AI/AN youth that are exposed to trauma early in life are at a significant risk of developing not only PTSD, but also major depressive disorder. The reduced stress coping effects of PTSD mixed with the hopelessness associated with major depressive disorder put children at a greater amount of risk when faced with a highly stressful environment.

**Connecting Adverse Childhood Experiences (ACEs) and Epigenetics.** A new field in epigenetic research has set out to establish a connection between adverse childhood
experiences (ACEs) and epigenetic modification. If such experiences cause alterations to the genes in the HPA axis, such a change may result in psychopathy. As stated previously, over or under expression of hormones released by the HPA axis is connected to varying levels of stress coping ability and mental illness.

Physiologically, multiple traumas do not merely show a correlation with the development of PTSD, but also show a strong relationship to suicide attempt later in life. A separate study conducted on adverse childhood events (ACEs) looked at the prevalence of traumatic events and their effects in adults (Felitti et al, 1998). Similarly to the Deters (2006) study, the ACE study found that those who experienced four or more traumatic childhood events were 12 times more likely to attempt suicide than those who had no childhood traumas. This same group was also six times more likely to attempt suicide than those who had experienced one childhood trauma (Felitti et al, 1998). Furthermore, a more recent study on the effects of ACEs on Native American youth found that abuse and neglect, witnessing violence against one’s mother, symptoms of historical trauma, and discrimination were strongly associated with PTSD and suicide attempt (Brockie et al, 2015). These findings combine both classic risk factors of PTSD with a few that are unique to the AI/AN population: historical trauma and discrimination.

**Suicide and Post-Traumatic Stress Disorder.** Common risk factors, as well as the nature of the post-traumatic stress disease, suggest that suicide and PTSD may have a correlation. The astronomical rates of suicide among AI/AN populations may be partially explained by the development and struggle with PTSD within this group of individuals.
These populations are already at a high risk for a reduced stress coping ability, historical trauma, and a higher incidence of trauma experience at a young age. At a rate almost double the national average, Native American Youth experience a wide variety of traumatic events. These traumatic events, which range from exposure to violence to sexual molestation, have been shown to have lasting impacts on youth and their development later in life. It has been reported that AI/AN children experience a level of posttraumatic stress disorder that is consistent with that of Iraq and Afghanistan war veterans (Brave NoiseCat, 2015). This same rate is three times higher than the national average (U.S. Department of Justice, 2014). Such a high reported rate of PTSD is a clear indicator of trauma among these communities. A study conducted with Native youth enrolled in a program for substance abuse (N= 89) found an average of 4.1 lifetime traumas (Deters et al, 2006). In addition, this same study linked PTSD to several other comorbidities, one of these comorbidities being major depressive disorder. Being exposed to multiple traumatic events increases the chances of the youth developing symptomatology along the spectrum of posttraumatic stress disorder. Furthermore, once PTSD was developed in trauma exposed children, it has shown a high instance for the development of a comorbidity. A combination of PTSD and/or major depressive disorder may further lead children to a sense of despair and suicidal ideation.

Several studies have illuminated the connection between PTSD and suicide ideation as well as PTSD and completed suicide. These studies found significant evidence to suggest a correlation between the two morbidities. A national epidemiologic study found that PTSD alone was a risk factor for suicide attempt, and this
same risk became amplified when paired with a comorbidity (Nepon et al, 2010). Furthermore, another study explored this same relation, and found that PTSD, as opposed to all other anxiety disorders, was the only illness significantly related to suicide ideation (Sareen et al, 2005). A study conducted on the correlation between PTSD and completed suicide found that those with PTSD were 5.3 times more likely to commit suicide compared to those without PTSD (Gradus et al, 2010). Though these studies connect PTSD with suicide ideation and attempt, they are not a predictor of PTSD as a correlate of completed suicide. Though concrete biological causation has not yet been able to be drawn between the diagnosis of PTSD and the attempt or completion of suicide, much scientific evidence suggests that these two morbidities may be linked. PTSD symptomatology, along with its comorbidity of major depression, may lead to a reduced quality of life, and thus, to suicidal ideation.

Current Available Services. In the United States, AI/AN youth face the highest burden of suicide (Fig. 1) and the lowest amount of funding for health care services (Fig. 6). A significant barrier facing the proper treatment of PTSD in AI/AN youth is the lack of funding and understaffing of the Indian Health Service (Roubideaux, 2005). Though IHS is a government funded program, unlike Medicaid, it has not received an adequate amount of funding to keep up with medical inflation (Warne & Frizzell, 2014). This lack of funding has led to health disparity in many AI/AN populations. Furthermore, it is being discovered that AI/AN who seek health care tend to do so from a traditional healer, rather than from one practicing western medicine. Though western medicine is used to relieve acute symptoms of an ailment, traditional medicine is used to achieve
holistic healing and wellness (Grandbois, 2005). A study conducted across two separate Native American reservation communities found that those with comorbid disorders were much more likely to seek medical help than those with an anxiety or substance disorder alone. In addition, among this same group of help seekers, help was more commonly sought from traditional healers over western medical professionals (Beals et al, 2005b; Koithan & Farrell, 2010). Traditional healing practices (Buchwald et al, 2000) as well as cultural spirituality (Garroutte et al, 2003) are closely tied to an overall state of wellbeing in AI/AN peoples. As the health of AI/AN populations is closely linked to spirituality and cultural traditions, the community often takes a crucial role in the healing process. Singing, dancing, ceremonies, prayer, herbs, and manipulative therapies are a staple of the traditional healing process (Koithan & Farrell, 2010). Choosing traditional healers over western health professionals may be something that is tied to cultural history, or it may be a result of mistrust in the federal government (Warne & Frizzell, 2014).
Figure 6. A comparison of the expenditure per enrollee of various health programs, in blue, with the average national expenditure, in orange. The national health expenditure is defined as the total amount of money spent to obtain medical goods or services. Data was obtained from (Centers for Disease Control and Prevention, 2016; The Henry J. Kaiser Family Foundation, 2016; Indian Health Service, 2016b; National Public Radio, 2015)

Conclusion

Solving the crisis that stems from the youth suicide epidemic is not a quick fix, but a long and strenuous process that must come through changes brought forth in neighborhoods, communities, and society as a whole. Both PTSD and suicide ideation have been discovered to arise from multiple traumatic events. These morbidities are especially prevalent when these events occur during youth, a key time in neurological development. These traumas can either be personally experienced or witnessed as a bystander. Faced with a rate of childhood maltreatment that is double the national average, the burden of historical trauma, and heritable epigenetic traits, AI/AN youth
are at an abnormally high risk for developing both PTSD and suicidal ideation. This risk becomes even more dangerous when there exists a lack of support systems both at home and in the community at large. A strong support environment comes from a healthy community. To begin to solve the poverty and violence that plagues several reservations, the problem must first be confronted, dealt with in a healthy manner, and incorporated into a plan for moving forward. For indigenous youth, acknowledging and understanding the injustices of the American Indian and Alaska Native past are an important part of a cultural move forward (Wexler, 2009). A restoration of health comes through knowledge of and pride in one’s culture. Love and acceptance for one’s self comes about partially through the discovery of one’s cultural background.

A return to strength for Native youth will come about only if specific steps are taken to resolving the problem:

- An acknowledgement of the problem at hand.
- An effort to pinpoint the possible clinical manifestations of illness.
- A willingness to provide culturally sensitive and exceptional care for those suffering the ramifications of PTSD.

If youth PTSD is met with exceptional preventative and post developmental care, youth with a history of trauma will have the tools to combat and overcome suicidal ideation.

**Areas for Further Research**

Given the above evidence, there is reason to believe that PTSD may be the causative factor of the abnormally high suicide rate among AI/AN youth populations.
Though diagnosis of PTSD and suicide may be strongly correlated in Native youth as well as other trauma survivors, this literature review has not established causation. It is suggested that empirical scientific evidence be gathered between the variables of PTSD and suicide ideation. A discovery linking PTSD and suicide ideation would not only benefit the Native community, but all trauma survivors.

**Tribe Specific Research.** AI/AN populations are often lumped together when considered for demographic study. One must be extremely cautious when performing such an analysis as many Native tribes are vastly different from each other (Grandbois, 2005; Libby et al, 2008; Wahab & Olson, 2004). As stated by Grandbois (2005), “the differences in the backgrounds of two AI/AN individuals might be greater than the differences between two Europeans from dissimilar countries.” Differences in economic stability, infrastructure, access to healthcare, tribal beliefs on illness, and education can range vastly between reservation communities, and are a serious predictor of health. Given these differences, one must be aware that the health, culture, and beliefs of each tribe’s members may vary.
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