



**The intergenerational transference of  
Post-Traumatic Stress Disorder amongst  
children and grandchildren of Vietnam  
veterans in Australia: An argument for a  
genetic origin.  
Review of current literature.**

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**The intergenerational transference of Post-Traumatic Stress Disorder amongst children and grandchildren of Vietnam veterans in Australia: An argument for a genetic origin.  
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Where does Post-Traumatic Stress Disorder (PTSD) have its origins? Does it have a genetic basis, or is it a learned psychological response to a severe life-endangering experience? If PTSD has genetic origins, then this condition could be passed down from one generation to the next and put the offspring at risk for developing or acquiring related conditions. If PTSD is a learned condition from our environment, then it could be 'taught' to our children. In either case, there is an increasing awareness in the behavioural research community that more young people are being diagnosed with PTSD than before. This paper examines a current trend in recent research that proposes a radical, yet rational perspective.

With the increase in recent terrorist activity and conflict situations world wide, the risk that individuals may experience a severe or life-threatening trauma is likely to increase. The common subsequent behavioural dysfunctions manifest in a condition called post traumatic stress disorder (PTSD). Do environmental influences contribute more to the development of PTSD and related disorders in the offspring of parents with PTSD, or do genetic factors have a larger impact? The overwhelming majority of study into PTSD has been conducted on subjects who have directly acquired the condition through the experience of an intense trauma in their environment. Less research exists concerning parental PTSD and its contribution to an environment that develops behavioural anomalies in their children. Some researchers have ventured away from the conservative norm to investigate the possibility of a PTSD-prone personality (Stein et al: 2002). Even fewer researchers have investigated the influences of genetics in this condition, although the trend appears to be shifting. This paper will present recent findings of related studies as a review of available literature to support the assertion that PTSD can be genetically based, and therefore transferred intergenerationally to our offspring. Data based on Australian studies specific to this issue is very limited, therefore this paper will attempt to identify links in the available literature, mostly from abroad, to support the claim of a genetic influence. There is no intention to downplay or devalue the environmental and learned influences on PTSD, as these significantly influence the overall issues and outcomes.

### *What is PTSD?*

The latest version of The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) defines PTSD as an individual's symptomatic reactions following direct exposure, or witnessing or learning about an extreme traumatic stressor involving actual or threatened death or serious injury. The response must involve intense fear, helplessness or horror or disorganized or agitated behaviour (American Psychiatric Association (APA), 2000). Individuals with PTSD often suffer from symptoms that reinforce the trauma such as nightmares, hypervigilance, flashbacks, or even emotional numbing (Yehuda, 2004). The individual must re-experience intrusive distressing recollections of the event, experience illusions or hallucinations, intense psychological or physiological distress to stimuli that resembles an aspect of the traumatic event. The individual also persistently avoids thoughts, feelings, activities, places or people that arouse memories of the trauma. They may have lapses in memory for periods before and after the trauma and the trauma itself. They may describe feelings of detachment and diminished interest and participation in social events. They may even have difficulty sleeping, be irritable, have difficulty concentrating and have an exaggerated startle response. These symptoms must not have been present before the traumatic event occurred (APA, 2000).

There are numerous ways an individual can experience a life-threatening trauma. Such experiences include road traumas, rape, sexual and physical abuse, armed holdups, hostage situations, unexpected medical procedures, natural disasters, holocaust, war, or witnessing any of these. Quite often the onlooker to a trauma can experience higher levels of post-trauma issues than the individual who experienced it directly. This form of Secondary Trauma is frequently overlooked (Fullerton et al: 2001).

PTSD is not a new disorder. Written accounts of similar symptoms are documented in the American Civil War, when a PTSD-like disorder was known as "Da Costa's Syndrome." Accurate descriptions of posttraumatic stress symptoms exist in the medical literature on combat veterans of World War II and on Holocaust survivors. However, careful research and documentation of PTSD began in earnest after the Vietnam War. PTSD has subsequently been observed in all veteran populations including World War II, Korean, and Persian Gulf conflicts, and in United Nations peacekeeping forces. Similar findings of PTSD in military veterans exist in other countries (National Centre for Posttraumatic Stress Disorder (NCPTSD), 2004).

Over the years, this condition has had many names, such as, "soldiers heart", "shell shock", "war neurosis", "combat fatigue", "combat stress reaction" and most recently, "gulf war syndrome". In some cases, the condition does not manifest for several months, or even years after the trauma occurred (National Centre for War-Related Post Traumatic Stress Disorder: 1999).

### *Environmental influences.*

Besides developing the disorder through direct exposure to a traumatic event, PTSD can be acquired through the family environment (Westerink & Giarratano, 1999). In this Australian study, the researchers concluded that the familial environment where one or more members had PTSD, impacted significantly on the likelihood that others within that environment would develop somatic symptoms, anxiety, insomnia, social dysfunction and depression. However, the children in this study showed no significant differences on measures of psychological distress and self-esteem than the control group. This article is typical of the research and findings from related Australian studies.

### *Epidemiology of PTSD in Australian Vietnam veterans.*

PTSD affects between 12-15% of Australian Vietnam veterans while a further 12-15% will experience at least some of the associated symptoms. 24-30% of returned service men and women can become diagnosed with PTSD. Around 50% of males and 25% of females in Australia with chronic PTSD report having a drug and/or alcohol problem (National Centre for War-Related Post-Traumatic Stress Disorder: 2000).

Australian soldiers were involved in the Vietnam conflict from 1962 to 1973 (National Centre for War-Related Post Traumatic Stress Disorder: 1999). During the course of the conflict, Australia sent at least two military contingencies.

Many servicemen and women did both tours. Most did not. Their children may have been conceived after their return from their first or second tour (post-trauma). If so, they may have genetically transferred their condition to their children, thereby encoding a prevalence for developing risky behaviours and depressive and/or anxiety type disorders, including PTSD.

*Suicide in Australian Vietnam veteran children.*

In support of this assertion, the Australian Institute of Health and Welfare (2000) conducted a study into suicide by Vietnam veterans' children and revealed that an alarming 92% of confirmed cases were born during or shortly after this period. The highest risk groups were between 15 and 29 years at the time of the study-1997. They confirmed that children of Vietnam veterans are more than three times more likely to suicide than the general Australian community and deaths of Vietnam veterans' children from illness and accident were also higher than expected. These patterns need to be considered for those children who were born after 1979 as they move into the age groups of increased risk of suicide. In 2004 we can predict this group to now be between ages 15 and 35 years. This information is reflected in the study on the morbidity of Vietnam Veterans by ACNeilson for the Department of Veterans' Affairs (DVA) (1998) which revealed that there was significant concern regarding leukemia, Wilms' tumor, cancer of the nervous system, congenital abnormalities and mortality rates in children of Australian Vietnam veterans. The recommendations strongly suggested urgent attention to address these issues.

From the information from both sources it can be suggested that children conceived during the risk periods may have genetically acquired difficulties in dealing with emotionally stressful events. These difficulties will be discussed in more detail later in the paper.

This article will discuss recent research and present the hypothesis that PTSD can have a genetic origin. If a valid case is to be presented, then a specific time frame must be identified for the possible genetic transference of PTSD from parent/s to offspring. This would be the period of conception.

To illustrate this argument further we identified two distinct classifying periods for conception that impact significantly on whether the offspring will inherit the genetic predisposition to PTSD. These are the post-trauma and pre-trauma periods. The investigation of available literature reveals no previous research has been conducted on the aspect of conception timing for the acquisition of PTSD. The following sections will provide evidence for a reason to commence a study in this area.

*Post-trauma.*

In our first scenario, a man or woman serves in the Vietnam conflict and experiences a life-threatening trauma. In its most brief form, the onset of PTSD occurs as follows: The individual's brain is flooded with corticotrophic steroids. Their central brain structures responsible for emotional and hormone regulation and management are physically and chemically altered. These regions shrink in size. Their adrenal cortex becomes hypersensitive. They exhibit symptoms of PTSD. (Stein et al: 2002. McKinney: 2002, Van der Kolk: 1994). The anatomical and neurological processes and transformations that occur with the onset of extreme trauma are far more complex than this.

With these significant structural and chemical alterations occurring with intense trauma, it may be plausible to suggest that the same mutative effects of the corticotrophic steroids also affect the individual's reproductive system. It is well documented that PTSD sufferers have a reduced libido than the general population without PTSD and men with PTSD have lower sperm counts than those without the condition (AIHW: 2000). It would be equally plausible to suggest the effects of PTSD may be transferred through altered mental and reproductive systems in the parent to their

offspring. Van der Kolk (1994) confirmed that the brain undergoes significant hormonal changes and adaptations during and after a severe traumatic event, and many of these hormones are utilized by the body for sexual reproduction and sexual development.

Children born post-trauma to parents with PTSD may then have a genetic source for any dysfunctional behaviours and difficulties in coping with life-stressors, and have a susceptibility to lifelong PTSD, as suggested by McKinney (2002). Even though the child themselves did not experience intense trauma, they may genetically acquire the symptoms from their parents. In effect, the symptoms still occur "post-trauma".

#### *Pre-Trauma.*

Our alternative scenario postulates a man and a woman with no familial or individual history of PTSD or comorbid conditions conceive a child together, then one or both parents experience a life-threatening trauma. It can be suggested their child may be at risk of acquiring behavioural variations due to their environment. The child may be "taught" PTSD by their parents. Due to the limited scope of this article, there will be no investigation in any particular detail, of the affects of intense trauma experienced by the expectant mother on the developing fetus. That can be left to other aspiring researchers.

#### *Parental PTSD.*

This idea is supported by Yehuda, Hallig and Grossman (2001) who identified parental trauma exposure and parental PTSD as being significant factors contributing to the experience of childhood trauma, and concluded that this experience may be an important factor in the transmission of PTSD from parent to child.

Yehuda, Halligan and Bierer (2001) investigated the relationship of parental PTSD and the depressive and anxiety disorders in their adult offspring. The findings demonstrate a significant specific association between parental PTSD and the occurrence of PTSD in offspring. The researchers further state in their study, that other risk factors for PTSD in children can include parental trauma exposure and parental PTSD, and concluded that the experience of childhood trauma may be an important factor in the transmission of PTSD from parent to child. They do not discuss whether this is a learned transfer or a genetic one.

#### *PTSD-prone personality*

If a PTSD prone personality does exist, then could these personality traits be heritable? Stein, et al (2002) found that

*"genetic influences on "PTSD proneness" might be mediated through personality traits (e.g., neuroticism) that would predispose an individual to assaultive trauma and subsequent PTSD"* (pg 7).

Simply stated, this theory suggests that an individual's genetic propensity toward neuroticism would lead them to experience more anger and irritability, making them (1) more likely to get into fights (thereby increasing the risk of experiencing assaultive traumata) and (2) more likely to become highly emotionally aroused as a result of experiencing such traumata, thereby increasing the risk for PTSD symptoms. This would tend to support recent research into criminal genetics and other personality disorders (Retz et al: 2003). The Australian Centre for Posttraumatic Mental Health (ACPMH) recently released a report that confirmed a personality with high levels of neuroticism and previous exposure to trauma can be a significant risk factor in the future development of PTSD (Australian Centre for Posttraumatic Mental Health, 2004). There appears to be a growing pool of research into the development of PTSD-type personality traits in the children of Vietnam veterans world-wide that suggests there may be a heritable personality trait to PTSD.

#### *PTSD in Australian children: Epidemiology*

In order to understand the effects of PTSD in children and grandchildren of Vietnam veterans in Australia, we first need to look at its prevalence in the veteran community. Data is currently unavailable pertaining specifically to the number of Australian Vietnam veteran offspring who report depressive or anxiety related disorders, although a very recent media release from the office of the Minister for Veteran Affairs stated the Australian Government would examine the feasibility of conducting a study of the health of the children of Australian Vietnam veterans (Department of Veterans Affairs, 2004).

In the following sections we will be discussing the genetic, psychological and physiological alterations that intense trauma has on the individual. Although this refers directly to the individual attributed with the condition, it emphasizes the probability of the intergenerational quality of PTSD, and if reason follows research, we can identify similar predictable behaviour patterns in the offspring of Vietnam veterans with PTSD.

#### *PTSD in Australian children: Symptomology*

The DSM-IV-TR is the most widely used and preferred diagnostic tool for mental health professionals in Australia (Rosenman, 2002). This publication states that children, repetitive play may occur where the trauma is acted out and frightening dreams may be experienced that have no recognizable content (APA, 2000). Young children may report separation anxiety or avoidance behaviours, a preoccupation with an object or words that may or may not be related to the trauma and even lose a developmental skill such as toilet training. School-aged children often experience a "time skew" where recall of the trauma loses consecutiveness in sequence. This is not typically seen in adults. Elements of the trauma may be exhibited through play, art or verbal expression. Adolescent behaviours begin to resemble adult symptoms including the strong desire to "rescue" a potential victim of trauma that is younger than themselves and viewed as helpless (NCPTSD, 2004).

#### *Genetic Evidence.*

Schiffmann (2003) reports in the Jewish Post of New York, that a genetic mutation of the Dopamine Transporter (DAT) has been positively linked to Post PTSD by doctors in the Psychiatry Department at Hadassah Medical Centre in Jerusalem. This report has been authenticated by Segman et al (2002). The DAT gene, which itself was identified in 1963, is responsible for transporting Dopamine within the nervous system. As discussed further in the article this study has confirmed the role of genetics in PTSD.

Dopamine is a specific organic compound that functions as a transmitter of electrical activity in specific regions of the brain. As a chemical messenger, dopamine is similar to adrenaline. Dopamine affects brain processes that control movement, emotional response, and ability to experience pleasure and pain. Regulation of dopamine plays a crucial role in our mental and physical health. Dopamine appears to act as an inhibitor and is suggested to be important in motor control systems and whose actions have been implicated in various processes including sleep, pain and the psychobiology of various affective disorders, specifically depression and bipolar disorder (Ericksen: 2000).

Dopamine has also been found to have significant implications on Attention Deficit Hyperactivity Disorder (ADHD) (Barr: 2000), alcoholism (Johnson: 1996; Laine: 2001) and depression (Nierenberg, Dougherty & Rosenbaum: 1998). Studies have shown these conditions to be genetically linked to PTSD (Tarrier & Gregg, 2004; Donnelly, 2003; Xian et al., 2000).

Further recent studies have confirmed the existence of a gene responsible for PTSD. Comings, Muhleman and Gysin (1996) identified and confirmed the dopamine D2 receptor (DRD2) gene as having a significant affect on the severity and period of susceptibility to PTSD in Vietnam veterans. They concluded that a significant proportion of subjects carried a genetic anomaly called the D2A1 allele and this determines the level and effectiveness of the D2 receptor, and the likelihood of PTSD onset.

Stein, Jang, Taylor, Vernon and Livesley (2002) concluded that PTSD symptoms are moderately heritable in combat veterans after both combat related and non-combat trauma. Moreover, many of the same genes that influence exposure to trauma appeared to influence susceptibility to PTSD. This is interesting for the PTSD-prone personality theorists.

Bessel A. van der Kolk, MD of the Harvard Medical School in Boston, USA has produced a comprehensive article outlining the significant findings with hormonal changes and adaptations the brain experiences during and after a severe traumatic event. These are the same hormones utilized by the body for sexual reproduction and sexual development. In one section of the article, Van der Kolk (1994) states that there is a rising concern towards recognizing that a range of neurobiological abnormalities are beginning to be identified in the younger population.

Several studies have been conducted that investigate the genetic relationships between PTSD and other disorders such as generalized anxiety disorder and panic disorder (Chantarujikapong et al., 2001; Scherrer et al., 2000), borderline personality disorder (Golier et al., 2003) and alcohol and drug dependence (Chantarujikapong et al., 2000; Xian et al., 2000). Each study found a strong statistical relationship behind the genetics for each disorder and concluded that, not only was each of the disorders manifested by PTSD in the individual, but was causal in their offspring.

Further supportive research comes from a conference held by prominent academics in the field of trauma. From the 13<sup>th</sup> – 15<sup>th</sup> of December 2002, the University of Los Angeles, California, held a conference on Trauma, Culture and The Brain. This conference discussed the latest in the neurobiology and treatment of PTSD. However, of particular interest was a paper presented by on the intergenerational effects of trauma from a psychological and neurobiological perspective (Yehuda, 2002). This paper demonstrated that children of trauma survivors with PTSD constitute a high risk group for developing or genetically acquiring the condition because they have a greater prevalence of lifetime PTSD than others. It also suggest these children may perpetuate the condition to their children and that children of trauma survivors show a greater prevalence of mood and other anxiety disorders and comorbid conditions to PTSD, indicating the presence of genetic factors. Furthermore, it found there are significant interactions among variables associated with risk for PTSD and cortisol levels.

#### *Physical Evidence.*

There is also a rising awareness in the behavioural research community that the physical structures within the brain are significantly altered shortly after a life-threatening trauma. One such article discusses the discovery of shrinkages in the hippocampus and amygdala of sufferers of PTSD (Stein et al: 1999). They found that the smaller these brain regions became after the trauma, the more severe the PTSD symptoms. These brain structures are responsible for monitoring emotional responses.

There is a growing pool of current research on this issue that is becoming more succinct. McKinney (2002) discusses an interview with Dr. Mark W. Gilbertson of the Veterans Administration Medical Center in Manchester, New Hampshire, England, concerning a recent study relating sufferers of PTSD with smaller hippocampus sizes. The hippocampus is a curved, sea-horse-shaped brain structure that plays a complex role in managing our behaviour. As part of the limbic system (*larger group of brain structures that regulate and manage emotional responses that lead to behaviours*) it is intimately involved in emotion and motivation and hence in learning and, interestingly, some recent research implicates the formation in the establishment of long-term memory. They discovered, among Vietnam veterans, the hippocampus tended to be smaller in the men who had experienced PTSD upon returning home than in those who did not develop the disorder. What's more, the size of the hippocampus corresponded with the severity of PTSD, with the region being smallest in men with the worst cases of the disorder. These findings mirror those of Stein et al (1999).

In further support of McKinney, Stein and his colleagues, Lubit (2002) confirmed that a range of significant structural abnormalities and changes occur in the physical brain with the onset of PTSD. These findings were:

- Hippocampal volume is smaller in individuals with PTSD.
- Areas of the brain that are involved in threat perception, such as the amygdala, have altered metabolism in adult trauma survivors with PTSD.
- Activity of the anterior cingulate which is an area of the brain that inhibits the amygdala and other brain regions involved in the fear response, is decreased in people with PTSD.
- Basal cortisol levels are low.
- Concentration of glucocorticoid receptors and, possibly, glucocorticoid receptor activity in the hippocampus are increased.

Further supportive research comes from Joseph's (1998) who found that trauma affects not only memory, but causes deterioration in areas of the physical brain. Joseph found that, with the onslaught of corticotrophic steroids, the hippocampal pyramid cells, temporal lobe and amygdala regions actually decreased in physical size. These regions are largely responsible for emotional control and reactions to stress.

Recently, the ACPMH published a report confirming a significant risk factor for biologically acquiring PTSD is reduced cortisol levels (ACPMH, 2004). Cortisol is an inhibitory steroid neurotransmitter that calms the individual after a response to stress. If an individual has reduced levels of this steroid, then it will be more difficult for them to return to a "normal" resting state of calmness. Research has recently suggested that with repeated exposure to traumatic situations, this reduced cortisol level leads to a lifetime prevalence of adverse reactions to minimal stressors (Delahanty, Nugent, Christopher & Walsh, 2004).

Yehuda [et al] (2000) investigated the relationship between PTSD onset and the level of cortisol in their brains. Low cortisol levels were significantly associated with both PTSD in parents and lifetime PTSD in their children, suggesting that low cortisol levels in PTSD may constitute a vulnerability marker related to the intergenerational affects of PTSD and strongly supporting the hypothesis that PTSD is genetically based and therefore heritable.

#### *Gender Differences.*

Are daughters and grand-daughters of Australian Vietnam veterans more likely to inherit PTSD or PTSD-type behaviours from their parents? The research suggests they are, indicating females are more at risk of developing PTSD both through direct personal exposure and genetic influences.

In the context of personal exposure, Carter-Snell and Hegadoren (2003) found their studies reported women were more likely to report symptoms of stress-related disorders such as acute stress disorder, post-traumatic stress disorder, and major depressive disorder than men. In an Australian study on PTSD coping by Bates, Trajstman & Jackson (2004) it was found that females coped better than men and endorsed more positive change.

Stein, Walker & Ford (2000) found that females were found to be at significantly increased risk for PTSD following exposure to serious trauma despite lower exposure, even when sexual trauma - which predominates in women - was excluded. This would support the hypothesis that daughters are more likely to exhibit PTSD symptoms than sons. This finding is supported by Fullerton (et al) who in their abstract categorically stated that women have higher rates of posttraumatic stress disorder (PTSD) than men and that women were more at risk of acquiring PTSD.

In support of this postulation, a study by Breslau, Davis, Andreski, Peterson and Schultz (1997) found that posttraumatic stress disorder is more likely to develop in general population females than males after exposure to a traumatic event. A subsequent study by Breslau (2002) supported this claim by further examining the gender differences in exposure to traumatic events and in the

emergence of PTSD following exposure in the general population. In both studies, they found that lifetime prevalence of traumatic events was slightly higher in men than in women, yet the risk for PTSD following traumatic experiences was twofold higher in women than in men.

One study by Rosenman (2002), however, did not support conventional models of female susceptibility to post traumatic stress disorder due to combat. Using the Australian National Survey of Mental Health and Wellbeing, Rosenman factored out the gender differences as a vulnerability risk, asserting that females developed PTSD from rape and sexual molestation, while Bryant & Harvey (2003) found 38% of PTSD in women is causal to motor vehicle accidents. Interestingly, Rosenman found that clinicians who used the ICD-10 as a diagnostic tool diagnosed twice as many women with PTSD than clinicians using the DSM-IV. This illustrates a discrepancy between the two prominent diagnostic tools and may explain some epidemiological anomalies. The DSM-IV is more restrictive and used more commonly in Australia.

In the context of a genetic origin, and in light of the significant hormonal steroid levels that cause structural and neurological alterations, the findings that women are more likely to suffer from PTSD after an intense trauma than men, raises the questions of whether the transference of genetic information can occur whilst the mother is still pregnant with her child, even if the both parents were PTSD-free prior to conception, and whether women are more likely to transfer PTSD to their children than men.

The DSM-IV-TR (APA, 2000) describes the gender prevalence for genetically related disorders to PTSD as being significantly higher for women than men. Major Depressive Disorder is 1.5-3 times more common among children of parents with PTSD with women reporting more than twice the prevalence rates. Dysthemic Disorder is two to three times more likely in women and the effects of Bipolar 1 Disorder are more severe in women, particularly in postpartum periods. Panic Disorder is diagnosed twice as often in women and has an 800% chance of being inherited by children of sufferers and PTSD itself has a strong heritable component, especially if a history of depression in parents exists. Even Premenstrual Dysphoric Disorder is genetically implicated in PTSD, which is specifically a female condition (Perkonig A, Yonkers KA, Pfister H, Lieb R & Wittchen HU, 2004).

*Intergenerational transference: Support from twin studies.*

PTSD is highly comorbid with a range of mental disorders (Koenen et al., 2003). Research into the genetic transmission of PTSD benefits from studies conducted on twins of the Vietnam conflict era, where one went to Vietnam and the other did not, to predict whether PTSD is due to genetic or environmental factors. Stein et al (2002) finds support for genetic influences on PTSD symptoms from studies on American twins. The researchers investigated whether one twin possessed a precursor to PTSD as a personality trait before their exposure to combat trauma that their twin did not have. They found that the psychological characteristic that predisposes an individual to panic and anxiety sensitivity might be inherited, instead of the more common studies of physiologic characteristics, and concluded that a significant psychological risk factor for the development of panic disorder via genetics exists.

Other influences that affect the heritability of PTSD have been researched on twins of this era. Chantarujikapong and colleagues (2000) found that alcohol and drug dependence accounted for a 15.3% genetic contribution to PTSD and a further 20% specific to PTSD itself. Drug and alcohol dependence was found to be both causal and consequential of PTSD. In a similar study Chantarujikapong and colleagues (2001) utilized the same subject group to reveal that the genetics behind generalized anxiety disorder and panic disorder contributed 21.3% to PTSD and 13.6% specific to PTSD itself. The same group of researchers, utilizing the same subject group estimated the genetic and environmental contributions specific and common to generalized anxiety disorder and panic disorder and found that the lifetime co-occurrence of these disorders were best explained by a model that did not include family environmental factors. Instead, they

concluded that the acquisition of these conditions was best explained by genetics. No female twin pairs were included in these studies.

True and colleagues (1993) found through quantitative genetic analysis, that inheritance has a substantial influence on the liability for all symptoms relating to PTSD.

Other twin studies examined the physical changes in brain structure between twins. McKinney (2003) found that traumatic stress caused changes in the size of hippocampal regions and amygdale size in the twin who experienced the trauma while serving in the Vietnam conflict, but not for the twin who remained at home. These studies demonstrate a genetic link in twins between PTSD and related behaviours and disorders.

#### *Further research*

The idea that PTSD related behaviours can be intergenerationally transferred through a complex series of genetic alterations is an interesting and somewhat controversial topic of discussion and research. The overwhelming majority of research has been conducted in the United States, where their federal funding supports such research. A large pool of research has also been conducted in the UK. Such resources are scarce in Australia. There is much that still needs to be done in this area.

This issue would benefit from organized empirically based studies. Researchers have found alterations in function and size in specific brain structures in size and function (Yang, Wu, Hsu & Ker, 2004; McKinney, 2003; Lubit, 2002) of PTSD sufferers using MRI scans. Similar tests could be conducted on the children and grandchildren of Australian Vietnam veterans to determine whether they have inherited the physical characteristics of PTSD. Urine samples tests for cortisol levels could be conducted to reflect the findings of Delanty et al., (2004) and Yehuda (2001) showing that PTSD offspring have higher urinary cortisol levels than non-PTSD offspring.

Additionally, Australian studies could be initiated that investigates the timing of conception and its impact on the acquisition of PTSD.

#### **Discussion.**

Can PTSD have a biological basis, or is it purely a learned individual response to a severe life-endangering experience? What is the likelihood of PTSD being transferred genetically from one generation to the next? The research discussed above seems to support the argument for the affirmative. There seems to be a volume of current and recent research that is suggesting a genetic origin for PTSD. If a flood of corticotrophic hormones can produce a mutation of a specific dopamine receptor in the DNA as confirmed by Comings et al., (1996) and Schiffman, (2003) and sexual hormonal changes and adaptations in the brain and during and after a severe traumatic event, and a resultant decrease in the physical mass of significant brain structures, it would be reasonable to predict a significant possibility of this condition being genetically transferred to the offspring. Much of the research presented supports this assertion.

Additionally, if the children of a PTSD sufferer are identified as strong potentials for heritable PTSD, then we may suggest that their children in succession would also inherit this condition. If this holds true, then PTSD and PTSD-type symptoms could exist in the descendants of not only Vietnam veterans in Australia, but in decedents of all victims of intense trauma, from all the major wars and conflicts to sexual, psychological, physical and road traumas.

Dopamine has been identified as having particular importance in this issue. Its role in this paper relates to the probability that the offspring of Vietnam veterans with PTSD may inherit this dopamine imbalance and limited reuptake potential. If the sufferer of PTSD has a physically smaller hippocampal region following the onset of severe trauma, and the subsequent dopaminergic imbalance, then it could be plausible to predict specific behavioural abnormalities or patterns of behaviour, such as ADHD, alcoholism and depression in their children.

There appears to be a shifting trend of research that leans towards investigating the influence of genetics on this condition. This paper presents only a small proportion of the supportive research found that identifies PTSD as a condition that can be heritable and causes physical structures in the brain to change, not only in size, but in structure, performance and composition.

*Limitations.*

As comparable Australian data and research could be found, much of the discussion has been limited to American research. This forms the precursor for large volumes of research to be conducted on the Australian experience of this global condition. Given the growing body of research suggesting that there may be a genetic component to PTSD, it is important that researchers and practitioners be aware of the differences between the two and the affects that are manifested. This will also critically affect the way the individual is treated in both the short and long term.

This paper has reviewed and discussed much of the latest literature concerning the genetic influence of intergenerational PTSD in Australian families where at least one of the family members served in the Vietnam conflict. There are numerous sources of PTSD. If the scope of research were to be opened to all sufferers of all causes of PTSD, then it would become far too complex and large for a single researcher to manage. This may be an area rich in opportunity for other aspiring researchers.

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