The socialization of inherited aspects of trauma: secondary behaviours in the lived experience and identity of children of Vietnam veterans with clinical PTSD in a microsocial Australian context.

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Abstract.

There is little known about the heritability of Post Traumatic Stress Disorder (PTSD) among the children and grandchildren of Vietnam veterans in Australia. There is speculation and investigation by the several prominent perspectives to claim that they alone can explain how this complex issue is transferred from one generation to the next. However, they fail to consider the secondary behaviours that are embedded in the interaction cues and rituals existing in the reclusive contexts of families with a history of PTSD, and the significant roles these play in its heritability. Although each of these prominent perspectives make valuable contributions, none are able to adequately account for these secondary behaviours that forge identity and meaning for these children and grandchildren. While presenting some of the major research from these perspectives, this thesis proposes a point of intersection for them to present these secondary behaviours and asserts their inclusion in developing and implementing effective and appropriate management strategies.

Introduction.

Recent research suggests that Posttraumatic Stress Disorder (PTSD) may be heritable (Galea, Acierno, Ruggiero, Resnick, Tracy, & Kilpatrick, 2006) providing a multi-generational social management concern which impacts on several social and cultural institutions. Several modes of heritability have been proposed that assert absolution, but fail to consider the secondary behaviours that arise as a consequence of the symptoms, but are not directly due to the condition or impairment itself (Adkins, Smith, Barnett & Grant, 2006) such as self-harming as a result of depression, anxiety and guilt where individuals are experiencing recurring, intrusive, distressing memories of the traumatic event.

This paper sets up the premise of how interactional cues and rituals in the family environment influence the development of identity and concept of self in children and grandchildren of Vietnam veterans in Australia with clinical PTSD that give meaning to its experience in everyday interactions. What therefore is needed, is a study that makes a contribution to the understanding of the experience of PTSD through an investigation of the secondary behaviours that significantly influence the heritability of this condition.

This document first outlines the literature on PTSD as it is understood from the dominant perspectives (neuroanatomical, genetic, epigenetic and psychological) that comprise the medical model of PTSD. The document will then review the dominant literature that comprises the social model of PTSD. This review argues that while each of these perspectives provides valuable insights into various aspects of PTSD, none of them

are able to account adequately for what has come to be known as secondary behaviours. The study of these behaviours is critical for informing management strategies in relation to this disorder because they are the site at which the physical and psychological aspects of this condition intersect with everyday life.

The document then turns to an outline of research questions and strategies for data selection, collection and analysis suited to exploring the experience of PTSD in the microcosm of intergenerational family life.

What is PTSD?

Post Traumatic Stress Disorder, or PTSD, is a condition that manifests as a result of a direct or indirect exposure to an actual or perceived serious threat to one's safety and survival. It specifically relates to the loss of any or all elements of control for the individual while exposed to this threat (Clayton, 2004). PTSD has become a global phenomenon, affecting many families in almost every country in the world. The incidence of traumatic events seems to be ever increasing with more people suffering its effects on a daily basis. This condition is viewed by current researchers and mental health professionals as an emotional disability that can have intergenerational influences (Yehuda, 2002). PTSD is experienced by an overwhelming majority of survivors of all traumatic events globally (O'Brien, 2004). These psychologically traumatising events include sexual assault, automotive accidents, terrorism, armed hold-up, holocaust, war, and even workplace harassment. PTSD has existed for generations and undergone several definitions due to its interaction with a changing society (Ewing, 2005).

The Diagnostic Criteria.

The latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), the most widely used and preferred diagnostic tool for mental health professionals in Australia (Rosenman, 2002), 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 (American Psychiatric Association (APA), 2000). To further meet the criteria for PTSD, the response must involve intense fear, helplessness or horror or disorganized or agitated behaviour (APA, 2000). Sufferers often report symptoms that reinforce the trauma and further traumatize the individual, such as hypervigilance, flashbacks, nightmares and emotional numbing (Yehuda, Golier, Halligan, & Harvey, 2004). Other symptoms include memory impairments, irritability, sleep disturbances, distress, hallucinations and avoidance. Similar symptoms are often reported

in individuals who witnessed the event, or heard about it, or saw it on television or read about it in the paper. Evidence suggests that PTSD may be heritable in the first generation offspring of sufferers (APA, 2000). Many researchers have claimed that first and second generation offspring can inherit the same psychiatric condition as their parents, forcing the DSM-IV-TR to mention heritability in several diagnoses including PTSD (American Psychiatric Association, 2006). Indeed, studies have shown children born into families with a history of PTSD have high risk of developing emotional conditions (Yehuda, Halligan, & Grossman, 2001). This “PTSD-Prone personality” (Stein et al., 2002) places the child/grandchild at significant risk of exaggerated social responses to “normal” stress. The corticotropic steroids that flood our HPA axis and limbic system as a result of a severe stressor are considered toxic to our calm, homeostatic stasis (Meaney et al., 1996) and the effects may span several generations.

There is a vast amount of research on PTSD. The identification and treatment of this pathology has been extensively researched and discussed from the paradigms of neuroanatomy, genetics, psychology and sociology. There is not much the medical model doesn’t know about PTSD. Its descriptive symptomology is well documented and evidence for effectiveness of treatment is well established.

Primary Behaviours.

The primary behaviours are the immediate action responses to the trauma. They are the obvious behaviours and are frequently the only ones studied and treated. They form the diagnostic criteria for the condition and hence the range of mental health policies and treatment approaches (Green, 2000). Primary behaviours would therefore be the accepted identifiers of the condition and the ones that are recognised by practitioners and general society to apply labels and stigma. As there exists a range of varying approaches and treatment strategies for PTSD, there would also exist a range of interpretations of these primary behaviours. As described earlier, the definition and description of the primary symptoms have changed over time, as society has changed. This suggests PTSD is a social condition, not just an individual one. It also suggests that there are neglected or hidden social and intergenerational influences involved than simply the primary behaviours that have been the focus of study for decades. These neglected or hidden influences are the secondary behaviours of PTSD. The problems associated with this raise the issue of neglect of the secondary behaviours as a significant contributing factor to the awareness, acceptance, and heritability of PTSD.

Secondary Behaviours.

Secondary behaviours are also important in understanding the PTSD phenomenon, for it is these that both reinforce the primary behaviours, and give meaning to them (Adkins, 2007; Greene, 2000) as these are at the core of the response. Secondary behaviours are those that arise out of that primary response and are often overlooked or misinterpreted. It is the secondary behaviours that may be the root cause of the emergence and perpetuance of the primary behaviours. These must be studied to acquire a complete picture of the issue. Again, the DSM-IV-TR makes no mention of secondary behaviours, neither in broad, nor concise description. This is evidence of a short-circuit in the feedback loop: a lack of insight from data collected in the lived experience of PTSD and the impact of treatment. It begs to ask what influence do secondary behaviours have on the development of identity and self image in relation to PTSD and mental conditions in general.

A review of the available literature on this topic revealed no relevant applicable information. This demonstrates a significant gap in research on this vital component of identity development acquisition and is evidence of the need for this study to be conducted. It may be due to the emphasis on the medicalisation of PTSD, or other factors.

Medicalisation of PTSD.

The medicalisation of PTSD has forced a focus on the individual as the source, cause, treatment and responsibility for its symptoms (Joseph & Lindley, 2006) removing any portion of responsibility for its existence from social institutions that perpetuate it. As a recognised disability (Department of Veterans' Affairs, 2007), PTSD should become a social management concern, yet its perception through this medical model enforces a reductive approach controlling the individuals who experience its symptoms. This perspective is supported by Adkins et al (2006) who examines the experience of cognitive impairment in public spaces, such as shopping centres. It is understood that people with PTSD report similar impairments.

The medical treatment of PTSD creates issues in itself that are often overlooked or misunderstood as a consequence of neglect of the sociology of this condition. This thesis does not aim to devalue or avoid the medical model of PTSD as it is important it is recognised as the "coal-face" of practice, intervention and management. However, while these studies shed light on the ways in which psychological and physical aspects of the condition might influence behaviour, there is much less known about the way the disorder

is experienced in the contexts of everyday interactions within families and the role of these contexts in the experience of PTSD.

The Family as Focus.

As distinct from perspectives based on the medical model of PTSD, its symptoms and their heredity, this study of families experiencing PTSD seeks to identify the microsocial aspects of this condition in everyday contexts of interaction. The family itself is a social institution that may be a highly significant environment having an impact on the development of identity and sense of self, and of the personification of PTSD. The methods or processes that are used by families to transfer PTSD to subsequent generations through verbal and non-verbal language and microsocial cues and rituals is a field rich in research opportunity, yet significantly under-investigated. Therefore the understanding of these everyday experiences for the children of PTSD sufferers is the gap in literature, practice and research.

Position in the Literature.

Very little research has been undertaken on the micro-interactional aspects of disability; less for mental conditions, and almost none for PTSD itself. The search included several online databases, restricted access databases and personal interviews. There is no literature available on Australian family contexts. This leaves a significant and opportunity-rich gap in research concerning aspects of policy and management surrounding people's experience of this condition. These experiences can be used to assist practitioners develop highly effective and individual strategies. There is currently no unified system that collects and analyses data on the lived experiences of the intergenerational effects of PTSD to compliment the medical model and inform subsequent mental health management systems. The bulk of the existing literature focuses on identifying and controlling the negative social and individual effects of PTSD and on minimising the risk by controlling the physical environment of the individual with the condition, rather than trying to manipulating their environment to match their altered abilities. To fully benefit from this rapidly spreading phenomenon we need to understand its alternative contexts, its influence on family, community and society, and how best to manage it from the testimonial accounts of the children, grandchildren and families who have inherited the psychological legacy of an event they did not directly experience.

In doing so, this thesis discusses the research available in respect of the various theories of heritability of PTSD among children and grandchildren of Vietnam veterans in

Australia. It will collect stories of the lived experienced of the children of Vietnam veterans in Australia in their daily battles with inherited symptoms and discuss how the family context influences the experience of PTSD as an object of intellectual enquiry. There will be a distinct focus on the language used in families and the interactional cues and rituals (Goffman, 2003) that support the secondary behaviours inherent in its intergenerational transmission.

Language of Intergenerational PTSD.

This may be achieved by deliberately and purposefully selecting settings to illuminate the way in which context influences the experience of PTSD through the analysis of language. It has been established by Miller and Glassner (2004) that language shapes meanings and that the discourse of language can manifest and predict behaviour. This raises the question of how the language of PTSD itself is shaped, and creates meaning for those living with it. It employs a qualitative approach to data collection and analysis. Each of these is linked back to how we can understand the experience and context of the heritable microsocial elements of PTSD. This will inform medical models.

The document now turns to an overview of the dominant perspective in the literature on PTSD – the medical model – and how it pertains to the heritability of this misunderstood condition.

Medical Model Perspective.

Intergenerational Transmission of PTSD.

To understand how such a debilitating condition can be transferred to subsequent generations, we must first review what the medical model currently knows about it. Research suggests that the major contributors to the heritability of this condition are genetic factors (Kendler & Greenspan, 2006; Moffitt, Caspi, & Rutter, 2006; O'Brien, 2004; Schiffman, 2003): environmental factors, which includes parenting styles (Barry, Dunlap, Cotten, Lochman, & Wells, 2005; Chase-Lansdale, Wakschlag, & Brooks-Gunn, 1995; Tan-Roldan, 2005; Welberg & Seckl, 2001; Westerink & Giarratano, 1999; Yehuda, Halligan, & Bierer, 2001; Yehuda, Halligan, & Grossman, 2001): personality (Gallagher, 1996; Johnson, Vernon, Harris, & Jang, 2004); and socio-economic status (Jayakody & Stauffer, 2000; Lupien et al., 2005; Peach, 2005; Porter, Lawson, & Bigler, 2005; Wildes, 2005; World Health Organisation, 2001). These studies further support the wide range of genetically related conditions to PTSD that are becoming increasingly apparent in our young people.

Genetically Related Conditions.

The heritability of mental disorders has generated much discussion and even argument between mental health scientists and geneticists. There appears to be a growing body of research that argues there is a genetic link in many, if not most, psychiatric disorders. Some of these disorders that have identified genetic links to PTSD include Schizophrenia (Masters, 2005; Ridley, 2003), Bipolar Disorder (Durán, Serrato, & Virguez, 2005; Wildes, 2005), Depersonalisation Disorders (Simeon, 2004), and recently, depressive and anxiety disorders (Hasler, Drevets, Manji, & Charney, 2004; Scherrer et al., 2000). Additionally, conditions that manifest as disorders in children common in children have strongly suggested heritable links with PTSD that may be genetic. These include Conduct Disorder (Alexander & Pugh, 1996), Borderline Personality Disorder (Golier et al., , 2003), Attention Deficit-Hyperactivity Disorder (Brown, 2000; Hudziak, 2000; Spencer, Wilens, Biederman, Wozniak, & Crawford, 2000; Tannock, 2000), depression (Gatrtstein & Sheeber, 2004; Kaslow, Deering, & Ash, 1996) and anxiety (Chantarujikapong et al., 2001; Goodwin & Hamilton, 2003; Scherrer et al., 2000), a milieu of emotional disorders (Burger & Lang, 1998; Gatrtstein & Sheeber, 2004; Goodwin & Hamilton, 2003; Kaslow et al., 1996; Kidd & Ford, 1998) and even alcoholism (Johnson, 1996; Xian et al., 2000).

As this body of research grows to support assertions that PTSD is familial and heritable, it raises the problem of identifying the source or range of causes of PTSD in each person, and, most importantly, how to tease information out to inform practice, intervention and therapy. This body of research also illustrates the heritability of the neurological elements of PTSD, indicating that children of PTSD sufferers may be born with neurological indicators that limit their ability to process and manage stress in a socially appropriate manner.

Neuroanatomical Perspective.

What happens in the human brain with the onset of PTSD? How does this affect the individual's daily functioning? The predominant argument is that the tsunami of steroids that overwhelms the individual's neurological emotional management structures in the first few milliseconds after a significant stressor results in immediate alterations in the size, composition, and, therefore, function of limbic system structures (Bary, Carey, & McMahon, 2005; Beers & De Bellis, 2002; Bremner et al., 1995; McKinney, 2002). Yehuda (2004) asserts that the greater the reaction to trauma, the greater the reduction in size of these structures.

Limbic System.

It is well-established that the limbic system is largely responsible for the regulation of emotional input and output, and therefore the associated behaviours (Meaney et al., 1996; Palmer et al., 1999). Of particular interest and concern is the assertion that this dramatic change in structure and function of the brain's emotional management system can be heritable (Abdolmaleky, Thiagalingam, & Wilcox, 2005; Beatty, Heisel, Hall, Levine, & La France, 2002; Bjorklund, 2006; Hooper, 2006; Ridley, 2003).

Similar Neurological Findings in Children of Parents with PTSD.

Magnetic Resonance Imaging studies have reported neuroanatomical alterations in both adults with clinical PTSD and their children (Bremner et al., 1995; Porter et al., 2005; Van Voorhees & Scarpa, 2004; Villarreal & King, 2004). Beers & DeBellis (2002) found that children with PTSD mirrored the limitations in cognitive functioning that adults with PTSD reported. This may also include their parents. The specific functions where children with PTSD were significantly limited were attention, abstract reasoning and executive functioning, including learning and memory. Although there was only a small number of subjects used in Beers and DeBellis' study (n=14) it supports the assertion that there is a difference in the cognitive abilities of children with and without PTSD. In support, Joseph (1998) finds evidence of hippocampus injury due to emotional distress. Similarities in the size of specific brain structures between parents with PTSD and their children and grandchildren indicate probable heritable factors (Smith, 2005). Emerging literature is reporting other structural brain changes in people who experience clinical PTSD (Villarreal & King, 2004)) reporting decreased brain volumes, increased ventricle (cerebral fluid cavity) volumes and decreased area of the corpus callosum in children with PTSD (Bary et al., 2005; Van Voorhees & Scarpa, 2004). This raises the likelihood that people with clinical PTSD will develop more generalized brain atrophy or developmental abnormalities. Adults report changes in white matter, increased brain atrophy, smaller volume of the anterior cingulate, and smaller area of the corpus callosum and a range of other neurological changes (Porter et al., 2005; Van der Kolk, 1994). Concerning evidence strongly suggests that these physical and neurological changes, based on neurotransmitters, may be heritable.

Neurotransmitters.

Several authors assert that PTSD is also a consequence of dysfunctions in adequate levels of neurotransmitters (primarily dopamine and serotonin) and other glucocorticoids which are crucial for emotional management and memory processes. Recent studies indicate links between these neurotransmitters, PTSD and its intergenerational transmission (Abdolmaleky et al., 2005; Elzinga & Bremner, 2002; Herbert et al., 2006; Rowe et al., 2001). This research further indicates that a change in one brain structure will cause a change in all. Therefore, not only can these effects be heritable, they can affect multiple brain systems, including those responsible for memory processing.

Memory.

Most researchers agree that PTSD affects the individual's ability to adequately process and access memory (Azarian, Lipsitt, Miller, & Skriptchenko-Gregorian, 1999; Beers & De Bellis, 2002; Bracha, 2006; Bremner, 1999; Joseph, 1998; Lupien et al., 2005; McNally, Ristuccia, & Perlman, 2005; Moradi, Taghavi, Neshat-Doost, Yule, & Dalgleish, 2000; Nishith, Weaver, Resick, & Uhlmansiek, 1999; Peres, Mercante, & Nasello, 2005; Yehuda et al., 2004) which can also affect learning (Yehuda et al., 2004) and transform personalities (Bohart & Greening, 2001; Hamer & Copeland, 1998; Kendler, Gardner, & Prescott, 2003; Lensvelt-Mulders & Hettema, 2001; Linley & Joseph, 2004; Shakespeare-Finch, Gow, & Smith, 2005).

Evolutionary Response.

Gallagher (1996) suggests that the neurological adaptations produced by PTSD are an evolutionary response to fear by postulating that the human brain is constantly changing due to a constantly changing environment: one that is frequently being shaped by violence, horror and trauma. This perspective is shared by several other researchers (Bjorklund, 2006; Bracha, 2006; Clayton, 2003; Crews & McLachlan, 2006; Ridley, 2003) and may suggest that the children and grandchildren in families with histories with PTSD may be able to function and cope better when under duress than other children without familial histories of PTSD, giving them an evolutionary edge in and increasingly hostile environment.

The brain is perpetually adapting to its environment and its genetic signals. Palmer, et al, (1999) point out that, although the brain reaches around 90% of its structural and functional development by age five, cognitive development continues into young adulthood. Lupien, et al., (2005) claims that the damage to cognitive systems from traumatic stress can occur at any stage of life, not just during the critical phases, as the

brain is constantly developing. This is supported by Porter, et al., (2005) who states that environmental events have been found to influence the structure and function of the human brain, particularly during critical developmental stages.

These neurological and anatomical changes to the brain are both catastrophic and long-term. If these changes are heritable, there must be a genetic basis to them. The impact on subsequent generations of families with histories of psychological trauma would then be highly significant to this study.

Genetic Perspective.

Genetic theory is based on findings of Gregor Mendel regarding dominant and recessive genes. It postulates that the transmission of PTSD and many human conditions from one generation to the next is genetically predetermined (Ridley, 2003). This theory is deeply entrenched in scientific evidence and laboratory studies and is commonly perceived as “fact”. Observations and records of familial histories of human conditions have supported this theory even further. It is almost a ‘common understanding’ that if the parents had a particular trait or condition, their children would get it. Apparently there are over 1600 human psychological conditions that have genetic sources and more are being “confirmed” almost weekly (Bary, Carey & McMahon, 2005). These studies are based on decades of laboratory research and field studies. However, much of this research is based on ad hoc results in highly controlled and manipulated environments, questioning the social validity of their findings as Mendel originally based his theories on the hybridization of a garden pea (Mendel, 1865; Ridley, 2003). One ponders the association and relevance to human genomics.

Hasler, et al., (2004) discuss the mutual genetic markers associated with the heritability of depression, a condition frequently associated with PTSD and one half of the PTSD symptomology structure. The other half of the symptomology structure is anxiety. Kendler & Greenspan (2006) support the argument for a classic genetic basis of the intergenerational transmission of PTSD and report that several studies have indicated anxiety phenotypes in mice, and that these replicate human studies on the heritability of anxiety disorders. The authors examine the degree of similarity between genetic origins of mental and psychiatric conditions and the influences that genes can have on behaviour in simpler organisms such as the mouse and fruit fly. Although mice and fruit flies are far less complicated genetically than humans, it provides insight into the genetic complexities and multitude of possible variations in gene expression that causes individualistic qualities.

Twin studies.

Many studies that attempt to validate the impact of genetics on the transmission of mental conditions in families involve the study of twins, where one twin acquires the familial condition, and the other does not. While some studies investigate monozygotic (identical) twins others compare dizygotic (fraternal) twins. Those that focus on monozygotic comparisons claim that individual differences must be primarily a result of environmental influences since both twins share identical genetic information. Beatty, et al., (2002) present findings that compare heritability traits between both mono and dizygotic twin pairs and assert that up to 70% of personality traits are genetically based and not dependant on whether the twins were either monozygotic or dizygotic. This implies that there may be an influence upon the genes themselves that determines individuality. These assertions are supported by Chantarujikapong, et al., (2001), Stein, Jang, & Livesley (1999), Torgersen & Janson (2002), True, Rice, Eisen, Heath, Goldberg, Lyons, & Nowak (1993) and Kendler, et al., (2003). Villarreal & King (2004) examined the size of the hippocampus in monozygotic adult twin children of Vietnam veterans and found that both twins reported smaller hippocampal size, even though only one twin had directly experienced a significant trauma. This suggests the hippocampal anomaly was present before trauma exposure, indicating a strong genetically heritable component.

Personality traits.

There is some research that has extended the context of genetic heritability to personality styles and character traits. As PTSD produces significant and dramatic changes in personality and character, this research has particular influence on this study. Genetic studies into the transmission of personality traits support the possibility of genetic transmission of mental illness. Many such researchers examine the heritability of the Big Five personality traits (OCEAN - openness, conscientiousness, extraversion, agreeableness, and neuroticism) and assert that genetics play a significant part in describing and predicting our behaviour, and that the environment manages both the input and the outcome (Ilies, Gerhardt, & Le, 2004; Johnson, et al., 2004; Lensvelt-Mulders & Hettema, 2001; Torgersen & Janson, 2002). It seems that roughly 17% of the construction of leadership qualities is dependent on genetics (Ilies et al., 2004). Other studies also acknowledge that leadership traits have been shown to be heritable (Johnson, Vernon, Harris & Jang, 2004). These studies raise the question of whether other personality characteristics are also transmittable via genes, specifically those associated with PTSD. For example, research from (Johson, 1996) and Xian, et al., (2000) indicate a strong

genetic influence on alcoholism, and claim that traumatic stress can affect the expression of this gene. Indeed, this may be the case as Johnson (2006), Peach (2005) and Savarese, Suvak, King & King, (2001) all report there is a high degree of alcoholism in the Vietnam veteran community. It may be interesting to discover if a similar prevalence of alcoholism is present in the children and grandchildren of these Vietnam veterans, and if there are any gender-specific trends. Currently, no such data has been found to confirm or refute this, although Kendler and Greenspan (2006) report that hormones are implicated in the acquisition of personality and physical traits, suggesting that there may be some gender-specific genetic factors involved, but whether they refer to alcoholism remains a mystery.

Indeed, for pure geneticists, there are many mysteries surrounding the heritability of PTSD and other emotional disorders. There may be several reasons for this.

Gaps in Genetic Theory.

Even a theory as powerful as genetics, has its gaps and flaws. It fails to explain how a condition or trait can be initiated in one generation when there are no existing signs of it previously in that family's history. Some children are born into families with long histories of PTSD, yet do not themselves exhibit any of the problematic symptoms. This requires further explanation. Gregor Mendel proposed a complex system of recessive and dominant genes that were either dormant (but still present) or active, and the activating process was simply whether the trait came from the mother or the father (Mendel, 1865). Mendel's research has recently attracted extensive criticism (Fairbanks & Rytting, 2001; Hartl & Orel, 1992; Novitski, 2004; Shapiro, 1991). This criticism asserts that some children do not exhibit symptoms due to a redeeming characteristic called "resiliency" that protects them or toughens them against the adverse affects (Bleich, Gelkopf, Melamed, & Solomon, 2006; Carr, 2004; Cassitto, Fattorini, Gilioli, Rengo, & Gonik, 2003; Miller, 2003; Punamaki, Qouta, & El-Sarraj, 2001; Yehuda, 2004). However, this still leaves many unexplainable phenomena, such as how a trait or characteristic becomes evident in one generation – such as asthma – another apparent side effect of significant stress (Su, Becker, Kozyrskyj, & HayGlass, 2006) even though it has not existed in the family previously. If traditional genetics were absolute, all offspring would be identical, regardless. The fact that they do not, leaves the door wide open for alternate hypotheses that explain the individual differences between siblings, including identical (at least in the physical sense) twins. Even Mandelan Genetics, describing dominant and recessive genes, cannot fully account for the vast array of individual differences evident in every human.

Despite the strong supportive evidence and push from the scientific community to advocate genetic influences, Abdolmaleky et al., 2005 claim that no specific gene has been identified for any major disorder (such as schizophrenia and bipolar disorder). They state that there are several possible reasons for this failure, from poor study design to hidden (or undiscovered) genetic mechanisms.

The question of what created the condition in the progenitor (first person in the family to acquire the condition) is still unanswered. What is needed is a sound explanation of how these anomalies “appeared” and what their cause is. One theory that proposes to do just this is epigenetics.

Epigenetic Perspective.

Epigenetics suggests that what your parents, grandparents and even great-grandparents experienced (including diet) can affect your well-being (Paterson, 2006). It involves the study of whether genes get activated or not and offers the plausible suggestion that the environment can provide the stimulus to force changes in genetic expression (Paterson, 2006). It asserts that a protein or gene segment can be switched on or off by the presence or attachment of a specific chemical or hormone or even another gene (Ridley, 2003). Therefore we may all be genetically capable of developing any condition or characteristic possible in the entire human genome, depending on whether the protein or gene sequence responsible for that trait is turned on or off by our reaction to an environmental event. This is the case for PTSD as it leads to question the evolutionary purpose of such an adaptive response. It may be part of the intensive complex human survival strategy.

Foundations.

Hariri & Lewis (2006) and Fishbein (2000) assert that PTSD is not, in its entirety, genetically transmittable, but predispositions for developing PTSD and associated conditions are moderated by the effects of the steroids that can switch a gene or series of genes on or off that are responsible for the specific neurological dysfunctioning associated with PTSD. This theory commands respect and consideration in the scientific community as it is based on prominent, proven theories that incorporate and predate the Darwinian Theory of natural selection, August Weissman’s chromosome theory, Hugo de Vries’ mutation theory, the ancient theories of pangenesis and epigenesis (which may have spawned Epigenetics) and the most recent interpretation of Mendelain theory of

inheritance and chromosome theory by Theodore Boveri and Walter Sutton (Wu & Morris, 2001).

Human Genome Project.

Epigenetics has come of age. This may be a result of the frustration expressed in the mental health profession at the lack of rational and logical explanations for the intergenerational transmission of mental conditions. It may also be due to a significant fault in the famed Human Genome project: The Book of Life. This was an attempt to accurately map the human gene code. As described by Hariri& Lewis (2006), Paterson (2006) and Ridley (2003) this project was a global concept to describe and genetically map all races and creeds of the human race and create a gene code (much like a recipe) for every characteristic and condition of the 'typical' human. The original estimated genetic variations mounted over one hundred thousand. However, the biological scientists and geneticists were dismally disappointed to discover only around 28,000 individual sequences exist (Paterson, 2006): a far cry from their original estimate. A lot of questions were left unanswered from the Human Genome Project. However, it was simply a beginning. With a significantly reduced number of genetic variations apparent, the Human Genome Project stakeholders were at a loss to explain the millennia of mental conditions being transmitted from parent to child to grandchild. The questions raised by this significant margin in error of estimate opens many pathways for research and investigation. In the context of PTSD, this has incredible, yet plausible implications. The individual is adapting to a hostile environment to survive, and these adaptations are being passed down through the generations to ensure species survival. Humans both respond to changes in their environment, and create changes based on those adaptations, and this occurs at a genetic level. Hence, it may be that with modern dietary supplements we are having a direct impact on our genes that lead to obesity and behavioural disorders (Masters, 2005; Reading, 2002). We may not see the effects in this generation, but maybe the next and the next. Hamer and Copeland (1998) and Masuzaki, Yamamoto, Kenyon, Elmquist, Morton, Paterson, Shinyama, Sharp, Fleming, Mullins, Seckl, and Flier (2003) assert that our "take-away" culture is creating genetic anomalies that is resulting in obesity and diabetes epidemics in our children and grandchildren. Thus the lives and societies of these children and grandchildren will have to be constructed to accommodate these genetic changes.

Now the document turns to the psychological perspective of the heritability of PTSD as it attempts an explanation from the standpoint of the individual's interaction with their environment.

Psychological Perspective.

The psychological perspective is mostly concerned with the medical model of mental illness (O'Brien, 2004) and applies the reductive approach to its diagnosis and management. Although this paradigm acknowledges the influence of both genetic factors (based on twin and personality studies) and environmental factors on the acquisition of PTSD in the individual, it provides little explanation for its intergenerational heritability. The most prominent concept put forward by this perspective concerns the effects of modelling and conditioning of behaviours from parents to their children.

Parenting Styles.

Parenting styles play a crucial role in understanding how PTSD is transmitted within families. It may be that children born into families with histories of PTSD and its comorbidities, come with a predisposition or heightened risk of developing those conditions, should they be exposed to an environmental event that triggers the expression of the gene responsible for its development. O'Brien (2004) presents a description of these predisposition sensitivities by illustrating how the individual's tolerance for managing stressful information is reduced in individuals with PTSD. In effect they have a reduced threshold for stress. This reduction can occur as a direct result of a traumatic event, or the individual can be born with it. Either way, they cannot process the same amount of emotionally stressful information as their counterparts with no history of PTSD.

(Parent et al., 2005) assert that stress illnesses in parents are transferred to their children through inadequate parenting styles, and that the source of this transmission lies in the gene expression in brain regions that are known to regulate stress responses. This presents a dichotomy of dilemmas. On the one hand this adaptation may be highly effective and assist in ensuring survival of the individual under extreme stress, while also involving an increased risk for psychiatric and physical illness in society. This suggests a socially derived definition of mental illness and disorder, for it is the social environment that sets the standards for what is normal and acceptable.

Combined Approach.

Brutsaert and Parra (2006) and Moffit et al (2006) report that the majority of studies are currently asserting that a combination of genetic and environmental influences are responsible for the acquisition of psychological conditions. This majority advocates for a combined influence of the environment on our genes but hold on to traditional beliefs promoting genetics as the dominant paradigm (Kendler et al., 2003; Torgersen & Janson, 2002). These studies further point to the emerging evidence from epidemiological and neurological research that shows individual variations in response to stress is directly associated with pre-existing individual differences in cognition, temperament, psychophysiology, and other personality traits that are known to be under genetic influence. They further propose that environmental influences and genetic influences could be operating not only as one unified system, but as two independent systems that each operate in synchronicity to each other to contribute to a collective influence on our behaviour. Currently, we do not possess the technology nor knowledge to identify which genes affect which neurological pathway leading to a disorder.

The document now turns to a review of the social model and its approaches that places PTSD in the social context with emphasis on the social interactions that perpetuate and contribute to the heritability of this condition.

Social Model.

The social model takes an opposing perspective of the medical model in that it focuses the cause and consequences of mental illness on the social institutions and systems themselves (Lemay, 2006). It perceives all disability (aka impairment, or “difference”) as being the result of a mismatch of needs between persons and the systems and institutions that govern mainstream populations (Michailakis, (2003). The society in which one lives largely determines and defines problematic behaviour, and thus what is dysfunctional and inappropriate. Socio-economic influences are claimed to have a significant impact on the heritability of PTSD. The cohorts this is most prevalent in are single, unemployed mothers (Jayakody & Stauffer, 2000), youth (Juntunen & Wettersten, 2006; Lupien et al., 2005; Peach, 2005; Porter et al., 2005) and refugees (Ingleby, 2005; Pupavac, 2002).

Big Picture Analysis Neglects the “Experience”.

Many of the founding studies concerning the influence of society on behaviour were conducted at the institutional level. They examined the individual’s interaction with large social institutions and how the individual navigates and negotiates the rules of these

institutions in developing their social identity (Cahill, 1998; Fine, 1991; Kogan, 2002). However, they were very rigid in their approach and did not allow for other influences and manipulations (such as genetics and neurology). As Adkins, et al (2006) point out, in an effort to compensate for this neglect, studies that focus on PTSD using the social model often center on “the dynamics of societal exclusion at the expense of an examination of the lived experience” (Adkins, et al., 2006:5).

One piece of literature that does break new ground is by Galea, et al., (2006) who acknowledge the emergence of the molecular and genetic factors that predetermine risk for the acquisition of PTSD and further illustrate the influence of the social context in its cause and prognosis. However, Galea and colleagues remain at the social institutional level in their analysis, examining interactions between individuals and large social institutions. To get to the nitty-gritty (as it were) the issue of the heritability of PTSD in a social context must be examined from the lowest, most elementary level of social interaction in conjunction with the genetic, environmental and neuroanatomical paradigms. These are interactional cues and rituals.

This thesis builds on this perspective and approach by examining the way the condition is experienced in everyday family life across generations. It thus overcomes the problems of a purely medical or social explanation. Because it is located at the intersection between these two often competing paradigms, it has the capacity to inform social policy and management of PTSD in a key and critical context: family life.

PTSD by Social Design.

In contrast to the literature presented above that discusses the various causes and manifestations of PTSD, there is a small body of research that argues against any real existence of it in the individual. These studies claim it is essentially a socially and politically created phenomenon designed to fuel economies and industries forged in supporting and protecting mental illness. They assert it is society-specific, not person-specific. Summerfield (2001) and Lee (1997) are two such authors with this controversial and radical view who assert that PTSD is not a condition that lies within the individual, but with the society in which they live. They describe the historic and political events that established PTSD as a common condition among survivors of the Vietnam war and claim that PTSD was created to provide a social identity of victimization as a way of diverting attention away from the individual soldier as a perpetrator of violence. This perspective purports that mental diagnoses have social uses, specifically for the social groups for which they are constructed to legitimize industries of mental health, compensation and

disability. This puts an interesting skew on the diagnosis, description, acquisition and management of PTSD. If PTSD is a socially identified, categorized and controlled condition, then it is anticipated the collection of lived experiences from the subject group will clearly identify this. To do this effectively will require an analysis of the microsociological and interaction elements that are embedded in the everyday language of families with intergenerational PTSD.

Microsociology and Interaction Analysis.

Erving Goffman's analysis of the micro-components of social interactions goes far to achieve this goal by describing the minute factors that determine and distinguish everyday human interactions (Hacking, 2004). In this microsociology, it is asserted that there are several tiers of interaction that have both asserted and assumed rules and ethics (Fine, 1991). It is based on qualitative, rather than quantitative data and is concerned with the "rituals" that people perform in these interactions that provide them with feedback from which they form their identity and focuses on the individual person as a player in a group, and the group as a player in an institution (Rawls, 1987). There is a shift in attention away from the reductionism of the medical model and focus on the individual towards one of social causation and responsibility. The distinction is also made between an individual and a person. The individual is described as a social construct without identity: like a number on a very long list of numbers. The person is described as a social product: a cumulative result of highly specific experiences, interpretations and interactions (Rawls, 1987). This study will adopt these descriptions as this has considerable impact on the medical model and dominant paradigms behind mental health practice.

The investigation of language components in deriving meaning is not a new concept. A recent field of study into this phenomenon focuses on disability as a social product and the application of the language elements is termed Discourse Analysis.

Discourse Analysis.

Several studies focus around a conceptual analysis of the language of the discourse of social interactions. This "Discourse Analysis" (DA) is a method for examining naturally occurring talk (Potter, 2004). It focuses on language as the medium for interaction; but it stops there. In the case of PTSD, DA needs to focus on the medium for MEANING embedded in the everyday experience that is inherent in the language of interactions. As Miller & Glassner (2004) assert, language shapes meanings, but how is the language of

PTSD itself shaped and perpetuated throughout generations? These are the microsocial elements.

Emotional interactions.

A significant factor in interaction cues and rituals are the emotions enveloping and developing from the context in which that interaction occurs. Many studies examine the experience of mental illness and the way in which it can be an emotional response to environmental and social pressures. One such item of literature focuses on the role of emotions in the interaction processes by suggesting that it is our emotions that house our history of experience, interpretation and evaluation and that these emotions influence how we react to the “rituals” of each interaction (Lawler & Thye, 1999). This is a particularly prevalent perspective in the case of PTSD, due to the difficulties experienced by those with this condition in appropriately managing their emotions (O'Brien, 2004).

Recap.

This literature review has described PTSD from the perspectives of various elements contributing to the medical model’s interpretation of this condition. It has also presented the significantly influencing literature pertaining to the social perspective of it, and the microsocial and interactional elements that perpetuates its multigenerational heritability.

The principal focus has been to tease out of the available literature the need for an analysis of the secondary behaviours of PTSD that are embedded in the contexts of everyday interactions in the families of children and grandchildren of Australian Vietnam veterans. These are the foundations and cornerstones of the lived experience, and what subsequently forms identity and concept of self for the person and families living with PTSD. It provides meaning for the symptoms, experiences and the interactions between social experiences and the symptoms. In effect they personify PTSD. This is the information that is not currently available to mental health practitioners that is essential to develop practical, client-focused therapies.

Investigations demonstrate that research of this nature has not been conducted previously and data from testimonies of the experiences of children of Vietnam veterans (many with children themselves) has also not yet been incorporated into such a project in Australia. The investigation of the microsocial rituals that individuals use to forge a personal and social identity is essential to understand the way disabilities and impairments

are experienced, socially defined, and embodied into the social participants and the society they inhabit. PTSD is being used as a prime example of this phenomenon.

Implications for Theory & Practice.

Being diagnosed with a “mental condition” can be a depressing confrontation in itself, especially if the person is suffering depression in the first place. This can only serve to reinforce the negative prognosis presented to many clients when diagnosed by a mental health practitioner. Through awareness of the process of identity development via secondary behaviours embedded in social interactions, and the microsocial cues and rituals that establish these processes, the person can be better prepared for dealing with negative feedback and optimise their social identities.

This research has far-reaching implications in many fields and facilities. As previously described in this document, by raising awareness of the secondary behaviours of PTSD in children of Vietnam veterans and the family environment that perpetuates them, a better mental health strategy can be devised and implemented.

Epigenetics in Mental Health Plans.

There is currently a significant gap in research concerning appropriate and relevant feedback to the field of mental health. There is no system to inform policy and the medical model describing how PTSD is experienced by those it affects. This study takes a step in filling this gap. Presently, the Nature vs Nurture argument continues. No decision has, nor can, be made. Consequently intervention and therapy follows suit and the field of mental health remains divided. In a bold, but plausible approach, epigenetics (a relatively new stakeholder in the argument) asserts that the answers lie in the influence that our environment has over our genes, claiming it is Nature via Nurture. Those researchers who still prescribe to the traditional perspectives are adamant that it is not one, nor the other, but an equal combination of the two that prescribes the heritability of PTSD. However, the argument still cannot come to a conclusion as it fails to consider the minute cues in our social interactions that define PTSD for social participants. For it is these that determine the development of the social diagnosis of PTSD, its acceptance by society and its influence on the development of the person’s identity and image of self. The current dominant medical paradigm fails to examine the contextual variables in the social environment that give rise to the symptoms. The argument doesn’t consider “when” or “how” PTSD is. This thesis aims to highlight the perspective of epigenetics as an explanation of the heritability of PTSD and to assert its inclusion in considering a management strategy for mental health professionals.

Transference of Data.

The data collected in this study can be used to inform both social policy and mental health practitioners of the "experience" of PTSD. Although this data will be collected from the cohort of children and families of Vietnam veterans, it is easily translatable to other families living with PTSD from other events. This data can be used as a contribution to practice by informing practitioners of how PTSD is experienced socially, and therefore personally. This influences both the practical and intellectual issues surrounding its prognosis and effective management of PTSD.

Positive Psychology.

It can also suggest approaches to optimise this seemingly dysfunctional condition. With support from the approach of Positive Psychology, children of Vietnam veterans who have inherited qualities of their parent's PTSD may learn to focus on the strength aspects, instead of the weak ones. If managed effectively, these "difficulties" may be refocussed to become powerful contributors to society. This would support current trends in research around "posttraumatic growth" (Shakespeare-Finch, Gow, & Smith, 2005) and positive psychology (Seligman, 2006). Secondary behaviours of PTSD would begin to be reframed from problematic in nature, to enhancing.

Context Reframing.

All behaviours are appropriate in the right context. There are many social contexts in which the primary and secondary behaviours of PTSD are not appropriate, because of the way it has been framed. A reframing of the condition, redefinition and re-diagnosis emphasizing the potentialising qualities will assist in reducing the cost of management, improve productivity, creativity, participation, and self-image. Studies may be designed to further investigate the factors that affect individuals and their families to assist in the construction and implementation of appropriate policies and guidelines for the optimal management of intergenerational PTSD.

Conclusion.

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