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The role of genes and environment on trauma exposure and posttraumatic stress disorder symptoms: A review of twin studies ☆

Tracie O. Afifi [a,*], Gordon J.G. Asmundson [a], Steven Taylor [b], Kerry L. Jang [b]

[a] Anxiety and Illness Behaviour Laboratory and Department of Psychology, University of Regina, 3737 Wascana Parkway, Regina, SK, Canada S4S 0A2
[b] Department of Psychiatry, University of British Columbia, 2255 Wesbrook Mall, Vancouver, BC, Canada V6T 2A1

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ABSTRACT

Behavioral-genetic (twin) methods are important tools for understanding the etiology of trauma exposure and posttraumatic stress disorder (PTSD). The purpose of the present article is to synthesize the results obtained from twin studies and outline important avenues for further investigation. Twin research to date suggests that: (1) exposure to assaultive trauma is moderately heritable whereas exposure to non-assaultive trauma is not, (2) PTSD symptoms are moderately heritable, and (3) comorbidity of PTSD with other disorders may be partly due to shared genetic and environmental influences. Remarkably little is known about whether the observed comorbidity of PTSD with particular personality traits and poor physical health is due to shared genetic or environmental factors. Similarly, little is known about whether gene–environment interactions play an important role in trauma exposure and PTSD. Further research is required to clarify these issues and to determine whether findings to date, obtained mostly from male combat veterans, generalize to other populations. Research programs that integrate behavioral-genetics with molecular genetics and with cognitive–behavioral conceptualizations and research methods may deepen our understanding of the complex links among genes, brain, cognition, emotion, and the environment.

Contents

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* Corresponding author. Tel.: +1 306 337 2473; fax: +1 306 337 3275.
E-mail addresses: toafifi@hsc.mb.ca (T.O. Afifi), Gordon.asmundson@uregina.ca (G.J.G. Asmundson), steven.taylor@ubc.ca (S. Taylor), Kerry.jang@ubc.ca (K.L. Jang).

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1. Introduction

According to the Diagnostic and Statistical Manual of Mental Disorders fourth edition (DSM-IV), a traumatic event is something that is experienced or witnessed and involves actual or threatened death, serious injury, or threat of physical integrity of oneself or others and invokes a response of fear, helplessness, or horror (American Psychiatric Association, 1994). Examples of traumatic events include war-related events, natural disasters, physical assault, sexual assault or violence, threats with weapons, serious accidents, illness, and the unexpected death of loved ones. After an exposure to a traumatic event, some individuals may develop posttraumatic stress disorder (PTSD). PTSD is characterized by reexperiencing a traumatic event along with avoidance of reminders of the trauma, emotional numbing, and increased arousal (American Psychiatric Association, 1994).

1.1. Epidemiology of traumatic events and PTSD

Many people experience traumatic events at some point during their lifetime. The prevalence of exposure to traumatic events and PTSD reported in epidemiologic studies varies depending on the method and criteria used for assessing traumatic events (Breslau, 2002). Epidemiologic research from several countries has indicated that experiencing a traumatic event in the general population is common; using DSM-IV criteria the lifetime prevalence ranges from 64% to 90% (Breslau et al., 1998; Creamer, Burgess, & Mcfarlane, 2001; Frans, Rimmo, Aberg & Fredrikson, 2005; Norris et al., 2003; Stein, Walker, Hazen & Forde, 1997). The lifetime prevalence of PTSD estimated in epidemiologic studies using community and national samples and DSM-IV criteria ranges from 1.4% to 11.2% (Alonso et al., 2004; Breslau et al., 1998; Frans et al., 2005; Hapke, Schumann, Rumpf, John & Meyer, 2006; Kessler, Berglund, Demler, Jin, & Walters, 2005; Kessler, Chiu, Demler, & Walters, 2005; Norris et al., 2003).

Epidemiologic research has identified risk factors that increase likelihood of PTSD after exposure to a traumatic event. A meta-analysis of 77 studies published between 1980 and 2000 identified 14 risk factors for PTSD, with psychiatric history, reported child abuse, and family psychiatric history being the most uniform predictors of PTSD regardless of the study population and methods (Brewin, Andrews & Valentine, 2000). PTSD is also associated with comorbidity of other mental health disorders (Breslau & Davis, 1992; Creamer et al., 2001; Kessler, Sonnega, Bromet, Hughes & Nelson, 1995), suicidal behavior (Kessler, Borges & Walters, 1999; Sareen et al., 2007), and physical health problems (Lauterbach, Vora & Rakow, 2005; Sareen, Cox, Clara & Asmundson, 2005). Epidemiologic studies demonstrate that PTSD is an important public health concern; however, these studies cannot adequately disentangle the genetic and environmental etiology of PTSD. Understanding the genetic and environmental etiology will have important implications for identifying causal pathways that lead to PTSD and its comorbidity with mental and physical disorders and for informing treatment efforts.

1.2. Current objectives

Using behavioral-genetic analyses of responses from monozygotic (MZ) and dizygotic (DZ) twins, researchers have begun to understand the influence that genetic and environmental factors have on exposure to traumatic events and PTSD. The purpose of the present article is to review the current twin study literature on: (1) the heritability of traumatic events, (2) the heritability of PTSD, (3) the role of genetic and environmental influences on comorbidity of PTSD with other mental disorders, (4) the role of genetic and environmental factors on the relationship between PTSD and physical health, (5) importance of gene and environment interactions and molecular genetics study designs, (6) integration of behavioral-genetics with cognitive–behavioral approaches, and (7) future directions for PTSD twin studies based on the current state of knowledge and limitations in the present literature.

This review extends the current literature in several important ways. First, previous review articles on PTSD and genetics have focused largely on molecular genetics (studies using DNA analysis) with only a brief summary of PTSD twin studies and behavioral-genetics (Amstadter, Nugent & Koenen, 2009; Koenen, 2007; Koenen, Amstadter, & Nugent, 2009; Koenen, Nugent, & Amstadter, 2008; Nugent, Amstadter & Koenen, 2008). The present article is unique because it moves the focus to a comprehensive review of the twin studies literature on genetic and environmental factors that influence exposure to traumatic events and PTSD. Second, this novel focus allows for an examination of several important areas of research that have not been previously discussed in other reviews, including genetic and environmental overlap of PTSD symptoms with symptoms of other DSM-IV Axis I disorders (e.g. anxiety disorders, major depression, conduct disorder, substance use disorders), twin studies of PTSD and physical health, and the integration of behavioral-genetics with cognitive–behavioral approaches. Third, reviewing twin studies is important because these studies can provide a roadmap for guiding molecular genetic studies on PTSD, which can help refine the phenotype and help identify whether various clinical phenomena are etiologically related; for example, if twin studies indicated that generalized anxiety disorder and PTSD are related, then this would guide molecular genetics research to look for genes shared by these disorders. Finally, a review of PTSD twin studies that distinguish between genetic factors and shared environment can inform family studies that are unable to clarify the sources of familial covariance. If twin studies suggest that shared environment is unimportant, then family studies can generate important hypotheses about genetic effects.

2. Twin studies

Commonly used behavioral-genetics terms and notations are provided in Table 1.

The most popular twin study design involves comparing the similarity of MZ and DZ twins that were raised together on a variable of interest (Jang, 2005). Greater similarities in MZ twin pairs determined through correlations may suggest a heritable basis of a trait because MZ twins share 100% of their genes while DZ twins share approximately 50% of their segregating genes. Therefore, measures of twin similarity, such as intra pair correlations or their covariance, can be used to estimate heritability. Quantitative genetic theory (Falconer, 1960) partitions genetic effects into additive genetic effects (often denoted as A) and non-additive genetic effects (often denoted as D). Additive genetic effects are the sum of the average effects of the individual alleles (alternative forms of a gene at the same site of a chromosome) (Neale & Maes, 2004). Non-additive genetic effects, including dominance (interaction between alleles at the same