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Scoping Review of Risk Factors of Anxiety Disorders and Post-Traumatic Stress Disorder: A Public Health Perspective

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Abstract

Objective: To identify risk factors for Anxiety Disorders and Post-Traumatic Stress Disorder (PTSD) in the general population. Knowledge of risk factors is useful for identifying at-risk populations, guiding the development of prevention programs, and improving early detection of these disorders.

Method: A scoping review of English and French language articles published between 2003 and 2015 was conducted. Longitudinal studies of risk factors in the general population using definitions of anxiety disorders and PTSD as per the DSM-IV nomenclature were included. Associations between risk factors and anxiety disorders or PTSD were summarized and compared.

Synthesis: Twenty-two longitudinal studies focusing on anxiety (N=17) and PTSD (N=5) were retained. The bulk of evidence came from ten large, population-based, longitudinal prospective studies. Six categories of risk factors emerged: 1) genetic/biological, 2) neurodevelopmental, 3) environmental, 4) temperamental, 5) behavioral, and 6) stress-related.

Conclusion: Risk factors for anxiety disorders are multifactorial and often mutually interactive. Childhood is a critical period. Children of parents with psychopathology, who demonstrate behavioral inhibition, who demonstrate antisocial behavior, who have low IQ, or who live in chronically adverse conditions such as poverty, are at risk of developing anxiety disorders. A history of juvenile disorders, severe maltreatment, poor quality of parental care, as well as symptoms of PTSD themselves, can increase the likelihood of PTSD. PTSD symptoms may develop in response to traumatic/life threatening events as well to accumulation of stressful, but non-traumatic events. Successful prevention of anxiety disorders and PTSD should focus on modifiable behavioural, environmental, and stress-related risk factors.

Keywords: Anxiety disorders; PTSD; Risk factors; Prevention; Public health

Introduction

"Anxiety disorder" is a term that encompasses the spectrum of mental disorders for which intrusive, intense, and persistent fear is the most prominent symptom [1]. Anxiety disorders are pervasive, chronic, and incapacitating [2]. To prevent the onset and minimize the impact of anxiety disorders and PTSD, it is necessary to understand the risk factors for these disorders and identify populations at risk [3]. Delineating risk factors for any mental disorder is a complex process because of numerous interconnected and co-occurring risk factors. Risk factors may not only make one more vulnerable to develop anxiety disorders and PTSD, but can also predict and mediate their development. For these reasons, large epidemiological studies and longitudinal, population-based prospective studies are recommended to better ascertain which variables can be considered risk factors [4].

The impact of risk factors can be reduced through prevention and counteracted by early intervention. Risk-factor research can inform the development of models and programs of prevention in the context of public health as well as address the optimal timing of interventions [5]. In this article we summarize results of a scoping review of evidence on risk factors for anxiety disorders and PTSD published between 2003 and 2015; thus during approximately the last decade prior to the arrival of the fifth edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [6].

For the purposes of this review, anxiety disorders were defined according to the text revision of the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) [7] because there are at present no longitudinal studies published using the new DSM-5 diagnostic criteria. The disorders included in this review therefore include panic attacks (PA), agoraphobia, panic disorder (PD) with or without agoraphobia, specific phobia (SP), social phobia or social anxiety disorder (SAD), obsessive-compulsive disorder (OCD), anxiety disorder due to a general medical condition, substance-induced anxiety disorder, generalized anxiety disorder (GAD), anxiety disorder not otherwise specified, acute stress disorder, and post traumatic stress disorder (PTSD). Although the DSM-5 issued in May 2014 reclassified acute stress and PTSD as trauma- and

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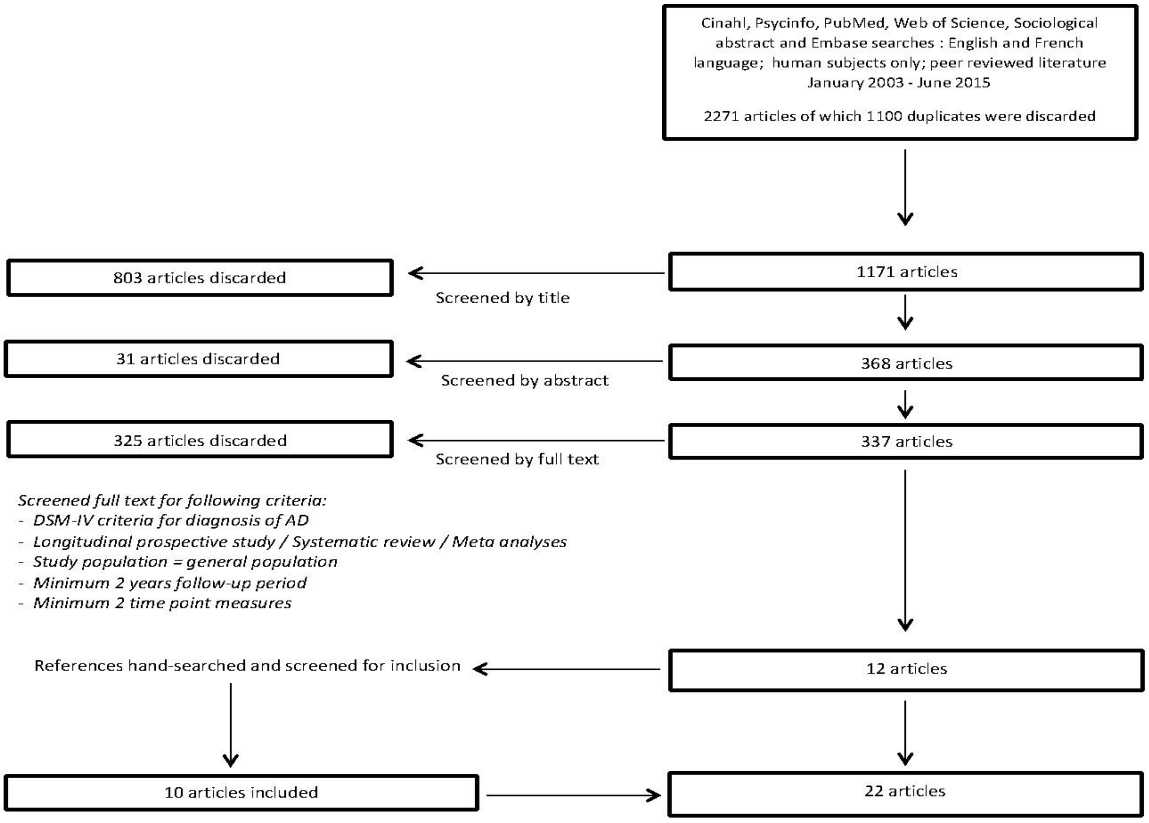

Figure 1: The scoping review flow diagram.

stressor-related disorders (American Psychiatric Association, 2013) the studies included in this review included acute stress and PTSD as anxiety disorders for the reasons outlined above.

Method

This scoping review followed the method outlined by Arksey and O'Malley [8]. PubMed, PsychINFO, Embase, Sociological Abstract, Web of Science, and CINAHL were searched using the following terms and combinations for each database: (anxiety OR phobia OR agoraphobia OR traumatic stress OR acute stress OR obsessive OR compulsive OR panic) AND (factors OR predictors OR precursors OR precipitation OR predisposition). The search spanned the period from 1993 to 2015 but was reduced to the last 12 years due to the large number (nearly 4000) of citations generated by the initial search. The search was therefore repeated for 2003 - 2015 (end of June) and was limited to human studies published in English and French. Scanning of reference lists and hand searching followed the search of specialized databases.

The following inclusion criteria applied: 1) Study includes at least one risk factor for at least one anxiety disorder, 2) Anxiety disorders are defined following the DSM-IV nomenclature, 3) Data is from a longitudinal prospective study or a systematic review of longitudinal studies using the DSM-IV nomenclature, 4) Study was conducted in a general population, 5) Follow-up period is at least 2 years, 6) Minimum 2 points of follow-up measure reported (T1, T2… or Wave 1, Wave 2).

The flow diagram in Figure 1 summarizes the selection process. Two reviewers independently screened titles and abstracts and reviewed full texts of articles. Disagreements were resolved through consultation with a third reviewer. All eligible studies were evaluated for conformity with the Strengthening The Reporting of Observational Studies in Epidemiology (STROBE) Statement, a checklist of 22 items that should be included in reports of cohort studies (STROBE Statement; 2007) [9]. The STROBE Statement is particularly useful to facilitate critical appraisal and interpretation of studies [10]. Studies were most often excluded for failing to report diagnostic criteria, for not indicating the number of participants at each stage of the investigation, or for short follow-up (less than 2 years).

Synthesis

The systematic search from six databases yielded 1171 potentially relevant citations (Figure 1). A total of 337 articles were retrieved for full-text screening. Of these, 325 were excluded because they did not meet inclusion criteria, and 12 were retained. Hand-search and reference screening of key resources yielded 10 additional eligible articles. In total, 22 articles were retained for final review.

Seventeen of the final 22 articles were based on data from 10 large population-based longitudinal prospective studies. Nine articles addressed risk factors for anxiety disorders in general, five focused on PTSD, two covered SAD, two were about OCD, and four addressed PD. The majority of risk factors for anxiety disorders identified by these studies fall under the categories of biological/genetic factors,

neurodevelopmental factors, childhood temperamental and behavioural factors, environmental/contextual factors, and stress-related factors. When available, the Odds Ratios (OR), Relative Risks (RR) or Hazard Ratios (HR) and their 95% Confidence Intervals (CIs) or p values are presented in the main findings.

Risk factors for anxiety disorders in general

Table 1 presents summary or risk factors from studies focusing on anxiety disorders in general. Reports from the TRAILS study "Tracking Adolescents' Individual Lives Survey" [11,12] indicate that two types of risk factors predict the development of anxiety throughout adolescence [13]. Risk factors of stable high levels of anxiety included temperament, high frustration, low effortful control, lifetime parental internalizing problems, and being a victim of bullying. These factors predicted long-term risk of anxiety disorders. In contrast, converging risk factors typically decreased by 16-17 years of age and included low self-competence, rejecting and overprotective parenting, and being a bully-victim. Karevold, Roysamb, Ystrom and Mathiesen [14] used data from the TOPP study "Tracking Opportunities and Problems in Childhood and Adolescence" [15] and suggest that anxiety develops through two major pathways: temperament and early contextual risk factors from before age 5 [14]. The authors suggest that these risk factors behave differently; temperamental risk factors have exclusively indirect effects whereas contextual risk factors have both direct and indirect effects.

Behavioural Inhibition (BI) has been identified in the literature [16] as one of the important temperamental risk factors for SAD in children. Hudson, Dodd, Lyneham, & Bovopoulos [17] conclude that BI at age 4 was a strong predictor of social phobia (OR=34.77 p<0,001). With regard to SAD, Bittner, Egger, Erkanli, Costello, Foley, & Angold [18] tested the predictive specificity of several childhood anxiety disorders using data from the GSMS "Great Smoky Mountains Study" [19,20]and also found that SAD in childhood was significantly associated with adolescent SAD (OR=9.6 [1.1–78.4]).

Maternal anxiety is an example of a contextual risk factor. When it is present at a young age it affects child anxiety over time [17]. The authors found that maternal anxiety and maternal over-involvement are associated with BI, and increase the likelihood of anxiety later in life. It appears that temperamental and contextual risk factors have additive and interactive effects on the pathogenesis of anxiety in children [16].

Espejo, Hammen, and Brennan [21] investigated the impact of naturally occurring life events on the development of anxiety disorders in a sample of 653 adolescents at ages 15 and 20 from the MUSP study "Mater University Study of Pregnancy" [22]. When adolescents rated highly the negative impact of life events, the occurrence of subsequent first onset of anxiety disorders was significant (OR=1.35 95% CI [1.15-1.57]). It is noteworthy that when maternal depression was present, females were more likely than males to report higher negative impact appraisals (p<0.001p) and also more likely than males to experience anxiety disorders (38.2% versus 26.8%, $\chi 2$ (1, N=653)>9.50, p<0.002).

A longitudinal study in Norway [23] looked at a preschool population of 1000 children (age 4) at a two-year interval (4 and 6 yrs old). Parental anxiety (OR 1.10, 1.03-1.19) and behavioural inhibition (OR 1.39, 1.5-1.83) and peer victimization (1.49, 1.23-1.98) as well as ADHD (OR 3.95, 1.28-12.16) were predictors of anxiety disorders at age 6. High scores in social skills were protective, while low social skills predicted anxiety disorders. Their recommendations include the early implementation of effective treatments of parental anxiety and child ADHD.

Asselman, Wittchen, Lieb, Hofler, and Beesdo-Baum [24] reported on risk factors for anxiety disorders based on a 10-year longitudinal study conducted in a middleclass neighborhood of Munich, Germany, which at baseline included 3021 youth and young adults. They examined the relationship between Fearful Spells (FS) and Panic Attacks (PA) with the risk of developing anxiety disorders in help-seeking subjects and non-help-seeking subjects. FS/PA at baseline strongly increased any incident disorder. FS/PA increased the risk for Panic Disorder (PD) and depression only in individuals NOT seeking help. FS/PA at baseline and number of panic symptoms increased the risk for incident PD in non-help-seeking individuals PD (OR=8.31, 95%CI: 4.08, 16.93; p < .001). Findings suggest that early interventions in individuals with FS/PA especially those with a higher number of PD symptoms may be useful to prevent later exacerbation/development of panic pathology and depression.

In subsequent analyses conducted on data from the same longitudinal study from Munich, Germany, Asselman, Wittchen, Lieb, and Beesdo-Baum [25] examined more specifically examined whether child low valence (emotional connectedness), low child potency (individual autonomy), and mother psychopathology increase the risk for offspring anxiety and/or depression. Parent interviews were conducted with 1015 mothers of the 3021 participants, thus reducing the total study sample. Logistic regression analyses showed that low child potency as reported by the mothers increased the risk of anxiety (OR=1.24, 95%CI: 1.07, 1.44 p=.004). Moreover, in the presence of maternal anxiety, low potency predicted comorbid anxiety and depression in the child (OR=1.58, 95%CI: 1.23, 2.04 p < .001). In the subjects where the mother reported low valence, only females had a higher risk of co-morbid anxiety and depression. Thus, in this study only low potency was a predictor of "pure" anxiety disorders. Maternal anxiety on its own was predictive of depression, not anxiety.

Risk factors for Post-Traumatic Stress Disorder (PTSD)

A summary of risk factors for PTSD can be found in Table 2. As part of the DMHDS "Dunedin Multidisciplinary Health & Development Study" [26], a group of international researchers examined the association between temperamental, neurodevelopmental, behavioural, and family environmental characteristics assessed before age 11 and the development of PTSD up to age 32 [27]. Two sets of childhood risk factors emerged. One included children's externalizing characteristics (e.g. difficult temperament, antisocial behaviour, and hyperactivity), family history of mental-health problems (e.g., maternal distress), and family adversities (e.g. loss of a parent). The other included low IQ and chronic environmental stressors or adversity (e.g., low socioeconomic status). The cumulative effect of these childhood factors on the risk of developing PTSD at age 26 was significant: more than 58% of cohort members in the highest risk quartile for three developmental factors (neurodevelopment, temperament, behavior/family environment) later developed PTSD. By comparison, only 25% of those not at high risk for any factors developed PTSD. The likelihood of PTSD increases with increasing number of risk factors.

The effects of Potentially Traumatic Events (PTEs) on risk of PTSD underlie the research of Cougle, Resnick, and Kilpatrick [28]. Basing their investigation on the "sensitization hypothesis" (prior exposure sensitizes people to respond more intensely to subsequent stressors), they examined the effects of physical and sexual assault on the risk of PTSD in response to subsequent PTEs. Using longitudinal data (N=1725) with two assessment points in an eight-year period, it was predicted that among those without PTSD at Wave 1, prior assault would predict PTSD in response to a new PTE reported at Wave 2. The authors found that prior assault indeed increased the risk of developing PTSD following subsequent exposure. However, prior witnessed violence in the absence of PTSD did not demonstrate similar effects [28].

In 2012, Milan, Zona, Acker, and Turcios-Cotto aimed to identify how factors at the environmental (e.g., community quality), family (e.g., family adversity, parental support), and individual (e.g., mental health, cognitive vulnerabilities, interpersonal problems) levels jointly distinguish adolescents who will later be exposed, or not, to violence, as well those who will subsequently develop, or not, PTSD following exposure to violence. They also tested whether symptoms of PTSD contribute to the increase or maintenance of PTSD symptoms over time (i.e., stress generation processes). Finally, a secondary goal of this study was to examine if preexisting factors interact with characteristics of a traumatic event (i.e., proximity to the event, close relation to perpetrator or victim, and sustaining a physical injury) in influencing the likelihood of developing PTSD.

Milan, Zona, Acker, Turcios-Cotto [29] used prospective data over a two-year period from the PHDCN study "Project on Human Development in Chicago Neighborhoods" [30], a longitudinal cohort study designed to understand how families, schools, and neighbourhoods influence child development. The sub-population studied was a large, diverse sample of urban adolescents (n=1242, Mean age 13.5). Results indicated that 23 (1.9%) out of 1242 adolescents met criteria for PTSD. A total of 569 (46 %) adolescents experienced violence in the past year as either a victim or direct witness of severe acts. Prevalence of PTSD among those exposed to recent violence was 4.0 %. Demographically, boys and older youth were more likely to experience violence; but gender or age did not predict the likelihood to later develop PTSD in response to violence. African-American adolescents were more likely to experience violence, yet less likely than other racial/ethnic backgrounds to develop PTSD. While a history of interpersonal violence, externalizing behaviors, and association with deviant peers predicted subsequent exposure to violence, they did not predict PTSD in response to violence. Race/ethnicity, thought disorder symptoms, and social problems were predictive of PTSD in response to violence.

Among youth exposed to violence, Time 1 risk factors did not predict traumatic event features associated with elevated PTSD rates (e.g., parent as perpetrator), nor did interactions between Time 1 factors and traumatic event features add significantly to the ability to predict an eventual diagnosis of PTSD.

Milan et al. [29] underscore that vulnerability factors and environmental stressors are traditionally conceptualized as two distinct domains that interact to predict a third domain, made up of symptoms or a psychological disorder (in this case PTSD). Their study supports an alternative view that these three domains are rather mutually influential. Over time, PTSD symptoms themselves can become a vulnerability factor and contribute to the generation of environmental stressors such as violence exposure, which in turn serves to maintain PTSD symptoms.

In New Zealand, Mulder, Fergusson, and Harwood [31] examined if PTSD symptoms form an internally consistent distinct latent dimension, and if PTSD symptoms develop solely in response to traumatic events or also in circumstances that include non-traumatic, albeit negative, life events. By examining non-life event risk factors such as gender, personality, and family circumstances collected over 30 years, they were also able to study the role of individual vulnerability factors in generating PTSD symptoms. This study is based on data gathered within the CHDS "Christchurch Health and Development Study" [32], a longitudinal study of a birth cohort of 1265 children born in the Christchurch region in 1977. The surviving cohort (N=987) was studied on 22 occasions from birth to age 30 years, generating data from a variety of sources including parental interviews, teacher reports, psychometric testing, interviews with cohort members, medical, and other official record data.

Mulder et al. [31] communicate four main conclusions: First, the experience of PTSD symptoms in association with significant life events is common, with most individuals reporting no impairment, and a minority reporting severe symptoms and impairment. PTSD symptoms thus fall on a continuum of severity with mild stress at one end and severe PTSD at the other. Second, PTSD symptoms form a distinct syndrome reflecting DSM-IV symptom criteria B–F [7] and also reflecting the severity of PTSD symptomatology. Next, PTSD is not limited to being a response to overwhelming trauma and can be associated with non-traumatic life events. Studying PTSD symptoms in a general population sample with responses to a wide range of life events enables testing for a dose-response relationship. Individuals with the most elevated PTSD symptom severity reported having experienced more traumatic events and more other significant negative events when compared to individuals with lower symptom severity. Finally, personal characteristics and childhood family circumstances have emerged as potential risk factors; female gender, higher neuroticism scores, childhood anxiety, low self-esteem, and poor quality of parental care are significant predictors of PTSD.

In 2014, Breslau, Koenen, Luo, Agnew-Blais, Swanson, et al., [33] conducted analyses on a subset of data (n=928) obtained in the DMHDS that included complete data on exposure to traumatic events and PTSD for a 12 year adulthood period (from age 26 to age 38). They examined prospectively if childhood maltreatment in the first decade of life predicts the development of PTSD following exposure to trauma during adulthood as well as if a history of juvenile disorder, independent of early childhood maltreatment, also predicts adult PTSD. They found that the most severe cases of maltreatment, compared to no maltreatment, were associated with an increased risk of developing PTSD in adulthood. They also found that a history of juvenile disorders, independent of earlier maltreatment, was also associated with an increased risk of PTSD in response to adult trauma. This being said, the authors underlined that moderate maltreatment, which is experienced by the majority of adult trauma victims who were maltreated as children, was not statistically distinguishable

from absence of childhood maltreatment. The authors suggest more longitudinal studies are needed to clarify the role of moderate maltreatment as a risk factor for adult PTSD [33].

Risk factors for Social Anxiety Disorder (SAD)

Table 3 presents the summary of risk factors for SAD. Essex, Klein, Slattery, Goldsmith, and Kalin [34] investigated the effect of BI on SAD and found that chronic high levels of inhibition were significantly associated with a lifetime history of SAD by grade 9. Interestingly, there were no significant differences in inhibition at grade 1 between children with and without the diagnosis of SAD, but by grade 3 children diagnosed with SAD had significantly higher inhibition scores than those without SAD [34]. It is likely that BI could predict new onset of social anxiety in mid to late childhood and early adolescence. The authors observed that girls were at greater risk for developing chronic high inhibition and SAD. They suggested that cultural expectations and socialization patterns encourage some degree of inhibition and increased stress responsivity in girls but not in boys.

Knappe, Beesdo, Fehm, Lieb, and Wittchen [35] examined the associations between parental SAD and other parental psychopathology, parental rearing, and family functioning on the risk of SAD in offspring, with a special focus on the interaction of these risk factors in a prospective longitudinal 10 year follow up from the EDSP study "Early Developmental Stages of Psychopathology" [36,37], a study of 1022 youth and young adults ages 14-24 in Germany. The authors found that children of parents with SAD were more often affected by SAD (13.2%) than were those whose parents exhibited no psychopathology (4.2%). Rates of offspring with SAD were elevated if parents were affected by other anxiety, depression, or alcohol use disorders (range 10.5–11.7%, OR-range 2.6–2.9). The parent–offspring association was the strongest when parental SAD was comorbid with three or more disorders (OR=7.8, 95% CI: 2.8–21.9). Regarding family environment, offspring with SAD reported more parental overprotection, rejection, and less emotional warmth than those without SAD. Considering combined effects of parental psychopathology and family environment on the offspring's SAD, the risk for SAD was increased in offspring of parents with SAD who reported greater rejection (OR=3.2, 95% CI: 1.3–8.1), overprotection (OR=3.4, 95% CI: 1.4–8.5), and lack of emotional warmth (OR=3.5, 95% CI: 1.4–8.8), compared to offspring of parents without SAD. The authors found that SAD was particularly prevalent in mothers but not in fathers, but could not ascertain if the offspring risk for SAD would be increased if both parents were affected [35].

Risk factors for Obsessive-Compulsive Disorder (OCD)

A summary of risk factors for OCD is presented in Table 4. Based on data from the DMHDS, a longitudinal birth cohort of primarily white children born in 1972-1973, Grisham, Anderson, Poulton, Moffitt, and Andrews [38] hypothesized that deficits in neuropsychological test performance at age 13 would predict OCD diagnoses at age 32.

More specifically, they predicted that strong associations between childhood test performance and adult OCD would be within the visuospatial/visuomotor and executive domains, whereas general IQ and verbal ability would be unaffected. As predicted, the OCD group demonstrated poorer performance on tests of visuospatial and visuomotor ability than the rest of the cohort. However, mixed results were observed on tests of executive functioning, such as planning, cognitive flexibility, and set shifting. Although results are inconclusive, evidence points to neuropsychological deficits present prior to the onset of OCD. Of note is that not all participants with neuropsychological deficits developed OCD. Authors conclude that multiple factors such as parenting styles or critical stressful life events must account for OCD [38].

In another analysis of the DMHDS's data, Grisham, Fullana, Mataix-Cols, Moffitt, Caspi, and Poulton [39] prospectively identified a range of risk factors for OCD and Obsessive-Compulsive Symptoms (OCS) in adulthood (ages 26 and 32). The strongest risk factors were childhood physical (OR=7.7 95% CI [2.7-22.0]) and sexual (OR=3.5 95% CI [1.4-8.6]) abuse. Symptoms of greater internalizing and social isolation in childhood predict increased odds of OCD in adulthood. The OCD group differed from the healthy control and anxious groups with higher negative emotionality. Interestingly, risk factors for adult OCS included neurodevelopmental risk factors, several temperamental and behavioural characteristics, all of the personality dimensions, and most of the childhood stressors, notably physical and sexual abuse. Although childhood OCS predicted later diagnosis of OCD, it was not a stable diagnosis as only 11% of the OCD group met diagnostic criteria at ages 26 and 32 because of fluctuations in the severity of symptoms over time.

Risk factors for panic disorder

Four longitudinal studies describe risk factors for panic disorder (PD) Table 5. Two longitudinal studies investigated whether panic attacks were a risk factor for PD and other psychopathology. Goodwin, Lieb, Hoefler, Pfister, Bittner, and colleagues [40] report on the EDSP study and Kinley, Walker, Enns and Sareen [41] report on the NESARC study "National Epidemiologic Survey on Alcohol and Related Conditions" of a large sample (34,653) of young adults (18-24) conducted by the United States Census Bureau [42-45]. Both studies established that the presence of panic attacks was associated with a significantly increased incidence of any anxiety disorder. Goodwin, Lieb, Hoefler, Pfister, Bittner, et al., [40] also found strong associations between panic attacks at baseline and cumulative lifetime incidence of PTSD (OR=8.3 IC 95% [4.2-16.5]), agoraphobia (OR=6.9 IC 95% [4.2-11.5]), OCD (OR=6.3 IC 95% [2.6-15.3]), generalized anxiety disorder (GAD) (OR=6.7 IC 95% [3.6-40.4]), social phobia (OR=3.1 IC 95% [2.0-4.7]), and specific phobia (OR=3.1 IC 95% [2.2-4.4]). Among those with panic attacks at baseline, the likelihood of having three or more anxiety disorders at follow-up was about 14 times higher (OR=14.2 CI=8.4–23.9). Similarly, Kinley, Walker, Enns, and Sareen [41] found that panic attacks were significantly associated with increased incidents of any anxiety disorder, including PD, GAD, SAD, and also major depression, dysthymia, mania, and hypomania. Specifically, having panic attacks at Wave 1 increased the odds of having GAD (OR=1.70 IC 95% [1.21-2.40]), PD with or without agoraphobia (OR=2.73 IC 95% [1.93-3.87]), and SAD OR=1.89 IC 95% [1.12-3.19] at Wave 2 three years later. People with PD were also at an increased risk of a new onset of anxiety disorder than those with panic attacks (AOR 1.41, 95% CI 1.03–1.93). Evidently, there is a relationship between panic attacks and severe psychopathology (defined as the presence of multiple concurrent anxiety disorders), although the causal mechanism is not yet understood.

Mathyssek, Olino, Verhulst and van Oort [46] examined whether both parent- and self-reported internalizing and externalizing behaviour problems in childhood might predict the onset of panic attacks later in adolescence. Three subscales of the Child Behavior Checklist predicted the onset of panic attacks, including "social problems reported by parents" (HR 1.19, $p<0.05$), "social problems reported by youth" (HR 1.26, $p<0.01$), and "thought problems reported by youth" (HR 1.15, $p<0.05$) (adjusted for sex and socioeconomic status). Social Problems was the most consistent risk factor for panic attacks. The authors explain that social problems may result from poor social skills and difficulties in peer relations, may be an expression of genetic transmission of vulnerability from parents to their children, or yet be a gene-environment interaction.

Isensee, Wittchen, Stein, Hofler, and Lieb [47] followed 3021 adolescents and young adults ages 14-24 over a 4-year period as part of the EDSP study. They observed that nicotine can trigger and increase anxiety symptoms and might play role in the aetiology of PD. They investigated if smoking (occasional or regular) or nicotine dependence increased the risk of panic attacks and PD, and vice versa. Isensee et al., [47] found a heightened risk for all categories of smoking and nicotine dependence. Interestingly, prior smoking and dependence increased the risk of new onset of PD while pre-existing PD did not associate with later onset of smoking. Authors also established a dose-response relationship demonstrating that even occasional very low doses of nicotine could trigger panic. These associations were specific for PD but not for other anxiety disorders. Previous research supports these findings and also extends the risk of smoking to GAD [48].

Discussion

This review builds on the evidence from twenty-two longitudinal prospective studies of risk factors for anxiety disorders and PTSD published between 2003 and 2015. The identified risk factors are broad and biopsychosocial in nature, encompassing biological/genetic, neurodevelopmental, environmental, temperamental, behavioural, and stress-related spheres. Studies clearly show that anxiety disorders and PTSD are most often linked to the complex interplay of these factors rather than to a single factor.

Biological/Genetic risk factors

Several studies [35,39,46] in this review discuss the hypothesis that anxiety disorders might result from an expression of genetic transmission of vulnerability from parents to their children, or a gene-environment interaction. Similarly, studies with twins not included in this review demonstrate that while genes may predispose to GAD, panic, agoraphobia, and to a lesser extent, social phobia, the etiology of anxiety disorders is largely explained by environmental factors [49]. Within this viewpoint, it is hypothesized that a specific gene may increase the probability of developing a common intermediate phenotype, such as an anxious personality trait, but it does not guarantee the disorder. This phenotype has to interact with modifier genes or stressful life events to surpass the threshold for the development of one or sometimes even more disorders. This supports the notion that genetic risk factors do not express themselves in a vacuum, but rather in interaction with environmental risk factors. To date researchers have difficulty separating genetic contributions from environmental impact, like in the case of parenting and parental psychopathology. Considering that everyone has more or less of a biological predisposition or inherited vulnerability to develop anxiety and that the expression of this predisposition is mediated by environmental factors should be taken into consideration when developing intervention programs to help prevent or treat anxiety disorders. Educating the public about the interplay between biological/genetic vulnerabilities and environmental/contextual factors may help individuals better understand the complex nature of anxiety as well as clarify which elements in one's life may be more amenable to modifications, from those over which we have relatively little or perhaps no control. Considering anxiety in the context of an interaction between biological/genetic vulnerabilities and environmental/contextual factor scan also guide individuals make more informed decisions about various types of treatments (e.g. pharmacological versus psychological treatments) that may focus more or less on the biological versus psychological aspects of anxiety.

Neurodevelopmental risk factors

Several childhood neurodevelopmental and cognitive deficits were found to be predictive of symptoms of obsessive-compulsiveness, OCD and PTSD in adulthood, [27,39] including perinatal insults and motor skills [39]. Perinatal insults were linked to increased risk for symmetry/ordering and shameful thoughts dimensions, whereas poor childhood motor skills predicted harm/checking dimensions. This being said, not all people with the same neurodevelopmental and cognitive deficits go on to develop OCD [38,39]. This may depend on psychosocial factors, such as parenting styles, interpersonal relationships, or critical stressful life events. Low childhood IQ has been shown to be associated with increased risk and severity of anxiety disorders in adulthood [27] as well as with an increased risk for most dimensions of OCD [39]. In contrast, higher IQ appeared to reduce the risk of PTSD and agoraphobia [38]. Although the mechanisms by which low IQ increases the risk for PTSD are not well understood, individuals with less cognitive resources may be less able to interpret and process traumatic events in an adaptive way [27]. A range of childhood developmental factors may predict one's vulnerability to OCD or PTSD later in life. The fact that not all individuals with neurodevelopmental deficits develop anxiety disorders points to individual differences that may be leveraged through parent-training and school-based interventions.

Temperamental and behavioural risk factors

Although there is no consensus for the definition of "temperament," many agree that temperament refers to a distinctive profile of feelings, behaviours, and aspects of personality that originate in the child's biology and appear early in the development [50]. Although temperament is believed to be genetic in nature and thus fundamentally not modifiable, it is the behavioural expression per se of temperament that may be modifiable through the context or the environment. Behavioural Inhibition (BI), a temperamental trait consistent with a response of restraint, caution, and withdrawal to novel objects and situations, stands out as a specific risk factor for social anxiety [16,34]. However, some studies found that childhood BI was a strong predictor not only of SAD but also anxiety in general [17]. There appear to be gender differences in the development of BI, as girls were at greater risk for BI and SAD than boys and it has been suggested that cultural expectations and socialization patterns encourage some degree of inhibition and increased stress

responsivity in girls but not in boys [34]. Childhood and adolescence were identified as particularly sensitive periods when BI could impact new onset of anxiety disorders [16,34]. Children and adolescents, particularly those showing traits of behavioral inhibition, may thus benefit from interventions aimed at helping them develop tolerance and more adaptive responses to novel and unexpected life circumstances.

Researchers [27] found that difficult temperament, antisocial behaviour, and hyperactivity might increase vulnerability to PTSD through increased risk of trauma exposure and hypothesized that these temperamental and personality factors interfere with the ability to adapt to the impact of a particular situation in an adaptive way. Similarly, other scientists [23] noted that the presence of parental anxiety, behavioural inhibition, peer victimization, as well as ADHD, was found to be predictors of anxiety disorders at age 6. Having solid social skills is protective, while low social skills predicted anxiety disorders. Their recommendations include the early implementation of effective treatments of parental anxiety and child ADHD.

Evidence suggests that programs and interventions for the prevention of anxiety should target modifying early inhibition in children. Preliminary results from the Macquarie University Preschool Intervention Project, a longitudinal study of a brief parent education program for the reduction of inhibited temperament in preschool children, suggest that it may be possible to modify early risk for anxiety disorders [51]. For the purpose of prevention, addressing some temperament and behavioral factors, such as antisocial behavior, may reduce the risk of PTSD.

Although smoking is of interest in terms of its direct biological effects on organs and as a physiological trigger of anxiety, it may also be viewed as a modifiable behavior that could decrease one's vulnerability for anxiety disorders, namely in the context of panic disorder [47] and GAD [48]. Education about the impact of smoking on one's vulnerability to experience anxiety should be considered when developing educational and behavioral interventions.

Environmental or contextual risk factors

This review suggests that certain parental features such as over-control, overprotection, and parental psychopathology may be linked to childhood anxiety. Several studies in this review show that maternal anxiety, parental psychopathology, parenting styles, and social problems in conjunction with either neurodevelopmental, developmental, or temperamental features could have a synergistic or amplifying effect on the risk factors for anxiety [16,17,21,27,46]. Similarly, evidence from cross-sectional studies [16,52] not included in this review identifies parental separations and divorce, death of a family member, family conflict, and repeated moves of school to be greater among clinically anxious than non-anxious children.

This review identified the finding that social problems were the most consistent risk factor for panic attacks [46]. The authors explain that social problems may result from poor social skills and difficulties in peer relations, may be an expression of genetic transmission of vulnerability from parents to their children, or yet be the product of the gene-environment interaction.

A 10-year longitudinal study conducted in Germany [24] reported that the risk of panic attacks deteriorating into a panic disorder is higher in individuals NOT seeking help. Suggestions include early interventions in individuals experiencing high levels of panic symptoms to prevent later exacerbation/development of panic pathology.

A study in this review identified chronic environmental stressors such as poverty as a strong predictor of PTSD [27]. Although social factors have been overlooked in the studies of risk factors of anxiety disorders, limited evidence suggests that low socio-economic status plays central role in creating psychological distress and that these populations have greater odds of being classified as having anxiety disorders [53,54]. Some suggest that anxiety may be a reaction to severe environmental deficits, not a psychiatric disorder [54]. Researchers commonly recommend that prevention, assessment, and interventions for anxiety should be targeted at the environmental level to improve conditions of chronic adversity to reduce risk of anxiety disorders [27,53].

The role that parents and family environment play in the development of anxiety is a complex issue and is not yet well understood. Some researchers suggest that family factors may not play as large a role in the development of anxiety disorders as they do in other forms of psychopathology [51,55]. This lack of consistency in effect may be due to the poor operationalization of family quality and the wide variety and weak psychometric properties of many measures used in this type of research [51].

Stress-related risk factors

Types of stressful life events, such as a severe loss or a severe danger, have been long identified as "causal agents" associated with the onset of anxiety disorders [56,57]. This review confirms that life events have impact on the development of anxiety disorders, particularly on OCD and PTSD in adolescents and young adults [21,38]. A dose-response effect suggests that a new violence can be strong enough to cause PTSD in those who were resilient prior to a traumatic event. From another perspective, Koenen et al., [27] suggest that adequate adaptive processing helps regulate reaction to traumatic events. However, low IQ and antisocial behavior can disrupt adaptive processing and increase the risk of PTSD following the exposure.

In the context of traumatic stress, Milan et al. [29] underline the importance of viewing the emergence of PTSD symptoms themselves as coming into play and interact with both intra-individual vulnerability factors and environmental stressors. In other words, over time, PTSD symptoms themselves can become a vulnerability factor and contribute to the generation of environmental stressors such as violence exposure, hence exacerbating or maintaining PTSD.

Breslau et al., [33] found that severe maltreatment during childhood was a significant predictor of PTSD in adulthood. Moderate maltreatment, experienced by the majority of adult trauma victims maltreated as children, was not statistically distinguishable from absence of childhood maltreatment. The authors suggested that further investigation into the role of moderate maltreatment as a risk factor for PTSD necessitated further study.

This being said, one study [31] in this review suggests that PTSD is not limited to being a response to overwhelming trauma, and that non-traumatic life events can be associated with PTSD symptoms as

commonly as traumatic life events. The authors explain that there is no distinction between traumatic versus non-traumatic negative life events in causing PTSD symptoms; rather, it is the total exposure to negative life events that is related to the number and severity of PTSD symptoms as well as an individual's impairment in functioning. In essence, an accumulation of negative, but non-traumatic, life events can be a risk factor for the development of PTSD symptoms. Such symptoms form a continuum of severity with minor stress symptoms at one end and severe PTSD at the other.

Clinicians identifying populations at risk of developing PTSD as well as those offering treatment and developing programs should thus not only take into consideration prior experiences of overwhelming and life-threatening trauma but also be sensitive to a cumulative history of non-traumatic albeit negative life events. Identification and interventions should also be sensitive to the notion of a continuum of severity with everyday symptoms of stress at one end and severe PTSD symptomatic criteria at the other.

Limitations

This review has several limitations. Results from systematic reviews were not included due to the lack of homogeneity of operationalization of anxiety disorders as per DSM nomenclature. Focus on anxiety disorders in general limits the ability to retrieve specific differences across these disorders. Moreover, focus on general population does not permit to generalize findings to specific population groups, such as war veterans, post-partum women and others. We have not explored comorbidities common for anxiety disorders, such as depression, and also between different types of anxiety disorders.

Conclusion

Childhood has been identified as a critical period for the onset of anxiety disorders and PTSD. Children of parents with psychopathology, who demonstrate behavioral inhibition, who demonstrate antisocial behavior, who have low IQ, or who live in chronically adverse conditions such as poverty, are at risk of developing anxiety disorders. A history of juvenile disorders, severe maltreatment, poor quality of parental care, as well as symptoms of PTSD themselves, can increase the likelihood of PTSD. PTSD symptoms may develop in response to traumatic/life threatening events as well to the accumulation of stressful but non-traumatic events. Successful prevention of anxiety disorders and PTSD should focus on modifiable behavioral, environmental, and stress-related risk factors.

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