Understanding Traumatic Stress Following Myocardial Infarction: A Systematic Review

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Declaration

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The Complexities of Studying PTSD in Relation to Myocardial Infarction: A Review of the Evidence

Abstract

Research on the development of post-traumatic stress disorder (PTSD) following myocardial infarction (MI) is a relatively new area of scholarship that has attracted an increasing number of studies. The current review highlights that there is a lack of consistent and sound methodology within the existing literature. The presence of interrelated and overlapping factors such as unique symptomology and psychiatric comorbidities adds further complexity. Future studies may consider looking at the protective factors and long-term impacts of post-MI PTSD, as well as delayed-onset PTSD, to facilitate evidence-based preventive approaches and interventions.

Literature on the topic of traumatic stress routinely revolves around trauma-prone populations, such as veterans (Institute of Medicine, 2014), as well as in specific contexts, e.g., sexual assaults (Kline et al., 2018). However, the literature on traumatic stress in those who have experienced a life-threatening traumatic medical event is relatively new, of which there is a growing interest regarding the development of traumatic stress in individuals with cardiovascular diseases (Akosile et al., 2018; Spindler & Pedersen, 2005), particularly myocardial infarctions (Gander & von Känel, 2006; Vilchinsky et al., 2017). This review first looks at the broad literature surrounding the psychological sequelae of cardiac events, and following that, a critical examination and synthesis of the literature regarding the occurrence of traumatic stress in relation to myocardial infarction.

Psychological symptoms may develop in individuals following trauma exposure. When these symptoms persist over an extended period, an individual may be diagnosed with acute stress disorder (ASD) or post-traumatic stress disorder (PTSD). These trauma-related disorders are classified under the category of trauma and stress-related disorders in the DSM-5, separate from the anxiety disorders category where they were listed in previous versions of the DSM; this relocation signalled a conceptual difference from anxiety disorders (Zoellner et al., 2013). Notably, the trauma and stress-related category of disorders is also unique from other psychiatric disorders, in that an external stressor is a precondition (Pai et al., 2017). ASD describes an intense, unpleasant, and dysfunctional reaction that occurs shortly after an overwhelming traumatic experience and lasting up to one month. When symptoms persist longer than one month, a person may be diagnosed with PTSD. Manifestations of PTSD typically include symptoms occurring under four categories – intrusions, persistent avoidance, negative alterations in cognition and mood, and alterations in marked arousal/reactivity (American Psychiatric Association, 2013). Both ASD and PTSD bring about profound psychological distress to individuals experiencing them (e.g., increased

reactivity, and subsequent over-reactivity to environmental stimuli, as well as inability to shut off stress responses) and have far-reaching and wide-ranging negative consequences not just psychologically but also physically, where an increased risk of diseases has been reported (McFarlane, 2010).

The World Health Organization estimates the global lifetime prevalence of PTSD to be 3.9% (Koenen et al., 2017). In the United States and Canada, this figure has been reported to range from 6.1 to 9.2% (Duckers et al., 2016; Goldstein et al., 2016). Similarly, figures in Australia has been estimated to be between 5 to 10%, with 12-month prevalence at 6.4% (Phoenix Australia, 2013). Estimates of PTSD vary substantially, with greater risks following interpersonal traumatic events (e.g., sexual assault), as well as higher occurrences among developed countries where access to treatment (and thus diagnosis) is higher (Kessler et al., 2017; Liu et al., 2017).

Cardiovascular Diseases and Mental Health

Cardiovascular diseases (CVDs) are the leading cause of global health loss, with close to 18 million CVD-related deaths worldwide each year (World Health Organization, 2017), accounting for 31% of all deaths. This also makes it the leading cause of deaths worldwide. There are more than 400 million individuals currently living with CVDs – a figure that is projected to rise owing to an ageing population, rising obesity levels, and advances in life-extending treatments (Roth et al., 2017; World Health Organization, 2017).

CVD is an umbrella term for various diseases of the heart and blood vessels, which are commonly grouped under the banner of ‘heart disease’. These include coronary heart disease (CHD), such as myocardial infarction (MI), stroke, congenital heart disease, and heart failure (Mendis et al., 2011). CVDs can occur throughout the lifespan, such as congenital heart disease, a common form of birth defect characterised by malformations of heart

structures, as well as coronary heart disease (CHD), caused by a build-up of plague inside the artery walls and usually related to poor health behaviours (Mendis et al., 2011; World Heart Federation, 2017).

There appears to be a strong association between CVD and poorer mental health, with researchers identifying correlations between cardiac events and psychiatric disorders (De Hert et al., 2018; Gale et al., 2014; Kumar & Nayak, 2017). Anxiety and depression are two common mental health disorders with established bidirectional relationships with CVD, serving as risk factors as well as consequences of poor cardiac health (Cohen et al., 2015; Tully et al., 2016; Vogelzangs et al., 2010).

Anxiety has been associated with an increased prevalence of CVD (Tully et al., 2016), with some studies (Vogelzangs et al., 2010) documenting as high as a 3-fold increase in CVD incidence, and with varying rates of onset for different types of CVD, such as stroke and heart failure (Emdin et al., 2016). A meta-analysis found that anxiety is not just associated with an elevated risk of CVD, but also as a risk factor, it is independent and comparable in magnitude to traditional risk factors such as smoking and hypertension (Batelaan et al., 2016). Anxiety prevalence is also greater in the CVD population and has been linked to poor cardiovascular outcomes in those with existing or prior CVD, including higher risk of recurrent cardiac events and cardiac-related mortality, as well as increased symptom severity and healthcare utilisation (Celano et al., 2015; Emdin et al., 2016; Rutledge et al., 2013; Tully et al., 2016).

Likewise, depression is a strong predictor for CHD as well as cardiac-related mortality, and rehospitalisation with the onset of depression is also higher among those with CHD, consequently leading to poor prognoses (Cohen et al., 2015; Gale et al., 2014; Kumar & Nayak, 2017). For example, a systematic review found that around one-third of inpatients

developed depression following cardiac arrest (Wilder Schaaf et al., 2013). Furthermore, anxiety and depression also appear to be interrelated in their relationship with CVD, where the increased prevalence of CHD in relation to depression may be explained by comorbid anxiety disorders (Vogelzangs et al., 2010).

Depression and anxiety aside, PTSD is another area where an association with CVD can be observed – exposure to traumatic events, independent of PTSD symptom severity and diagnosis, is linked to an increased risk for incident CVD (Akosile et al., 2018; Burg & Soufer, 2016; Cohen et al., 2015). Poor cardiovascular health also increases the onset of PTSD (Edmondson et al., 2011; Vilchinsky et al., 2017); PTSD development has been observed in different forms of CVD, such as congenital heart disease in adults (Deng et al., 2016), cardiac arrest (Wilder et al., 2013) and myocardial infarction (Kumar & Nayak, 2017).

Myocardial Infarction

Across the broad spectrum of heart disease, CHD is the most prevalent, with myocardial infarctions (MI) being the most common type of CHD. In 2015, more than 7 million cases of MI were documented worldwide (Roth et al., 2017). In the United States alone, more than 8 million American adults (aged 20 years and above) have experienced an MI in their lifetime, with annual mortality rates of more than 300,000 documented (Virani et al., 2020); incidence data suggests that a heart attack occurs approximately every 40 seconds in the US. Within the UK, an individual is hospitalised for an MI every five minutes, and more than 200,000 hospital visits annually are related to an MI (British Heart Foundation, 2020). Additionally, close to 85% of all cardiac-related deaths worldwide are due to MI (i.e., “heart attack”) or stroke, with than half of this, approximately 8.93 million deaths, linked to MI (Roth & Geleijnse, 2018; World Health Organization, 2017).

MI is an acute coronary syndrome (ACS) which occurs when there is a sudden blockage of blood supply to the heart causing death of heart tissue; a sharp discomfort is usually felt in the centre of the chest with other symptoms being light-headedness and breathlessness (Thygesen et al., 2018). There are two main types of MI, which are differentiated by the extent of damage (blockage) to the heart – an 'ST-elevation myocardial infarction' (STEMI) and a 'non-ST-elevation myocardial infarction' (NSTEMI). A STEMI occurs when an artery is completely obstructed, whereas obstructions in NSTEMI are partial or temporary; these are distinguishable through differences in electrocardiogram (ECG) readings (American College of Cardiology, 2013). Unstable angina (UA) is another type of ACS, which is generally regarded to be similar to NSTEMI in several aspects yet without the defining feature of MI, which is the occurrence of myocardial necrosis, or heart cell death. If left untreated, UA typically leads to MI (Sheridan & Crossman, 2002). However, a recent large prospective study on NSTEMI and UA suggested substantial differences between these conditions, with UA presenting to be lower in rates of incidence and mortality, while still functioning as a risk factor for future MI (Puelacher et al., 2019).

While an MI is a medical emergency, survival rates have increased tremendously – for example, MI mortality rates over a 35-period from 1980 to 2015 in Australia fell by 86% from 204 to 28 deaths per 100 000 population (Australian Institute of Health Welfare, 2017). In the UK, the British Heart Foundation (2020) reports that in 2020, 70% of heart attacks are survivable, as compared to 30% in the 1960s. Such improvements in survival rates have been attributed to the greater understanding of cardiac health, risk factors and symptoms, as well as increased availability of treatments and medications (Luepker, 2016). However, without timely administration of these treatments and medications, an MI can cause irreversible damage to the heart and possible death (Fogoros 2019; Solomon et al., 2005). Furthermore, complications arising from the MI may occur, and result in fatalities – a common

complication is a sudden cardiac arrest, which is when the heart malfunctions and ceases to beat. Cardiac arrest is largely fatal without immediate treatment, and has an elevated risk of occurring due to prior heart damage (e.g., following an MI) (Hubbard, 2003; Solomon et al., 2005). Other complications include congestive heart failure and cardiogenic shock, both of which are time-specific cardiac events involving the impaired pumping function of the heart muscles because of the MI (Hubbard, 2003).

Traumatic Stress and MI

A growing body of literature has identified MI as a traumatic and distressing event that leads to the onset of PTSD, and consequently as a form of psychological complication among individuals post-MI (e.g., Kumar & Nayak, 2017). Previous studies looking at the prevalence of PTSD following an MI reported a substantial variation in figures, ranging from 4.1% (Roberge et al., 2010), 4.4% (Fortin, Dupuis, Marchand, & Bianca, 2013a), and upwards from 22% (Shemesh et al., 2006) and 26.7% (Malinauskaite et al., 2017). These studies suggest a large variability in prevalence rates of MI-induced PTSD, which has also been supported by several reviews – for example, Kumar and Nayak (2017) reported rates ranging from 4% to 25%, whereas Vilchinsky et al. (2017) found rates of between 3% to 21%. In a meta-analysis of 24 studies involving patients who had an ACS (MI or unstable angina), a 12% overall prevalence of PTSD was identified, with individual study estimates ranging from 0% to 32% (Edmondson, Shaffer, et al., 2012).

Upon closer examination of the literature, it appears that methodological differences and inconsistencies are a probable reason behind such variations in prevalence. Vilchinsky et al. (2017) posited that the timing of assessment and the type of diagnostic instrument utilised may account for the reported variations. In terms of diagnostic instruments, studies utilising structured interviews tend to exhibit lower rates of PTSD prevalence than those using self-

report screening questionnaires for diagnosing PTSD. Fortin et al., (2013a) highlighted that utilising a structured interview is more restrictive in diagnosing PTSD, thus leading to low diagnoses of PTSD, as compared to self-report screening questionnaires. For example, Fortin et al., (2013a) and Roberge et al., (2010) utilised the Structured Clinical Interview-PTSD (SCID-PTSD) and found estimates of 4.4% and 4.1% respectively, whereas Shemesh et al., (2006) used a self-report screening questionnaire (the Impact of Event Scale [IES]), which resulted in high PTSD prevalence estimates of 22%, as did Malinauskaite et al. (2017), who used the Post Traumatic Stress-Scale (PTS-Scale) and observed 26.6% prevalence. Likewise, diagnostic instrument was regarded as a source of heterogeneity in prevalence estimates in a meta-analysis of ACS-induced PTSD, with PTSD rates of 16% in studies that utilised self-report screening questionnaires, and 4% in studies utilising clinical interviews (Edmondson, Shaffer, et al., 2012).

Longitudinal data has illustrated the role of the timing of assessment in the heterogeneity of PTSD development rates. In an 8-year study of posttraumatic stress symptoms following MI (Ginzburg & Ein-Dor, 2011), it was found that 21.4% of participants exhibited posttraumatic stress symptoms within a week following the MI, 17.3% at seven months post-MI, and 13.8% at eight years post-MI. In another longitudinal study (Castilla & Vázquez, 2011), PTSD prevalence measured at two to three days post-MI, five months, and 13 months yielded estimates of 1.4%, 11.1%, and 3.1 % respectively. While these studies showed that estimates of PTSD differed depending on the timing of assessment, others found little changes in PTSD estimates over time, such as 12 months (12.2%) and 36 months (12.8%) post-MI (Wikman et al., 2008). Additionally, a meta-analysis of PTSD prevalence (Edmondson, Richardson, et al., 2012) post-MI found no relation between timing of assessment and prevalence rates. Rather, these studies highlighted the persistence of MI-related stress symptoms, as well as the ambiguous nature of symptom trajectory and severity.

For example, Abbas et al. (2009) found that two-thirds of patients still experienced symptoms after two years post-MI. However, some studies (Ginzburg & Ein-Dor, 2011; Wasson et al., 2014) have illustrated that symptoms may be short-lived and decreased over time. Importantly, these studies suggest that beyond methodological differences, there are several other possibilities affecting post-MI PTSD development, specifically, the subjective experience of a MI and the role of personality of patients.

Role of Subjective Experiences

Studies have shown that the subjective experiences of MI patients influenced the development of PTSD and its severity. These studies (Chung et al., 2011; Fortin et al., 2013a, 2013b; Roberge et al., 2010) highlighted that MI, while a life-threatening event, may be subjectively perceved by some patients to be a less intense threat than some other life events (e.g., being in a car accident), and for others, not a traumatic event itself. In a study of the fear of dying and its relationship with PTSD occurrence (Malinauskaite et al., 2017), it was found that post-ACS patients without such fear tend not to develop PTSD symptoms, and those with a fear of dying associated with MI predicted PTSD development, as well as greater psychological difficulties in responding to, and coping with the cardiac event. This suggests that the subjective element in patients' experiences of medical events, affects the onset and subsequent severity of mental health disorders.

Furthermore, personality traits has been documented to influence the degree of subjective element in patients' experiences, and consequently the development of PTSD. Chung et al. (2011) illustrated that neuroticism was associated with a greater liklihood of PTSD and general psychological distress, and functioned as a risk factor for post-MI PTSD – patients with high neuroticism heightened the impact of stressful events (e.g., MI) and this lead to subsequent psychiatric symptoms, as well as elevated symptom severity. The authors

also found that psychological distress brought about by neuroticism occured through the problem-focused coping, which involved attempts to change the situation. This form of coping is generally regarded as a protective coping strategy, however neurotic traits may lead to increased burden felt by patients when problem-focused coping was endorsed, through inflating the importance of such coping strategies, and the consequences of not following the (i.e., the magnification of negativity). Similarly, research on this area identified that neuroticism contributed directly, and significantly to PTSD risk (Pedersen et al., 2003).

Unique Features of Post-MI PTSD

Importantly, there appears to be unique characteristics present in the experience of MI-induced PTSD, as well as in the manifestations of symptoms. Fiat et al. (2018) conceptualised a future-oriented element of cardiac-related PTSD alongside past-related elements that are indicative of 'traditional' PTSD; this future-oriented nature is expressed by the concept of 'fear of illness progression' (FoP). Fiat et al. describe FoP as a fear that one's illness will worsen over time and this serves as a stressor for patients who have experienced a life-threatening illness. This fear affects how PTSD symptoms are displayed, particularly towards appointments with cardiologists – such as avoiding follow-up visits with cardiologists for fear of the possibility that illness has progressed or viewing cardiologists as painful reminders of the illness, as well as other future-oriented concerns (Fait et al., 2018; Vilchinsky et al., 2017). Moreover, Edmondson et al. (2012) reported that among hospitalised ACS patients, those having cardiac-induced PTSD had significantly elevated optimistic bias in MI risk perceptions; this finding was attributed to the avoidance and numbing symptomology of PTSD, which lowered patients' risk perceptions of future morbidity and mortality.

Additionally, the symptom profiles of PTSD, (namely the symptoms of intrusion, avoidance and arousal), varies and differ from each other; studies (e.g., Edmondson et al., 2011; Roberge et al., 2010) have found that intrusion symptoms (i.e., presence of unwanted thoughts and images related to the traumatic event, such as nightmares and flashbacks) were elevated and featured more prominently and chronically than other symptom clusters (avoidance, negative cognitions/mood and arousal) in patients with MI-induced PTSD. Intrusion symptoms also functioned as a risk factor for major adverse cardiac events, as well as mortality. However, in a longitudinal study of post-MI PTSD symptoms (Abbas et al., 2009), it was found that arousal symptoms (e.g., hypervigilance to environmental cues and difficulties in concentrating) were most resistant to change. The authors of that study suggested that the maintenance of this cluster of symptoms may be attributed to the overlap of physiological alternations subsequent to negative affect, which can be depression occurring as a comorbidity of PTSD, or as a generalised non-PTSD specific response to a difficult medical event (such as an MI). Nevertheless, researchers have posited that given the source of trauma (and subsequent threat) is the heart, and thus internal, and coupled with the long-term impact of the MI such as the medical obligations (e.g., medications and hospital check-ups) as well as the physical symptoms, it would be impossible to separate the individual and the threat (Abbas et al., 2009; Fait et al., 2018). As such, this may be a probable explanation towards the maintenance of different PTSD symptom clusters among post-MI patients.

The development of PTSD post-MI has also been found to be associated with depressive symptomatology; it has been argued that PTSD is a predisposing factor for later development of depression, which itself (depression) may also contribute to subsequent post-MI PTSD (Fortin et al., 2013b; Roberge et al., 2010). In this regard, this bidirectional relationship reflects the comorbidity of depression and PTSD following MI, as well as the

possible overlapping of symptoms inherent in both disorders. For example, Ginzburg (2006) found that initial stress reactions of intrusion and hyperarousal symptoms following MI were associated with both disorders (PTSD and depression) independently and comorbidly. The author of that study attributed this to a link between depression and loss – an MI brings about many losses, such as loss of health, functioning etc.

Shortcomings of Existing Research

While the increasing number of studies on MI-related traumatic stress are contributing to the fledgling literature on this topic, there continue to be notable shortcomings. Reviews on traumatic stress following MI, and broadly ACS and cardiac diseases, are warranted for cataloguing and synthesising the studies related to this topic. A key observation of studies to date is the lack of sound and consistent methodology. For example, the noticeable variations in clinical and statistical outcomes of MI-related traumatic stress appear to be attributed to a function of measurement, specifically the assessments of traumatic stress. Numerous individual studies have expressed the utilisation, and for some the reliance on self-reported outcome measurements, as a recurring methodological limitation that affected the estimations of traumatic stress following cardiac events; and with further acknowledgements that clinical interviews might have led to different results, often lower figures (Ginzburg & Ein-Dor, 2011; Pedersen et al., 2003; Shemesh et al., 2006; Wikman et al., 2008). This variation in prevalence has also been documented in previous reviews of this topic (e.g., Gander & von Kanel, 2006; Vilchinsky et al., 2017). Gander & von Kanel (2006), in their critical review of 31 studies on PTSD following MI, posited that the low figures derived from clinical interviews may be related to participants choosing not to be interviewed to avoid reminders of the traumatic event (i.e., their heart attack). Self-report measures are also subjected to respondent biases, which may lead to inaccurate figures (Tellis & Chandrasekaran, 2010). Whatever the reasons may be, the differential figures derived from different assessment

formats are not restricted to cardiac-induced traumatic stress. For example, existing studies on PTSD diagnostic instruments found that estimations for the development of PTSD varied according to the diagnostic instrument utilised in both cardiac and cancer patients – 29.2% when assessed using the posttraumatic symptom scale-10 item (PTSS-10), 7.6% when assessed using the Impact of Event Scale – Revised (IES-R),and 4.8% when assessed using a Structured Clinical Interview for Diagnosis (SCID) (Einsle et al., 2012).

In addition to the differences in traumatic stress assessments, it has also been expressed that a cross-sectional study design do not fully capture the trajectory and severity of MI-related traumatic stress; this surfaced as a limitation for many studies and expressed as a shortcoming of existing research (Fiat et al., 2018; Spindler & Pedersen, 2005). The subjective element in the presentation of stress symptoms as well as in the perceived severity of traumatic events by patients meant that a singular assessment of traumatic stress may not be adequate to understand fully the nature of MI and other cardiac-related traumatic stress (Pedersen & Denollet, 2004; Wasson et al., 2014).

Small sample sizes and the limited statistical power of studies have been identified as key concerns affecting the representativeness of samples and the generalisability of findings. For example, Shemesh et al (2006) expressed that a small sample of 65 participants was a key limitation in their study on MI-related PTSD. Small sample sizes and low statistical power was consistently identified and acknowledged across many other individual studies (Ginzburg, 2006; Ginzburg & Ein-Dor, 2011; Pedersen & Denollet, 2004; Pedersen et al., 2003; Wasson et al., 2014). Several reviews (e.g., Edmondson et al., 2012; Spindler & Pedersen, 2005) have also routinely highlighted the issue of a lack of sufficiently powered studies as well as relatively small sample sizes in existing studies – one potential consequence of this issue is the inaccuracy of prevalence estimates (i.e., inflated or deflated figures). For example, a meta-analysis of 24 studies by Edmondson et al. (2012) found that

no study had been sufficiently powered to provide conclusive evidence of the relationship between ACS-induced PTSD and adverse clinical outcomes. A contributing factor to this issue may be the occurrence of high attrition and low participation rates reflected in some studies (e.g., Fortin et al., 2013a; Ginzburg, 2006). Researchers posited that this lack of participatory interest and loss to follow-up to be related to an outward display of avoidance behaviours – studies served to be a reminder of the traumatic event that was avoided (Chung, Berger & Rudd, 2008; Fortin et al., 2013a). In addition to the size of the sample, it is also important to consider the composition of the study samples. With regard to studies on this topic, demographic characteristics – specifically gender and ethnicity, were not evenly balanced, which suggests limited representativeness and generalisability of findings. Participants were often predominantly Caucasian and male, as acknowledged by several studies (e.g., Edmondson et al., 2012; Fortin et al., 2013b; Wikman et al., 2008). In this regard, a study of gender differences in individuals after MI found that although the trajectory of stress were similar between both genders, higher levels of perceived stress were recorded in women than men in the first year of recovery (Xu et al., 2017).

The presence of confounding variables is another key limitation observed in the current literature. For example, the occurrence of other traumatic events experienced by individuals prior to the MI may affect the development of traumatic symptoms (i.e., serving as a predisposing factor), its severity or maintenance (Chung et al., 2011; Spindler & Pedersen, 2005). In this regard, there should also be a differentiation between participants experiencing an MI for the first time, versus those who have experienced multiple MI including recurrent episodes (Vilchinsky et al., 2017). Malinauskaite et al. (2017) identified traditional cardiovascular risk factors and attendance of cardiac rehabilitation as potential confounders, which were not controlled for in their study. Likewise, Abbas et al. (2009) did not assess for established longitudinal predictors of PTSD, such as co-morbid depression and

stressors, which served as potential confounders; although not the focus of their study, the authors acknowledged this to be a major drawback of their study.

Future Directions

The existing literature on MI-induced traumatic stress, despite its varying shortcomings, has illustrated the complexities, and more importantly, the depth of this topic, which has yet many unknowns. One such area is with individuals who exhibit traumatic stress symptoms without meeting diagnostic criteria for any traumatic stress-related disorders. A possible contributing factor to this may be delayed-onset PTSD, where symptoms may not present fully, at the time of assessment but manifest subsequently (Abbas et al., 2009; Fortin et al., 2013a). The literature on delayed-onset PTSD is relatively scarce and inconsistent, although some suggest its prevalence appears to be higher in military populations (Andrews et al., 2007). A 2013 study linked delayed-onset PTSD with current and cumulative stress, as well as mild traumatic brain injuries being the traumatic event (Bryant et al., 2013). Furthermore, the subjective element in individuals' experiences means that an MI can be subjectively perceived to be non-traumatic at time of assessment, and thus despite the presence of PTSD-like symptoms, a diagnosis is not warranted, Future research should consider the development of delayed-onset PTSD in individuals with MI, such as via longitudinal (prospective) study designs that incorporate multiple assessments to understand the natural course of PTSD following cardiac events (Spindler & Pedersen, 2005).

In relation to uncovering the course of post-MI PTSD, it has been proposed that future studies should be directed towards understanding the long-term impacts of PTSD on the daily functioning and health of post-MI patients, as well as consequences on health-related behaviours and compliance (Fortin et al., 2013a; Pedersen et al., 2003)

While much research has been devoted to understanding and addressing the risk factors for the onset of post-MI traumatic stress, such as personality traits (Chung et al., 2011), subjective perceptions (Ginzburg & Ein-Dor, 2011), and psychological comorbidity (Pedersen et al., 2003), less attention has been cast on protective factors. In a review of risk and protective factors for psychological symptoms in people with heart disease, Greenman et al. illustrated an imbalance of research focus (21 studies on vulnerabilities and none specifically on protective elements), particularly regarding posttraumatic stress, where risk factors dominated the literature in this population (Greenman et al., 2018). Although in psychological literature the presence of social support has been regarded as a key element in buffering against the onset of psychiatric disorders such as depression (Brinker & Cheruvu, 2017), narrowing the research focus to protective factors specifically pertaining to MI-induced traumatic stress is necessary to inform preventive approaches and interventions. In this regard, there is a dearth of research surrounding the treatment of traumatic stress following MI (Edmondson et al., 2012). A phase 1 randomised controlled trial of cognitive behavioural therapy that utilised an imaginal exposure component reported small-to-moderate symptom reduction, although this study was not sufficiently powered (Shemesh et al., 2011). Likewise, an exploratory study to address medication non-adherence related to post-MI PTSD utilised trauma-focussed CBT as an intervention and yielded modest improvements (Shemesh et al., 2006).

The unique characteristics of MI-induced traumatic stress differs from conventional traumatic stress-related disorders, such as the internal location of the threat as compared to the usual external location (Vilchinsky et al., 2017), suggesting that treatment approaches should be modified accordingly. In this regard, the comorbid and risk status of depression with PTSD in MI patients (e.g., Ginzburg, 2006) puts forward that depression should also be targeted during screening processes, as well as in interventions to improve rehabilitation

outcomes. Additionally, the presence of a fear of dying in MI patients has been linked to the development of posttraumatic stress symptoms and thus, it is likely to be useful that fear of dying is screened for in hospitals and targeted in interventions (e.g., via improving coping strategies) (Malinauskaite et al., 2017). Future studies may involve the development of randomised controlled trials (RCT) to determine an appropriate intervention for this population, and their effectiveness in improving both quality of life and cardiovascular outcomes (Edmondson et al., 2012; von Kanel et al., 2011). Notably, although psychotherapies are considered first-line approach to address PTSD (Karatzias et al., 2019; Reisman, 2016), outcome assessments of the two most widely referenced and studied evidence-based psychotherapies, cognitive processing therapy and prolonged exposure, are less than optimal. In a review of RCTs on PTSD psychotherapies, Steenkamp, Litz, Hoge, and Marmar (2015) found that patients often remain symptomatic even after treatment, with PTSD scores often remaining at or above the diagnostic threshold. The authors concluded that improvements to existing treatments and developments of novel treatment approaches are needed (Steenkamp et al., 2015) (Steenkamp, Litz, Hoge, & Marmar, 2015).

Summary

The current review summarises the state of the literature surrounding truamatic stress related to MI, which is the most prevalent cardiac condition globally. Notably, the current research highlights the discrepancies in the development of PTSD in MI patients, the methodological inconsistencies surrounding research on this topic, as well as the unique symptomatology apparent for post-MI PTSD compared to conventional PTSD. This review sheds light on the complexities of studying PTSD following MI, where there are many inter-related and overlapping factors involved, particularly with psychiatric comorbidity. There are still many unknowns and potential areas of interest to explore around this topic. What is certain is that individuals who develop MI-induced PTSD are likely to experience a detrimental quality of

life (e.g., Wasson et al., 2014), which can in part be attributed to the psychological symptoms inherent in PTSD. While it remains unclear which aspects of PTSD symptomatology are dominant following MI, its presence is likely to be both unpleasant and detrimental. Furthermore, affected individuals are also exposed to other negative consequences, such as developing an elevated risk of adverse cardiac event recurrence and mortality (Edmondson et al., 2012; von Kanel et al., 2011), as well as other physical comorbidities (McFarlane, 2010). In 2015, an estimated 7.29 million cases of heart attacks was documented globally (Roth et al., 2017). In the United States alone, the American Heart Association (2019) reports more than 600,000 new cases of MI and 200,000 recurrent cases annually (Benjamin et al., 2019). While treatments and survival outcomes have improved dramatically in recent decades, these figures illustrate that many individuals have the potential to develop traumatic stress symptoms post-MI. As such, greater resources and research is needed in this area, to facilitate the timely translation of research to policy and practice, and ultimately promote the ongoing wellbeing of this patient group.

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Patricia K. Kerig, Ph.D.
Editor
Journal of Traumatic Stress

October 1, 2020

Dear Dr Kerig:

I wish to submit an original research article entitled "Understanding traumatic stress following myocardial infarction: A systematic review" for consideration for publication in the *Journal of Traumatic Stress*.

I confirm that this work is original and has not been published elsewhere, nor is it currently under consideration for publication elsewhere. I have no conflicts of interest to disclose.

Please address all correspondence concerning this manuscript to me at [redacted]

Thank you for your consideration of this manuscript.

Sincerely,

[redacted]
Gregory Low

Understanding Traumatic Stress Following Myocardial Infarction: A Systematic Review

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Author Note

This manuscript has been prepared for submission to the *Journal of Traumatic Stress*. In line with the author guidelines for this journal (Appendix D), APA 7th edition formatting conventions have been adhered to for the research report component of this thesis. In its current form, the length of the manuscript, number of tables, and number of references is consistent with the thesis requirements. Before submitting the manuscript for publication, a number of actions will be taken to ensure it adheres to the journal requirements (30 page maximum length). These are as follows: reduce the word count, limit to one table, and reduce the number of citations.

The author has no known conflicts of interest to disclose. Correspondence concerning this article should be addressed to Gregory Low Wei Liang, University of Adelaide, SA 5005, AUSTRALIA. [redacted]

Abstract

More than one million Australians have some form of heart disease, with around half of those experiencing a myocardial infarction (MI) during their lifetime. The purpose of this systematic review is to summarise the literature on traumatic stress post-MI. This study aims to identify the different ways in which traumatic stress symptomatology is assessed and classified post-MI, and the implications this has for translation from research to policy and practice. A comprehensive search protocol, developed in collaboration with clinicians and a research librarian, was applied to six databases. This resulted in 3273 records identified for screening. The online tool Covidence was used for managing the study selection process, using predefined inclusion/exclusion criteria. A second reviewer independently screened a subset of the studies to assess the reliability of the inclusion/exclusion criteria. Following removal of duplicate records and further screening, 13 studies were identified and included in the review. Results suggest there is increasing evidence for the occurrence of traumatic stress post-MI. Detection of this condition is influenced by methodological differences, with clinical interviews measures identifying lower figures than self-report. Post-MI traumatic stress symptomatology presents atypical characteristics and chronicity, which poses important consequences for researchers, practitioners, and patients. While MI mortality rates are falling steadily, the increase in the number of survivors with traumatic stress requires timely translation of research to policy and practice, to promote the ongoing wellbeing of this patient group.

Experiences of traumatic stress are a common following exposure to life-threatening situations or events, such as armed combat, motor vehicle accidents, violent crimes, or natural disasters (Royal College of Psychiatrists, 2016). Post-traumatic stress disorder (PTSD) is commonly diagnosed in people who are exposed to such events that invoke strong feelings of distress. According to the World Health Organisation, the global lifetime prevalence of PTSD is estimated to be 3.9% (Koenen et al., 2017). In Australia, the lifetime prevalence of PTSD has been estimated to be between 5 to 10%, with 12-month prevalence at 6.4% (Phoenix Australia, 2013), which is one of the highest prevalence rates of PTSD globally (Duckers et al., 2016). A substantial proportion of the population are affected by PTSD – up to four million Australians (patients as well as their immediate families), are currently living with PTSD (Hilbrink et al., 2016).

The literature on traumatic stress and consequently PTSD typically focuses on trauma-prone populations, such as veterans (Institute of Medicine, 2014), as well as specific incidents (e.g., sexual assaults, motor vehicle accidents) (Kline et al., 2018). However, the literature on the development of traumatic stress in those who have experienced an invasive or otherwise traumatic medical event, specifically in relation to cardiac events, is relatively new. The topic of traumatic stress in relation to cardiac events was first discussed in a 1988 study of four patients who developed PTSD following myocardial infarction (Kutz et al., 1988). Following this landmark study, there has been a substantial increase in publications on this topic, as captured by several reviews (Edmondson, Richardson, et al., 2012; Singh et al., 2017; Vilchinsky et al., 2017). In addition, the understanding of traumatic stress has evolved substantially in recent decades, as indicated by multiple revisions in the diagnostic criteria of PTSD (Bovin et al., 2015). Taking into consideration the complexities of PTSD, this systematic review will explore PTSD in those who have experienced a traumatic cardiac event, specifically myocardial infarction (MI). The remainder of this introduction provides an

overview of PTSD, synthesises the literature surrounding the relationship between PTSD and cardiac health, with a focus on the occurrence of PTSD after MI, leading to the rationale and specific aims of this systematic review.

What is Post-Traumatic Stress Disorder?

PTSD is characterised by profound psychological distress and typically manifest in the form of intense feelings of fear and helplessness, pervasive negative thoughts as well as maladaptive behaviours consequent to trauma exposure (Phoenix Australia, 2013; Young, 2015). Historically, early accounts have been documented of maladaptive and detrimental symptoms present in soldiers and those afflicted by war (Chekroud et al., 2018; Crocq & Crocq, 2000). One such example is a study into the psychosomatic symptoms of soldiers during the American Civil War which resulted in a PTSD-like disorder known as Da Costa's syndrome (Bremner et al., 2020). Additionally, trauma research has been shaped by military conflicts, as evident by the emergence of informal terms such as "war or trauma neurosis", "shell shock", and "Vietnam syndrome", that were utilised by physicians treating and studying the disorder (Bremner et al., 2020; Chekroud et al., 2018). The inclusion of PTSD in the Diagnostic and Statistical Manual of Mental Disorders (DSM), introduced in the first edition of the DSM as "Gross Stress Reaction" and officially adopted in the third edition as a diagnostic category, signalled the increasing development of psychological distress among those affected by traumatic events, and consequently shaped the modern conceptualisation of these disorders (Crocq & Crocq, 2000; Pitman, 2013).

The fifth and latest edition of the DSM (DSM-5) categorises symptoms of PTSD under four dimensions, corresponding to Criteria B to E in the DSM-5 (Table 1) (American Psychiatric Association, 2013). These symptoms must persist for more than one month to facilitate a clinical diagnosis of PTSD, as well as cause functional impairment, and must be solely attributed to the traumatic event.

Psychological treatments are regarded as a first-line approach for addressing PTSD symptoms (Australian Psychological Society, 2018; Karatzias et al., 2019; Reisman, 2016). Empirically supported treatments include cognitive behavioural therapy (CBT), and its variants such as trauma-focussed CBT (Paintain & Cassidy, 2018), exposure-based therapies (e.g., prolonged exposure) (Morkved et al., 2014), as well as eye movement desensitisation and reprocessing (Chen et al., 2014). However, effective treatments for PTSD can only take place if the condition is detected, and thus a comprehensive assessment of symptoms is required. Assessment of PTSD typically involves clinician-administered and self-report symptom questionnaires (see Table 2), which serve to screen and diagnose PTSD, as well as track the effectiveness of treatments (Lancaster et al., 2016).

The Clinician-Administered PTSD Scale (CAPS) is one of the most common diagnostic tools for PTSD, having been studied and tested extensively and demonstrating excellent psychometric properties in a wide range of trauma populations (Weathers et al., 2018; Weathers et al., 2001). The CAPS-5 (Weathers et al., 2018), the latest version of the CAPS based on the DSM-5, is widely considered to be the ‘gold standard’ for the diagnosis of PTSD (Reisman, 2016; Watson, 2002). For screening instruments, the PTSD Checklist (PCL) and Primary Care PTSD (PC-PTSD) are the most widely utilised and have consistently presented with favourable performance characteristics as compared to other screening instruments. The PCL and PC-PTSD instruments also have high feasibility properties, being short and easy to administer for busy clinical settings (Brewin, 2005; Spoont et al., 2015).

Acute Stress Disorder

PTSD symptoms that persist for a month or less following trauma exposure are termed ‘Acute Stress Disorder’ (ASD), a diagnosis that was first introduced in the DSM-IV, which required PTSD symptoms to persist for at least two days to a month, and occurring within one month of trauma exposure (American Psychiatric Association, 1994; Bryant, 2017). In the DSM-5, this was revised to be at least three days in duration following trauma exposure (American Psychiatric Association, 2013). Subsequent studies across different trauma exposures found an association between the development of ASD and subsequent PTSD, with ASD severity being a strong predictor of PTSD (Hansen & Elklit, 2013; Pires & Maia, 2013). For example, Hansen and Elklit (2013) identified that ASD severity accounted for approximately 40% of PTSD severity variance.

However, the notion that ASD serves as a predictor for subsequent PTSD is debatable. The timing of the onset of symptoms is a key differentiator between ASD and PTSD. A diagnosis of ASD requires symptoms to occur within one month of the trauma exposure and to be contained to one month or less in duration. Several studies (Bryant, 2011; Bryant et al., 2015) illustrated ASD was at best, a modest predictor for PTSD, as not all who have PTSD have immediate post-traumatic symptoms that meet a diagnosis of ASD. These findings suggest that PTSD and ASD possessed a non-linear relationship, with future development of PTSD dependant on a magnitude of factors alongside, or independent of, the presence of ASD. Nevertheless, ASD possesses many overlapping and similar presentation of symptoms (e.g., intrusive and/or distressing memories, and hypervigilance), as well as sharing many of the same risk factors as PTSD (American Psychiatric Association, 2013; Bryant., 2018)

Cardiovascular Disease and Mental Health

One in 20 Australians live with some form of heart disease, also known as cardiovascular disease (CVD) (Australian Bureau of Statistics, 2018b). Coronary heart disease (CHD), such as angina and myocardial infarction (MI, commonly referred to as a "heart attack") constitute the leading cause of death in the country (Australian Bureau of Statistics, 2018a, 2018b). Although medical advances and public awareness have led to a steady decline in mortality rates (e.g., from about 295 per 100 000 population in 1981 to 55 per 100 000 population in 2018) , disease burden remains high in Australia at 14%, particularly among those aged 50 years and over, and especially in men (Australian Institute of Health and Welfare, 2019).

Researchers have identified correlations between cardiac events and psychiatric disorders (De Hert et al., 2018; Gale et al., 2014; Kumar & Nayak, 2017). Anxiety and depression are two common psychiatric disorders with established bidirectional relationships with CVD, serving as risk factors as well as consequences of poor cardiac health (Cohen et al., 2015; Tully et al., 2016; Vogelzangs et al., 2010). Additionally, anxiety and depression also appear to be interrelated with CVD. For example, the association between depression and cardiovascular disease may be partly accounted by anxiety disorders, with a greater prevalence of cardiovascular diseases occurring in individuals with comorbid depression and anxiety as well (Vogelzangs et al., 2010).

PTSD is another area where an association with CVD can be observed – exposure to traumatic events, independent of PTSD symptom severity and diagnosis, is linked to an increased risk for incident CVD (Akosile et al., 2018; Burg & Soufer, 2016; Cohen et al., 2015). PTSD development has also been observed in different forms of CVD, such as congenital heart disease in adults (Deng et al., 2016), cardiac arrest (Wilder et al., 2013) and myocardial infarction (Kumar & Nayak, 2017).

The main interest of this review is the development of PTSD after a cardiac event– this is termed cardiac disease-induced (CDI) PTSD. The specific cardiac event addressed in this review is myocardial infarction (MI).

Cardiac Disease-Induced PTSD – MI

CHD is a type of heart disease caused by a build-up of plague inside the artery walls (this leads to a complex pathological process known as atherosclerosis that develops over many years) (Mendis et al., 2011; World Heart Federation, 2017). Given the broad spectrum of cardiac diseases (Mendis et al., 2011), we have chosen to focus on MI as it is the most prevalent form of CHD in Australia. More than 500 000 Australians experience an MI in their lifetime, making it the single most common form of heart disease (ABS, 2018a; 2018b).

MI, is a type of acute coronary syndrome (ACS), which develops when the blood supply to the heart is obstructed, leading to myocardial necrosis (i.e., heart cell death); a sharp discomfort is usually felt in the centre of the chest with other symptoms being light-headedness, breathlessness, squeezing or aching sensation in the chest or arms that may spread to the neck, jaw or back (Thygesen et al., 2018). The two most common forms of MI are an ‘ST-elevation myocardial infarction’ (STEMI) that signifies complete blockage of an artery, or a ‘non-ST-elevation myocardial infarction (NSTEMI), where blockages are partial or temporary; these are distinguishable through observations using an electrocardiogram (American College of Cardiology, 2013). Unstable angina (UA), is a type of ACS similar to NSTEMI in several aspects such as pathophysiology, except for the occurrence of myocardial necrosis. If untreated, UA generally leads to MI (Sheridan & Crossman, 2002).

An increasing body of evidence has pinpointed MI to be a traumatic and distressing event for individuals who experienced it. Prior studies have identified the prevalence of

PTSD following MI to be ranging from 4.1% (Roberge et al., 2010) to upwards of 22% (Shemesh et al., 2006).

In-depth examination of the literature suggests that these variations in prevalence estimates may be attributable to methodological differences, notably inconsistencies in assessment and classification. For example, in a meta-analysis of 24 studies that looked at ACS-induced PTSD, estimates of 16% were reported in studies that utilised self-report screening questionnaires, as compared to 4% in studies utilising clinical interviews (Edmondson, Shaffer, et al., 2012). From this research, it appears that studies that used self-report screening questionnaires for diagnosing PTSD produced higher estimates of PTSD prevalence than those utilising structured interviews, which typically resulted in lower estimates of PTSD prevalence. The timing of assessment is another area of interest in understanding the heterogeneity of PTSD development rates. For example, an 8-year study of post-MI PTSD found that 21.4% of participants presented with symptoms in the first week after their MI, with 17.3% and 13.8% at 7 months and 8 years post-MI, respectively (Ginzburg & Ein-Dor, 2011), whereas a different meta-analysis illustrated that the timing of assessment was unrelated to prevalence estimates (Edmondson, Richardson, et al., 2012).

Notwithstanding the role of methodological differences in the detection of traumatic stress symptoms post-MI, there also appear to be other influences on the development of such stress symptoms. Prior studies have provided evidence that MI patients' subjective experiences and personality traits are also determining factors in the subsequent development of PTSD and its severity. For example, the subjective element in the perceived threat of an MI means that some patients may regard MI to be an intense traumatic event, whereas others may perceived it otherwise (Chung et al., 2011; Fortin et al., 2013a, 2013b; Roberge et al., 2010). As such, individuals who perceive an experience of MI as non-threatening may not develop any stress-related symptoms, while for thoese who percieve it as a threat, they may

have varying levels of MI-related stress symptoms. In terms of personality traits, a study identified that MI patients with a 'Type D' personality had a fourfold risk of meeting a PTSD diagnosis compared to non-Type D patients (Pedersen & Denollet, 2004). The Type D personality encompasses a combination of two personality traits, negative affectivity and social inhibition, and has been established as independent risk factor for CHD progression, as well as in patients with existing CHD, poor clinical and patient-reported outcomes (Kupper & Denollet, 2018).

Additionally, individual PTSD symptom clusters (see, Table 1) have been found to differ in severity and trajectory from each other. For example, intrusion symptoms were presented more noticeably and enduring than other symptom clusters in patients with post-MI PTSD (Edmondson et al., 2011). In the same study, these intrusion symptoms also served as a risk factor for major adverse cardiac events as well as mortality. However, another study (Abbas et al., 2009) found that arousal symptoms were most resistant to change, with authors of that study suggesting that this PTSD symptom cluster were least dependant on the trauma experience.

What has been covered thus far illustrates the complexities, and subsequent discrepancies surrounding the understanding of traumatic stress development following MI, where there are many inter-related and overlapping factors involved. It should be noted that not all stress-type symptoms related to the experience of MI and its treatment are necessarily best described as PTSD.

Rationale and Aims

More individuals are living post-MI due to improvements in health care related to the immediate event. However, poor outcomes are frequently observed in MI survivors, for example, elevated rates of PTSD diagnosis leading to an increased recurrent cardiovascular

risk, higher nonadherence to medications (Shemesh et al., 2006; Wasson et al., 2018), and elevated mortality (Edmondson, Richardson, et al., 2012; Edmondson & von Känel, 2017; Singh et al., 2017). While there exist some discrepancies in the prevalence rates for PTSD post-MI, rates are generally higher than in the general population (Edmondson, Richardson, et al., 2012; Fortin et al., 2013a; Kumar & Nayak, 2017). Therefore, the purpose of this study is to review and summarise the literature surrounding post-traumatic stress after MI, taking into consideration the diagnostic changes and evolving understanding of post-traumatic stress disorder (PTSD). Specifically, this study aims to identify:

i. The different ways that traumatic stress symptomatology is classified for individuals who exhibit symptoms following a myocardial infarction;
ii. The common clusters of psychological distress symptoms as reported by post-MI individuals;
iii. How traumatic stress symptoms are measured in this patient group;
iv. The implications of these issues for health care practitioners.

Method

Procedure

The current review was developed in consultation with evidence-based guidelines (Aromataris & Pearson, 2014; Aromataris & Riitano, 2014; Meline, 2006). The PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) Statement was utilised for documenting the search procedure (Moher et al., 2009). A research librarian from the School of Psychology was consulted for assistance in developing the search protocol, as well as refining search terms. A search protocol was customised for each database that incorporated search terms grouped into their respective database-specific key words and subject headings (see Appendix A for search protocol). Search terms were based on two

areas: traumatic stress (e.g., ‘post-traumatic stress’, ‘acute stress disorder’, ‘psychotrauma’) and MI (e.g., ‘heart attack’, ‘myocardial infarction’, ‘cardiac infarction’), and sourced from the respective databases and existing literature. Additionally, unstable angina (UA) and its related terms were included in the area of MI to ensure broad coverage of the literature.

Six electronic databases (*PubMed*, *Embase*, *PsycINFO*, *CINAHL*, *Cochrane Library*, and *PTSDPubs* – a PTSD and trauma focused database of publications worldwide) were searched to identify relevant studies relating to traumatic stress and MI. To ensure a thorough and extensive search process, multiple combinations of search terms were pre-tested and adjusted accordingly, and citation searching was conducted with included studies to ensure all relevant articles were identified. A series of pilot searches were conducted, and results discussed with a second reviewer (CB) to determine the feasibility and effectiveness, and refinement of the search protocol. The software program EndNote was utilised for cataloguing the search results.

The online software Covidence was used for the screening of articles in accordance with the eligibility criteria. Articles were eligible if they (1) enlisted adult participants (18 years and above), (2) reported a diagnosis of MI, following which, presentations of traumatic stress, (3) had a measure of traumatic stress, (4) included a statistical relationship between MI and traumatic stress, and (5) were published in a peer-reviewed journal (see Table 3. for more details). Additionally, this review examined articles published in the last 20 years from the search date; this is consistent with the documented intensification of research into this topic after the year 2000 (Edmondson, Richardson, et al., 2012). To ensure methodological consistency and specificity, articles were excluded if there was evidence of other forms of trauma (e.g., combat, sexual assault) or other cardiac (i.e., non-MI related) medical conditions, as well as unclear reporting of traumatic stress or MI diagnosis. Additionally, as the focus of this review is on MI, articles that used a classification of ACS or UA, were only

included if there was a clear distinction of MI present and studied (i.e., articles looking at UA only were excluded, as were articles that lacked a direct statistical analysis of MI and traumatic stress). Furthermore, articles that lacked clear directionality of either traumatic stress or MI were excluded, as were articles that did not report original data (e.g., reviews and meta-analyses).

A literature search was conducted on 1st February 2020 as detailed in Figure 1. Application of the search protocol yielded 3273 publications. One additional publication was added through citation searching of recent systematic reviews on this topic. Removal of duplicates (n = 1202) resulted in 2072 publications whose titles and abstracts were screened based on the eligibility criteria. Accordingly, this led to the retrieval of full text for 213 publications, and a further 1859 publications were removed. Additionally, two authors were contacted for missing articles; one was unable to provide because of copyright issues, and another did not respond, thus these were not included for screening. Upon reviewing the full texts (n = 213), a total of 46 articles were identified for inclusion into this current review. Reasons for exclusions were detailed, as listed in Figure 1. However, this number was deemed too large to meaningfully synthesise the results. Through discussions with the second reviewer (CB), the inclusion criteria were further limited to include only articles with reporting of the descriptions of traumatic stress symptoms (see Table 2). Additionally, it was observed that some of the preliminarily included publications utilised overlapping participants (i.e., reports on same sample); these studies were excluded as no new relevant information of interest (for this review) was reported. This led to an initial sample consisting of 15 publications for inclusion into this current review. The PRISMA flowchart (Figure 1) illustrates the selection procedure.

To assess the effectiveness and inter-rater reliability of the eligibility criteria, a second reviewer (CB) independently reviewed a subset of full-text articles (20% of the total, or 45

articles) as well as the initial sample of publications (n = 15). Through this process, two articles (Bluvstein, Moravchick, Sheps, Schreiber & Bloch, 2013; Caterino et al., 2018) were subjected to further discussion, which subsequently resulted in their exclusion (see Figure 1). The inter-rater reliability for the final selection of publications was 87%. Any additional discrepancies were resolved by consensus, with full agreement (100%) between both reviewers for the inclusion of 13 publications.

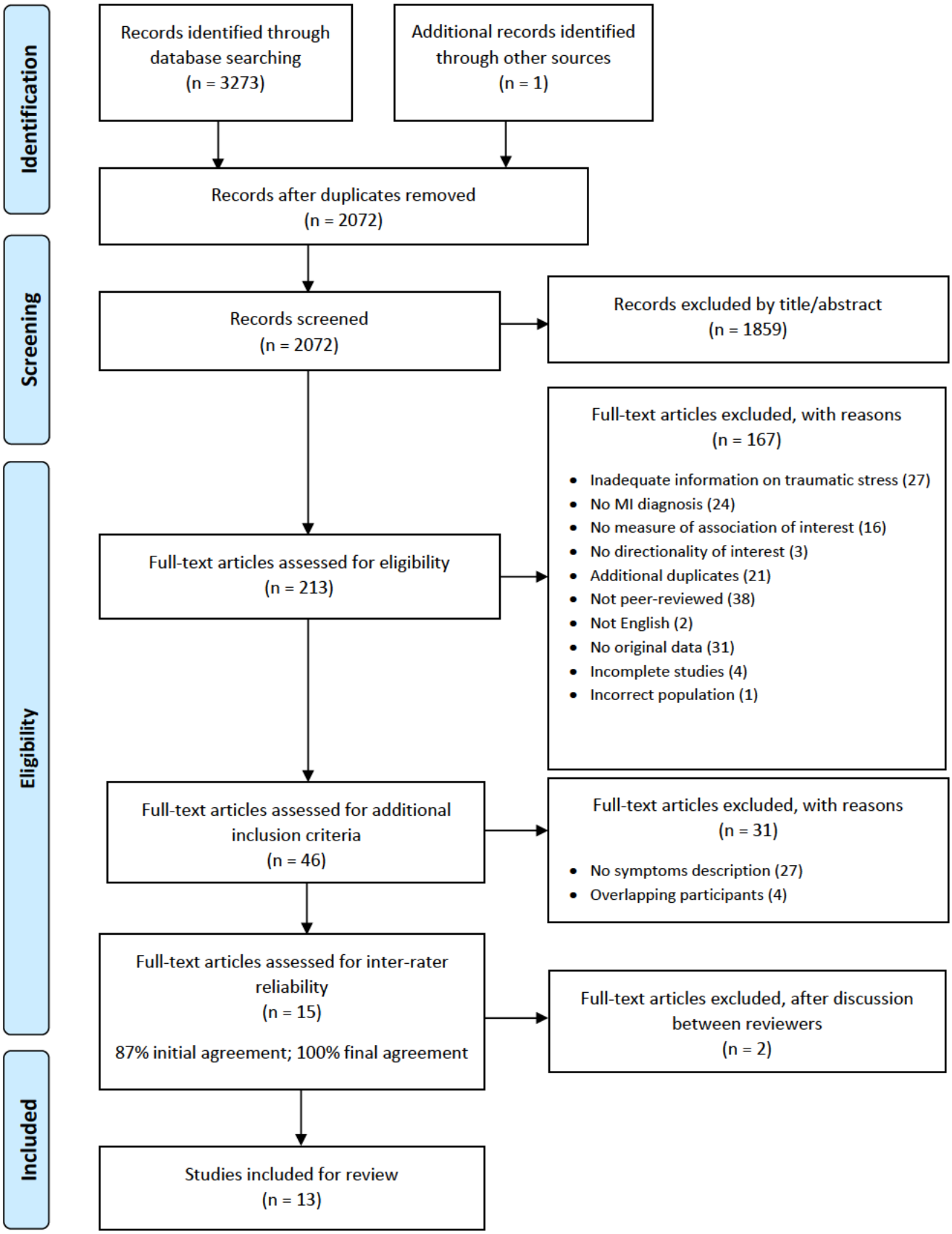

Figure 1. PRISMA flowchart of the selection procedure.

Data Analysis

A data extraction form was used to compile data for each included study. General and specific items of the form were developed to gather information relevant to the aims of the current review and for qualitative data synthesis of included studies. The items were discussed with the second reviewer (CB) for refinement and consensus. The data extraction form (see Appendix B) included: study characteristics; participants demographics; clinical and psychological characteristics; and association of interests.

Because of the heterogeneity in methodology and reporting styles across studies, it is necessary to conduct a critical appraisal of included studies to examine any flaws in study method or design that may contribute to the risk of bias, as well as gauge the validity of evidence. The 'National Institute of Health (NIH) Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies' (National Institutes of Health, 2014) was utilised to assess the methodological quality of included studies, with each individual study evaluated against fourteen criteria (Appendix C). Each criterion encompasses three distinct values with ratings of 'good', 'fair', and 'poor', however the tool does not compel a summative judgement of the quality of studies (NIH, 2014). The first reviewer conducted the critical appraisal of included studies, in discussion with the second reviewer (CB).

Results

Study and Participants Characteristics

A descriptive overview of the included studies is presented in Table 3, listing study characteristics including participants' demographics as well as the relevant results and measurements; studies are arranged in alphabetical order of first author and numbered to facilitate ease of reference.

Thirteen studies, published between 2001 and 2018, were included in this review. Studies were largely homogenous, with a majority being undertaken in Westernised countries with few exceptions (#8 from Israel and #9 from Turkey), and encompassed participants who were recruited in clinical settings (commonly described as cardiac-related locations). Additionally, most participants were male, between 69 to 87 % in each study, and were also of a similar age range (mean age was 50 to 70 years). About half of the included studies reported the ethnicity of participants, which was predominantly Caucasian. There was some variation in sample size, with the largest at 391 participants (#12) and the smallest with 39 participants (#2). In terms of study design, apart from a single case-control (#10), all other studies were cross-sectional (n = 5) or longitudinal (n = 7).

Myocardial Infarction

As part of the inclusion criteria, participants were required to have a clinically diagnosed MI. In this regard, while none of the studies reported the measurement procedure of the MI, evidence of the MI status of participants was present and derived from medical records provided by medical staff with the consent of participants. These records were examined by the authors of the respective studies, and in some cases (#1 and #10), in consultation with medical staff (e.g., nurses) to verify a positive MI diagnosis. For studies that reported the reference standard of a clinically diagnosed MI (n = 7), changes in ECG and elevated levels of cardiac enzymes signifying myocardial necrosis were commonly used.

Traumatic Stress

Included studies utilised several validated psychological measures to evaluate traumatic stress (see, Table 3); frequently described (n = 8) was the Posttraumatic Diagnosis Scale (PDS) (Foa, 1995). Other measures of traumatic stress included the PTSD Symptom Scale, the Stanford Acute Stress Reaction Questionnaire, the PTSD Inventory, the Clinician-

Administered PTSD Scale (CAPS), the Structured Clinical Interview for DSM-IV (SCID), and Modified PTSD Symptom Scale-Self Report.

Most of these measures were self-rated questionnaires (n = 9), with only four studies (#9, #11, #12, and #13) utilising interview-based measures such as the CAPS and SCID. Studies #11 and #13 employed both types of questionnaires (i.e., the CAPS and the PDS) in their respective studies. The mode of completion of these measures were as follows: self-rated questionnaires were mailed to participants, completed in-person, or through phone (#5, #6, and #7). Participants in studies #2 and #3 submitted their responses in-person as well as via mailing in questionnaires. Interview-based measures were administered with participants by trained personnel such as a psychiatrist (#9) or research assistants with psychology backgrounds (#7 and #12) through phone or in-person. Aside from a single study (#7) which referenced the DSM-III, all other studies referenced the DSM-IV and its revised edition, the DSM-IV TR (#4 and #12). The time since the initial diagnosis or experience of MI was substantially varied; this ranged from an average of 10 years (#5 and #6) to four days following the MI (#12). Lastly, there were also other measures of psychological distress (or otherwise) present in most studies, such as depression or anxiety as well as coping strategies; these are listed in Table 4.

Summary of Findings

Table 4 illustrates key information of included studies as per the research questions. The main classification of traumatic stress was post-traumatic stress disorder (PTSD), which was present in all studies, with some studies (#8, #9, and #12) adopting classifications of both ASD and PTSD. No other classification of traumatic stress was identified. Prevalence of traumatic stress diagnosis ranged from 1% to 31% in the included studies. Only one study (#2) did not quantify the prevalence rate, instead, reporting it as a “small percentage”

(Bennett et al., 2001). Additionally, studies frequently endorsed a *p*-value of less than .05 (*n* = 7) to denote a statistically significant effect between variables, often as a form of correlation and which indicated the presence of specific traumatic stress symptoms.

Base Symptoms

The base symptoms of traumatic stress aligning to a clinical diagnosis of PTSD or ASD as classified in included studies corresponded to symptomology reflected in the DSM-IV criteria of traumatic stress. 11 of the 13 studies were published prior to the 2013 publication of the DSM-5, whereas the two studies published afterwards continued to use the DSM-IV reference. These are symptoms of avoidance (of stimuli associated with the traumatic event), re-experiencing (of the traumatic event), and increased arousal (or hyperarousal). Mean scores of these symptoms were derived from their respective diagnostic measures and are reported in Table 4.

From the included studies, it is evident that some degree of avoidance, re-experiencing, and hyperarousal symptoms were present for participants (particularly for those with a positive diagnosis of traumatic stress). However, discerning the intensity of these symptoms was not possible as mean scores were reported for all participants that completed their respective diagnostic measures, without separation of participants that exhibited a positive diagnosis of traumatic stress. Additionally, the frequency of these symptoms was unclear, as most authors omitted reporting this aspect in their studies. Where frequency was specified however, this varied between studies. Three studies (#4 and #9, and #12) found re-experiencing symptoms to be most prevalent, whereas hyperarousal was most prevalent in one study (#6), and avoidance in another study (#9). In contrast, avoidance symptoms were identified to be least prevalent in other studies (#4 and #12).

Additional Symptoms

The base symptoms were compounded by additional symptoms of distress, where participants with a positive diagnosis for traumatic stress also exhibited poorer mental health as well as impaired physical and social functioning. Commonly reported across studies were symptoms of depression (n = 9), including negative affect (#2 and #3), and anxiety (n = 7), such as increased negative beliefs of future MI (#9 and #11), followed by greater somatic symptoms including high bodily pain (#7, #8, and #13). Other symptoms specific to respective studies included the onset of maladaptive coping strategies (#1 and #5) or social dysfunction (#1, #5, and #6), greater negative perceptions of the MI event (#4 and #9), and of oneself, including poorer emotional regulation (#7).

Symptoms Trajectory

The trajectory of symptoms was examined primarily in studies that were longitudinal in nature and encompassed multiple time points, as presented in Table 4. Generally, there were limited to changes observed in symptoms, for example, study #4 reported that hyperarousal increased briefly but reverted to baseline soon after, whereas moderate reduction of the frequency of intrusion and anxiety symptoms was observed in another study (#3). One study (#2) reported no changes and for two studies (#9 and #12), this was unclear because of non-reporting.

Methodological Quality Assessment

Included studies were evaluated to have multiple methodological shortcomings – these are noted in Table 5. Using the ‘NIH Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies’ (National Institutes of Health, 2014), it was found that several criteria were not met by most studies (see Appendix C for criteria). Where a given criterion was met, it has been omitted from Table 5. For criterion 9, which assessed the

quality of the exposure measurement, all studies derived an MI diagnosis from participant medical records, and although some studies (#3, #7, #8, #9, #10, and #13) provided a reference standard of a clinically diagnosed MI, none reported in detail the measurement procedure of the MI. This impacted criterion 12 – the blinding of outcome assessors, which was not likely to occur as authors were inevitably aware of participants' exposure status, and in most studies, approached participants directly for study inclusion. Criterion 11 focuses on the quality of the outcome measures and assessments. In this regard, a substantial proportion of studies used self-report measures, which were not as reliable nor accurate as an interview-based and clinician-administered measure. Additionally, while slightly more than half of the included studies controlled for key confounders (criterion 14), the number and type of confounders varied between studies, and there was no clear consensus across studies. Studies typically controlled for demographic variables (e.g., age, gender and educational levels), previous mental health history, previous non-MI cardiac events, clinical variables such as level of creatine phosphokinase, and psychological variables (e.g., depression, anxiety, fear of dying, perceived threat and helplessness). Three studies (#2, #5, and #6) did not adequately describe their respective study populations (criterion 2); the authors in these studies failed to specify the geographical location where participants were recruited from. Participation rate of at least 50% (criterion 3) was met in all but one study (#7), where only 35% of eligible persons participated.

Study Design

Included studies that were cross-sectional in nature ($n = 5$) as well as the single case-control study (#10) did not meet criterion 6 and 7, as these criteria looked at the timeframe between exposures and outcomes, specifically the assessment of exposure prior to outcome measurement (criterion 6) and a sufficient timeframe for an effect to be observed (criterion 7). Likewise, criterion 13 (follow-up rate), did not apply to these studies as there was no

follow-up. The remaining studies (n = 7) that were longitudinal (i.e., had multiple timepoints), were rated against criterion 13, and 5 studies (#2, #4, #8, #9, and #11) were found to have more than 20% loss to follow-up after baseline.

Non-Applicable Methodological Ratings

The correct classification of exposure status, as achieved by multiple assessments, entails a stronger study design, as illustrated by criterion 10. However, in the context of included studies, the exposure status was attained from medical records and verified by respective study authors and medical staff; this meant that multiple measurements to confirm diagnosis were not necessary. Therefore, ratings on criterion 10 were not applicable. The ratings on criterion 8 (levels of exposure) were also assessed to not be applicable to included studies, as the exposure (MI event) was measured as a dichotomous variable. In terms of sample size justification (criterion 5), most studies were exploratory and hypothesis-generating in nature. Thus, following the guidelines of the quality assessment tool, criterion 5 did not apply to these studies. For a select few studies (#10, #12 and #13), sample size justification was provided. This was generally achieved through a power analysis.

Discussion

This systematic review was conducted to summarise the literature on the topic of post-traumatic stress following MI. Thirteen publications were identified according to the inclusion/exclusion criteria. The aims and findings of the current review are now discussed in the context of three domains: classification (i.e., the different ways in which traumatic stress symptomatology has been classified), symptomatology (i.e., common clusters of psychological distress symptoms reported post-MI), and measurement (i.e., how traumatic stress symptoms are measured in this patient group). Limitations of this review are noted, and

implications of the issues identified throughout and recommendations for future research are presented.

Classification

The main classification of traumatic stress following a medical event (i.e., MI) used across the included studies was Post-Traumatic Stress Disorder (PTSD), based upon the DSM (specifically DSM-IV) -, and for several studies (#8, #9, and #12), an additional diagnosis of Acute Stress Disorder (ASD) was provided. No other classifications were present. Classifying traumatic stress disorders induced by medical events based upon the nosology of the DSM may be problematic because of the changes proposed with each new edition of the DSM, specifically relating to what constitutes as trauma. In this regard, what sets PTSD (and ASD) apart from other psychiatric disorders is the precondition of exposure to a stressful event (i.e., a trauma) (McNally, 2003; Pai et al., 2017). This precondition is explicitly formed as Criterion A in the DSM and serves a key gatekeeping function for diagnostic classification. However, the evolution of trauma definition in Criterion A from DSM-III (where PTSD was first included), to an expansion in the DSM-IV and subsequently narrowed in the current DSM-5 (Guina et al., 2017), has caused major controversies in the scientific community. Notably, the definition of trauma exposure in the DSM-IV was divided into A1 (firsthand or second-hand trauma exposure) and A2 (associated emotional reactions) (American Psychiatric Association, 1994), and this was regarded as extremely broad with some critics (McNally, 2003). It has been suggested that by virtue of meeting Criterion A, particularly the subjective aspect of emotional reactions, indirect exposures of less severe nature are comparable to severe catastrophic events, and thereby inaccurately increasing prevalence of PTSD cases. This hypothesis, termed as ‘conceptual bracket creep’ (McNally, 2003) was disputed because of findings that those who generally experienced A1 also reported A2, as found by some large-scale empirical studies (Kilpatrick et al., 2009), the joint

requirement of Criteria A1 and A2, as well as the threat (or loss) to life and serious injury as crucial aspects of psychological trauma, which encapsulates the definition of Criterion A (Kilpatrick et al., 2009; Weathers & Keane, 2007).

In the current DSM-5, the specifics of qualifying events for Criterion A has been narrowed considerably, particularly towards medically-based trauma – medical events do not just have to be life-threatening, but also sudden and catastrophic (Pai et al., 2017). However, studies on medical trauma show otherwise – for example notwithstanding the revision of Criterion A in the DSM-5, survivors of life-threatening illnesses have been routinely noted to meet diagnostic criteria for PTSD, particularly after undergoing intensive care (Davydow et al., 2008; Hall & Hall, 2013; Parker et al., 2015). These issues provide an insight into the debate surrounding broad versus narrow definitions of trauma, further discussion of which is beyond the scope of this review. However, it should be noted that the studies included in the current review illustrate that a medical event, in this case an MI, can lead to trauma-related symptoms (and consequently PTSD based on the DSM-IV), and moreover, that symptoms in this patient group can exhibit unique characteristics and further distress via comorbid psychiatric symptoms. This is explored further in the next section.

Symptomatology

Included studies in the current review reported a diagnosis of PTSD (or ASD) corresponding to the symptomatology listed in the DSM-IV – these symptoms were avoidance (of stimuli associated with the traumatic event), re-experiencing (of the traumatic event), and increased arousal (or hyperarousal). Notably, there was a lack of consensus and varying degrees of frequency across the studies in terms of which cluster of symptoms were most salient. For example, studies #4 and #12 reported re-experiencing symptoms to be most prevalent and avoidance to be the least. In contrast, study #9 reported avoidance to be most prevalent. Moreover, hyperarousal symptoms were most prevalent in study #6. A compelling

attribution for this divergent presentation of symptoms may be in the nature and specific characteristics of the trauma, which affects the manifestation of symptoms. Oflaz et al. (2014) suggest that the avoidance symptoms were more common because of participants endorsing an explicit aversion of behaviours and stress linked to the MI. Interestingly, authors of study #2 posited that avoidance symptoms were less likely to occur because of the medical nature of the trauma (i.e., internal), which makes avoidance difficult, as compared to situational traumas (i.e., external). The unique aspect of the internal nature of a medical trauma, such as an MI, has also been raised by other researchers. It has been proposed that a separation of the individual and the threat is impossible given the long-term impacts of a medical illness (e.g., an MI) that serves as reminders, such as medical obligations (e.g., routine check-ups) as well as physiological symptoms (Abbas et al., 2009; El-Gabalawy et al., 2018; Fait et al., 2018).

Moreover, Fiat et al. (2018) highlight that symptoms of cardiac-related PTSD take upon a supplementary future-oriented element that is expressed by a ‘fear of illness progression’ (FoP). This FoP serves as an additional stressor for those who have experienced a life-threatening illness by maintaining a fear that the illness will worsen over time – this is captured by the presentation of symptoms characterised by future-oriented concerns, such as avoiding follow-up medical appoints for fear of learning that illness has progressed, or medical professionals as painful reminders of the illness (Fait et al., 2018; Mundy & Baum, 2004; Vilchinsky et al., 2017). This future-oriented element has been raised as a key differentiating aspect in psychological distress and symptoms brought about by medical-related stressors versus that of conventional traumatic stressors, which tend to be acute as compared to the long-lasting nature of life-threatening medical events and illnesses (Mundy & Baum, 2004). The evidence that medical events, particularly if they are life-threatening, can have a traumatic effect on individuals and present with atypical traumatic symptoms has led to the concept of illness-induced PTSD (Sommer et al., 2018). Notably, an enduring

somatic threat (EST) model has been proposed to illustrate the psychological and behavioural sequelae following life-threatening illnesses (Edmondson, 2014). The three key dimensions are:

i. Illness-induced PTSD encompasses triggering events that are internal and ongoing, as opposed to 'traditional' PTSD whose traumatic events are external/discrete (e.g., patients lack separation from the traumatic occurrence due to the inherent nature of an illness);
ii. Unique characteristics of symptoms in illness-induced PTSD (e.g., the pathological responses associated with PTSD may themselves be a trigger for traumatic reoccurrence, thus creating a feedback loop;
iii. A heightened mortality awareness. This distinction between 'traditional' PTSD is further explored by Sommer and colleagues (2018), who identified unique characteristics in individuals with illness-induced PTSD (notably lowered risk for some psychiatric disorders but a higher association with substance use disorders), although both forms exhibited similar debilitating impacts on quality of life.

The atypical content of symptoms outlined in the EST model illustrates the element of subjectivity inherent in the development of PTSD, illness-induced or otherwise. Existing trauma and PTSD literature have underscored the importance of considering individuals' subjective experiences towards adverse events to influence the occurrence of trauma-related symptoms – the greater the individual's negative appraisal towards the adverse event, as evident by the presence of traumatic distress (e.g., fear, helplessness), the higher the likelihood of PTSD developing, as well as increased severity of symptoms (Boals, 2017; Rasmussen et al., 2007; Weinberg & Gil, 2016). This also means that individuals exposed to

adverse events may not develop PTSD due to subjectively rating the event as non-traumatic, although generally, those who subjectively rated events as traumatic were reacting to objectively-defined trauma exposure (Boals, 2017; Rasmussen et al., 2007). Findings of several included studies in the current review support the association between subjective appraisal and PTSD symptom development. The notion of illness perception and related to it, the different domains of appraisal (e.g., consequences, control), were found to contribute significantly to PTSD development and subsequent symptoms, as reported by studies #1, #9, and #11. For example, individuals were more likely to develop PTSD or have increased intensity of symptoms when they perceive the MI more negatively, such as a perceived loss of control or permanent change in quality of life. Moreover, authors of studies #8 and #12 highlighted that subjective experiences were a stronger predictor than the objective severity of the MI.

Importantly, negative appraisals of MI, and its resultant PTSD, have been associated with greater psychiatric comorbidities – the current review found that included studies reported additional psychological distress, the most frequent being depression (in 9 out of 13 studies). This is consistent with the broader PTSD literature, where it has been well-established that PTSD and depression commonly co-occur (Angelakis & Nixon, 2015; Caramanica et al., 2014; Ginzburg et al., 2010; Rytwinski et al., 2013; Shah et al., 2012). Additionally, comorbid PTSD and depression lead to greater symptom severity compared to a singular psychiatric disorder (Shah et al., 2012). Notably, researchers looking to untangle the temporal nature of comorbid PTSD and depression have identified several pathways that include a bidirectional causality, shared risk factors as well as cognitive and genetic vulnerabilities; suggestive of a complex relationship between both disorders (Angelakis & Nixon, 2015). Whatever that may be, psychiatric comorbidities have detrimental impacts on trauma-exposed populations, including the cardiac population, where their occurrence has

been associated with an increased risk of negative outcomes, such as adjustment difficulties and in-hospital mortality (Dao et al., 2010; Ginzburg, 2006).

Measurements

Numerous tools have been developed for measuring symptoms of PTSD, of which there are two broad formats: self-report instruments and interview-based instruments. Self-report instruments are brief, easy to administer, and routinely utilised for screening purposes including early detection, as well as identification of follow-up clinical assessment and risk assessment. Interview-based instruments are specialised and involve often lengthy procedures to establish a clinical diagnosis of PTSD and aid treatment planning and interventions (Brewin, 2005; Lancaster et al., 2016; Wisco et al., 2012). The accessibility, uniqueness, and authorships of instruments were identified to be influencing factors in their use across clinical and research settings (Elhai et al., 2005). In this regard, several instruments stood out as most frequently used by traumatic stress professionals, particularly the PDS, which measures all PTSD criteria, and the CAPS, which assesses both symptomatic frequency and intensity (Elhai et al., 2005). Not surprisingly, studies included in the current review typically utilised either or both instruments. The PDS was the most widely used instrument (n = 8) as well as among other self-report measures, whereas the CAPS was used in three of the four studies that utilised interview-based measures.

There are significant implications related to the choice of measurement format, and this is most evident in studies of PTSD prevalence estimations. Within the PTSD literature, the use of self-reported PTSD measures has been associated with higher rates of PTSD occurrence (Malinauskaite et al., 2017; Shemesh et al., 2006), which were identified to be overestimations when compared to interview-based measures (Cody et al., 2017; Edmondson et al., 2012; Griffin et al., 2004; Tang et al., 2020). Further studies on PTSD diagnostic instruments found that PTSD development rates indeed varied according to the format of

instrument utilised – for example, 29.2% via the self-report Post-Traumatic Symptom Scale 10 (PTSS-10) compared to just 4.8% using Structured Clinical Interview for DSM-IV (SCID) (Einsle et al., 2012). Similar discrepancies were also observed for studies in the current review – estimates of 1%and 4.7% were derived from the interview-based CAPS, whereas in the same studies, 18%and 17.9% prevalence was indicated in the self-reported PDS, for studies #11 and #13 respectively.

Several probable explanations have surfaced to account for such discordant findings between the two formats of assessments. For example, the evaluation of the precondition Criterion A (exposure to trauma) that is necessary for a PTSD diagnosis – without this, self-report instruments such as the PSS-I and SASRQ that measure only symptoms based on the DSM-IV (criteria B to D) are likely to generate positive overestimates in the presence of PTSD-like symptoms and thus contribute to PTSD prevalence, however, because of a possibility that symptoms may not be attributed to the trauma, a true diagnosis of PTSD may not be warranted (Hoffman et al., 2011). Additionally, the characteristics of dichotomous endorsement of items inherent in some self-report measures such as the PDS (versus continuous ratings of intensity and frequency in structured interviews) may inflate PTSD prevalence estimates, as these do not measure clinical significance or function (Griffin et al., 2004; Thombs et al., 2018). Another attribution lies in the screening purposes of self-report instruments, which when utilised, seeks to broadly identify probable cases than for diagnostic classification; cut-off thresholds are set low and this generates many false positives (Levis et al., 2019; Thombs et al., 2018). Moreover, interview-based measures when administered by trained personnel have an additional layer of vetting, which relies on the clinical judgement of the assessor, therefore this may result in lower estimates but higher accuracy (Griffin et al., 2004). The ability to derive accurate prevalence estimates via interview-based measures may also be hampered by avoidance behaviours (e.g., to avoid reminders of the traumatic event)

resulting in decreased participation rates (Gander & von Känel, 2006). Similar patterns of findings due to assessment format have been observed across a variety of health and psychiatric outcomes in differing populations – for example, a study on depression prevalence across 69 meta-analyses (consisting of 2094 included primary studies) found that self-report screening tools produced 14% percent higher estimates on average compared to interviews (Levis et al., 2019). Other examples include internet gaming disorder among adolescents (Jeong et al., 2018) and post-concussion symptoms in veterans with mild traumatic brain injury (Kondiles et al., 2015).

A third format of assessment, a computerised-adaptive test (CAT) has been proposed as a promising alternative to address the limitations of existing PTSD measures by reducing administration and processing times, while also increasing precision and offering individualised assessments (Del Vecchio et al., 2011; Eisen et al., 2016). A recent development, the P-CAT, or computerised-adaptive test for PTSD, functions as a psychometrically robust PTSD electronic assessment tool to improve and supplement existing PTSD measures (Eisen et al., 2016).

Limitations

The current review has several limitations. First, a meta-analysis was not conducted because of methodological heterogeneity such as different study designs, timings of outcome measurement since index traumatic event, as well as significant gaps in quality of included studies (see Table 5), and the absence of data because of incomplete or missing reporting. More importantly, the aims of the current review are not statistically driven nor require the level of statistical analyses inherent in a meta-analysis to address. A second limitation is that articles from non-peer-reviewed or unpublished sources (i.e., grey literature) were excluded to ensure scientific rigour generally expected from being peer-reviewed – however, this can potentially omit balanced and relevant findings, including negative results (Paez, 2017).

Finally, only English language full-text studies were reviewed – foreign language studies whose English abstracts showed promise were excluded due to practical reasons and this presents a potential loss of contributory findings.

Implications and Future Directions

The issues illustrated in the preceding sections highlight some key elements in understanding PTSD-induced MI that warrant further attention and research. These include the subjective element in the presentation of symptoms, which itself exhibits unique characteristics attributed to a medical event (in this case, an MI) as the trauma source, as well as differences in PTSD assessment formats and diagnostic criteria. The occurrence of depressive comorbidity also serves as another pertinent aspect, given its frequent presentations and greater combined impacts with comorbid PTSD. Several clinical implications can be derived. Given that there is compelling evidence that PTSD can develop post-MI, it may be advisable that clinicians should routinely screen for PTSD in patients following an MI occurrence, to facilitate early detection and intervention. Moreover, the differential presentations of symptoms, such as future-oriented concerns, can serve to guide treatment decisions, but importantly, warrant the development of tailored diagnostic instruments and treatments (Fait et al., 2018; Sommer et al., 2018; Vilchinsky et al., 2017). Likewise, depression should also be assessed and targeted for treatment, as a stand-alone occurrence or as a comorbid condition to reduce the likelihood of future PTSD development, as well as exacerbation of existing symptoms.

While there is no definitive evidence regarding best treatment approaches for PTSD with psychiatric comorbidities, it is reasonable to suggest that for either a combined or sequential treatment for PTSD and comorbid depression, there is a need to address the therapy-interfering nature of depressive symptoms (Angelakis & Nixon, 2015). The presence of negative subjective trauma appraisals as part of, as well as in addition to, the individual's

experiences following trauma exposure should be considered to guide individualised assessments and treatments. In this regard, clinicians should also consider more than the type of events, (i.e., the specifics of what stressor event constitutes as traumatic), which are ever-evolving according to different editions of diagnostic manuals when working with an individual following a life-threatening event. Instead, further considerations should be directed towards the individual's psychiatric history, for example, to determine if current symptoms are entirely new or have been exacerbated by pre-existing psychiatric conditions, as well as consequent dysfunction and distress (Guina et al., 2017; Kangas, 2013). Future research can look towards the clinical utilisation of artificial intelligence and machine learning for PTSD – recent studies have shown promising results, such as facilitating the processing of an objectively measurable biomarker for classifying PTSD (Zhang et al., 2020), discriminating between other psychiatric comorbidities (Shim et al., 2019), as well as accurately predicting symptoms (Wshah et al., 2019). Additionally, this review has highlighted that the majority of research on this topic has been conducted with Caucasian men. Future research should focus more on incorporating a greater diversity of demographic factors, particularly gender and ethnicity to improve generalisability of findings; participant characteristics in the current review are homogenous, consisting of mostly men and Anglo-Saxon ethnicity. The broad literature has identified the female sex and ethnic minority status to be associated with cardiac-induced PTSD, and PTSD in general (Edmondson et al., 2012; Fortin et al., 2013; Tolin & Foa, 2006).

Conclusion

Given the growing interest in this topic and the myriad of findings, it is pertinent to systematically review the available evidence, identify gaps in research and discuss implications. The existing literature have routinely associated negative consequences and poor outcomes with traumatic stress symptoms across a wide range of trauma types. In this

regard, empirical studies have increasingly identified the occurrence of traumatic stress symptoms following an MI, and few reviews have been conducted to bring together this body of evidence. The current review identified thirteen studies after a comprehensive search protocol across six electronic databases. A qualitative synthesis of these studies found that a common classification of traumatic stress was PTSD (or ASD) regardless of the nature of the trauma. This classification was largely based upon the DSM-IV criteria, and corresponded to symptoms of re-experiencing, avoidance, and arousal and were typically measured via self-report instruments. Notably, the divergent presentations and unique content of symptoms, as well as the presence of depressive comorbidity, suggest a need for tailored diagnostic and screening instruments as well as interventions. Moreover, the findings of this review illustrate that there are many interlinked and overlapping components in post-MI PTSD, and broadly, the multifactorial nature of PTSD; many unknowns still exist around this topic. Future research should consider the therapy-interfering nature of depressive symptoms, subjective trauma appraisals, as well as the clinical utilisations of machine-learning to guide assessments and treatments; methodological considerations include greater diversity of demographic factors such as gender and ethnicity. Overall, findings suggest that potential maladaptive emotional and behavioural symptoms, as well as distress experienced by an individual following an MI, are likely to contribute to a poor and impaired quality of life. As such, while MI mortality rates are falling steadily, the increase in the number of survivors with traumatic stress requires timely translation of research to policy to promote the ongoing wellbeing of this patient group.

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Table 1

Descriptions of PTSD Symptoms

Criterion	Examples
B. Intrusions, where traumatic event is re-experienced	• Unwanted upsetting memories • Nightmares etc.
C. Avoidance (of trauma-related stimuli)	• Trauma-related thoughts or feelings • Trauma-related external reminders
D. Negative alterations in mood and cognition that began or worsened after trauma	• Negative affect • Exaggerated blame of self or others for causing trauma etc.
E. Alterations in arousal and reactivity that began or worsened after trauma	• Irritability or aggression • Hypervigilance, etc.

Table 2

Brief description of assessment tools in the Australian (Phoenix Australia, 2013) and American (American Psychological Association, 2018) clinical practice guidelines for the treatment of PTSD

Interview-based	Self-report
Clinician-Administered PTSD Scale for DSM-5 (Weathers et al., 2018) • 30 items • Provide current (past month), lifetime and past week diagnosis • 45 - 60 minutes to administer	*PTSD Checklist for DSM-5* (Blevins et al., 2015) • 20 items • Two versions available (civilian and military)
PTSD Symptom Scale Interview for DSM-5 (Foa et al., 2016) • 24 items • Semi-structured • Approximately 20 minutes to administer	*Primary Care PTSD* (Prins et al., 2016) • 5 items • Designed for use in primary care settings
Structured Interview for PTSD (Davidson et al., 1997) • 17 items • Based on the DSM-IV • 20 – 30 minutes to administer	*Posttraumatic Diagnostic Scale for DSM-5* (Foa et al., 2016) • 24 items • Assess symptom severity in the last month

Table 3

List of Inclusion Criteria

Inclusion Criteria	
Preliminary criteria	Additional criteria
1. Adults, aged 18 and above 2. Must have had a diagnosed myocardial infarction prior to (3); can be classified as an acute coronary syndrome or together with other cardiac conditions 3. Must report traumatic stress as defined by the DSM-5 (or earlier) OR ICD-10 (or earlier), after (2) 4. Must have (3) as an outcome measure, i.e., traumatic stress diagnosis 5. Must be a peer-reviewed journal publication 6. Must include direct statistical analysis of relationship between (2) and (3) 7. English language 8. Published within 20 years of first search date, i.e., 1st Feb 2000 onwards	9. Must describe traumatic stress symptoms as defined by the DSM-5 (or earlier) OR ICD-10 (or earlier) after a diagnosed onset of myocardial infarction

Table 4

Summary of Results

Citation	Traumatic Stress measurements	Relevant results	Summary
[#1] Ayers et al., (2009) UK *Study design:* Cross-sectional *Setting:* Cardiac rehabilitation programs *Sample:* *N* = 74, 76% male, mean age 62y, 91% Caucasian. *Named data source:* None cited	*Reference standard*: DSM-IV *Evaluation tool*: PDS *Evaluation method:* Self-report *Time since MI diagnosis:* Max. of 12 weeks *Other psychological measures of distress or otherwise:* - General Health Questionnaire – 28 items - Subjective experience of MI questionnaire - Appraisal and coping questionnaire	*Classification:* Post-traumatic stress disorder *Prevalence of PTSD:* 16% 17.6% (for subthreshold) *PDS scores:* • Avoidance (out of 19) *M* = 3.62 • Arousal (out of 15) *M* = 3.01 • Re-experiencing (out of 12) *M* = 2.35 *Symptoms trajectory:* N/A	Negative perceptions of consequences and use of dysfunctional coping strategies account for 77% of variance in post-MI PTSD symptoms. Symptoms of depression, anxiety, somatic symptoms, and social dysfunction were also present. Individuals who experience an MI and perceived it to have negative lasting impacts on their life, and who have adjustment difficulties, may experience subsequent PTSD symptoms.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#2] Bennett et al., (2001) UK *Study design:* Longitudinal *Setting:* Hospital medical wards *Sample:* *N* = 39, 77% male, mean age 59.7y, 'Majority' Caucasian. *Named data source:* None cited	*Reference standard*: DSM-IV *Evaluation tool*: PDS – Part 3 *Evaluation method:* Self-report *Time since MI diagnosis:* Min. of three months *Other psychological measures of distress or otherwise:* - Cognitive Appraisal Questionnaire - Impact of Event Scale - Global Mood Scale	*Classification:* Post-traumatic stress disorder *Prevalence of PTSD:* Unclear – 'small percentage', at least 3% *PDS scores:* • Avoidance (out of 19) *M* = 4.79 • Arousal (out of 15) *M* = 5.22 • Re-experiencing (out of 12) *M* = 2.64 *Symptoms trajectory:* No change over the course of the study	Negative affect (fatigue and malaise) and low positive affect (energy and sociability), as well as fear at time of MI, predicted subsequent PTSD symptoms. Other symptoms include dissociation and intense emotions at time of MI and three months later. Additionally, appraisals and emotional reaction strongly predicted intrusive symptoms 3 months later. However, avoidance was the least well-predicted PTSD symptom.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#3] Bennett et al., (2002) UK *Study design:* Longitudinal *Setting:* Hospital medical wards *Sample:* *N* = 75, 78% male (time 1), mean age 60y (time 1), Ethnicity not reported. *Named data source:* None cited	*Reference standard*: DSM-IV *Evaluation tool*: PDS *Evaluation method:* Self-report *Time since MI diagnosis:* Min. of three months *Other psychological measures of distress or otherwise:* - Cognitive Appraisal Questionnaire - Impact of Event Scale - DUKE Social Support Questionnaire - Hospital Anxiety and Depression Questionnaire - Toronto Alexithymia Scale - Positive and Negative Affect Schedule	*Classification:* Post-traumatic stress disorder *Prevalence of PTSD:* 16% *PDS scores:* • Avoidance (out of 19) *M* = 3.81 • Arousal (out of 15) *M* = 3.95 • Re-experiencing (out of 12) *M* = 2.52 *Symptoms trajectory:* Moderate reduction of frequency of intrusive memories of MI, as well as anxiety symptoms No change in avoidance symptoms	Initial symptoms of intrusion and avoidance strongly predicted PTSD symptoms 3 months later, followed by dissociation at time of MI. Other symptoms present include depression (including negative affect) and anxiety, as well as dissociation at time of MI. An absence of confidant support was reported in relation to avoidance symptoms. Authors suggest that early screening of psychological distress at time of event, followed by brief intervention for patients with psychological symptoms would be appropriate.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#4] Castilla & Vazquez, (2011) Spain *Study design:* Longitudinal *Setting:* Coronary Intensive Care Unit *Sample:* *N* = 76 (Time 1), 48 (Time 2), 33 (Time 3), 76% male (Time 1), mean age 60y (Time 1), Ethnicity not reported. *Named data source:* None cited	*Reference standard*: DSM-IV-TR *Evaluation tool*: PCL-C *Evaluation method:* Self-report *Time since MI diagnosis:* Min. 48-72 hours *Other psychological measures of distress or otherwise:* - Perceived importance of heart failure - Goldberg Health Questionnaire - Life Orientation Test – Revised - Positive and Negative Affect Schedule	*Classification:* Post-traumatic stress disorder *Prevalence of PTSD:* 3.1% (T3), 11.1% (T2), 1.1% (T1) *PCL-C scores:* • Re-experiencing (out of 25) *M* = T1 (6.64), T2 (8.06), T3 (6.34) • Avoidance (out of 35) *M* = T1 (8.94), T2 (9.40), T3 (8.75) • Hyperarousal (out of 25) *M* = T1 (7.09), T2 (9.25), T3 (7.91) *Symptoms trajectory:* Increased perception of MI as traumatic at later months after MI occurrence No significant changes in symptoms of re-experiencing and avoidance over time Hyperarousal increased at T2, returning to baseline at T3	Positive affect including optimism, remain high (for this sample). Negative perception of event, reported at Time 2 and 3, and its associated severity did not equate to perception that one's life was in danger. Limited traumatic symptoms and perception at time of event. Additionally, re-experiencing most prevalent and avoidance the least. Thus, perception can be susceptible to significant changes over time and should be monitored to assist in mental health wellbeing. Additionally, traumatic event does not necessarily lead to traumatic symptoms/PTSD.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#5] Chung et al., (2008) UK *Study design:* Cross-sectional *Setting:* General practice *Sample*: *N* = 96, 81% male, mean age 70y, Caucasian 99%. *Named data source:* None cited	*Reference standard*: DSM-IV *Evaluation tool*: PDS *Evaluation method:* Self-report *Time since MI diagnosis:* Average 10 years *Other psychological measures of distress or otherwise:* - General Health Questionnaire – 28 - COPE Scale	*Classification:* post-traumatic stress disorder *Prevalence of PTSD:* 30% 42% (subthreshold) *PDS scores:* Mean scores not reported *Symptoms trajectory:* N/A	Linear relationship between severity of PTSD symptoms and severity of co-morbidity in older people experiencing post-MI PTSD. Coping strategies vary according to severity of PTSD. There was an endorsement of maladaptive strategies characterised by greater seeking of emotional support, greater focusing and venting of emotions, as well as problem-focused strategies such as suppression of competing activities and restraint coping. Symptoms of depression, anxiety, somatic symptoms, and social dysfunction were also present.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#6] Chung et al., (2011) UK *Study design:* Cross-sectional *Setting*: General Practice *Sample:* *N* = 120, 78% male, mean age 67y, Caucasian 99%. *Named data source:* None cited	*Reference Standard*: DSM-IV *Evaluation tool*: PDS *Evaluation method:* Self-report *Time since MI diagnosis:* Average 10 years *Other psychological measures of distress or otherwise:* - MI experience questionnaire - General Health Questionnaire – 28 - NEO – Five Factor Inventory - COPE Scale	*Classification:* Post-traumatic stress disorder *Prevalence of PTSD:* 31% *PDS scores:* • Avoidance (out of 19) *M* = 4.15 • Hyperarousal (out of 15) *M* = 5.01 • Re-experiencing (out of 12) *M* = 3.49 *Symptoms trajectory:* N/A	Severity of traumatic stress symptoms was influenced by complex factors such as personality traits, subjective experience of MI and coping strategies (problem-focused). Hyperarousal was most prevalent symptom, and characterised by fits of anger, trouble sleeping and concentrating, whereas avoidance was presented by feeling future (plans) or hopes would never come true, being less interested in important activities. Re-experiencing surfaced via feeling upset when reminded of MI, and upsetting thoughts and images about MI. Symptoms of social dysfunction (most prevalent), somatic and anxiety problems, and depression (the least) were also present.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#7] Doerfler et al., (2005) USA *Study design:* Cross-sectional *Setting:* Medical clinics *Sample:* *N* = 52, 69% male, mean age 57y, Caucasian 98%. *Named data source:* None cited	*Reference standard*: DSM-III-R, DSM-IV *Evaluation tool*: PSS *Evaluation method:* Self-report *Time since MI diagnosis:* 3-6 months *Other psychological measures of distress or otherwise:* - Impact of Events Scale - Medical Outcomes Study 20-item - Perceived Controllability Scale	*Classification:* Post-traumatic stress disorder *Prevalence of PTSD:* 7.7% *PSS scores* (out of 51)*:* *M* = 6.9 (individual sub-scores not reported) *Symptoms trajectory:* N/A	Perceptions of control were related to the likelihood of experiencing PTSD symptoms, particularly a perceived lack of control and a generalised sense of lack of control over adverse events. Symptoms of intrusion and high bodily pain were present, as well as well lower controllability of emotions during and of future MI. Reduced mental health and lowered functioning across several domains (social, role and physical) were reported.
[#8] Ginzburg et al., (2003)	*Reference standard*: DSM-IV	*Classification:* Acute Stress Disorder, Post-traumatic stress disorder	Dissociation, high levels of anxiety, somatic complaints, increased pain, and

Citation	Traumatic Stress measurements	Relevant results	Summary
Israel *Study design:* Longitudinal *Setting:* Cardiac intensive care unit *Sample:* *N* = 196 (Time 1), 116 (Time 2), 81% male, mean age 35 - 70y, Ethnicity not reported. *Named data source:* None cited	*Evaluation tool*: SASRQ, PTSD Inventory *Evaluation method:* Self-report *Time since MI diagnosis:* Mean of 3.5 days (T1), 7 months (T2) *Other psychological measures of distress or otherwise:* - Taylor Manifest Anxiety Scale - Physical residuals - Health-related quality of life	*Prevalence of PTSD and ASD:* 18% as having ASD (T1), 16% as having PTSD (T2) *SASRQ and PTSD Inventory scores:* Mean scores not reported; hyperarousal at T1 and T2. *Symptoms trajectory:* Having traumatic stress symptoms at occurrence increases risk by 3 times of having traumatic stress 7 months	lowered vitality were present. Additionally, poor social functioning and mental health, as well as lowered general health and perceived fulfilment of emotional and physical roles were reported. Initial traumatic stress symptoms did not predict sequelae of acute stress disorder (ASD), where its diagnosis was distinct from post-traumatic stress disorder. Additionally, having a prior ASD diagnosis had no bearing on adjustment or subsequent PTSD development. Results suggest perceived level of threat after MI should be measured to monitor for future development of PTSD.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#9] Oflaz et al., (2014) Turkey *Study design:* Longitudinal *Setting:* Cardiac intensive care unit *Sample:* *N* = 76 (Time 1), 59 (Time 2), 78% (Time 1) and 83% (Time 2) male, mean age 54y (Time 1) and 51.5y (Time 2), Ethnicity not reported. *Named data source:* None cited	*Reference standard*: DSM-IV *Evaluation tool*: CAPS - Turkish *Evaluation method:* Clinician-Administered *Time since MI diagnosis:* Max. 6 months *Other psychological measures of distress or otherwise:* - Hamilton Depression Rating Scale – Turkish version - Hamilton Anxiety Rating Scale – Turkish version - Illness Perception Questionnaire	*Classification:* Acute Stress Disorder, Post-traumatic stress Disorder *Prevalence of ASD and PTSD:* 9.2% as having ASD (T1), 11.9% as having PTSD (T2) *CAPS scores:* • Intrusion/Re-experiencing (out of 40) *M* = T1 (22.14), T2 (18.85) • Avoidance (out of 56) *M* = T1 (16.70), T2 (16.85) • Hyperarousal (out of 40) *M* = T1 (15.14), T2 (17.14) *Symptoms trajectory:* Base symptoms N/R, however observed increase in ability to evaluate emotions over time	Symptoms of anxiety and depression, as well as feelings of helplessness, being near death and horror were present. Additionally, sexual reluctance and avoidance with decreased sexual enjoyment were reported. Negative perceptions about illness consequences, identity and concerns were reported, and predicted ASD and PTSD progression over time. High intrusion/re-experiencing scores were the most prevalent symptoms, followed by avoidance.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#10] Pedersen et al., (2003) Denmark *Study design:* Case-control *Setting:* Cardiac clinics / national registers (controls) *Sample:* *N* = 112, 71% male, mean age 60y, ethnicity not reported. *Named data source:* None cited	*Reference standard*: DSM-IV *Evaluation tool*: PDS *Evaluation method:* Self-report *Time since MI diagnosis:* Min. 4 weeks *Other psychological measures of distress or otherwise:* - Trauma Symptom Checklist - Eysenck Personality Questionnaire	*Classification:* Post-traumatic stress disorder *Prevalence of PTSD:* 22% *PDS scores:* • Avoidance (out of 19) *M* = 2.09 • Arousal (out of 15) *M* = 2.33 • Re-experiencing (out of 12) *M* = 2.46 *Symptoms trajectory:* N/A	Symptoms of anxiety and depression were present. Patients who experienced MI had a higher risk of future PTSD than healthy individuals; age, school and work were not related to diagnosis of PTSD.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#11] Princip et al., (2018) Switzerland *Study design:* Longitudinal *Setting:* Coronary care unit *Sample:* *N* = 130 (Time 1) 96 (Time 2), 82% male, mean age 60y, Caucasian 100%. *Named data source:* MI-SPRINT Study	*Reference standard*: DSM-IV *Evaluation tool*: CAPS – German, PDS *Evaluation method:* Clinician-Administered, Self-report *Time since MI diagnosis:* Min. three months *Other psychological measures of distress or otherwise:* - Illness Perception Questionnaire – German version - Beck Depression Inventory	*Classification:* Post-traumatic Stress Disorder *Prevalence of PTSD:* 1% (via CAPS), 14.4% (subthreshold), 18.8% (via PDS) *PDS scores:* • Avoidance (out of 19) *M* = 2.08 • Arousal (out of 15) *M* = 2.40 • Re-experiencing (out of 12) *M* = 1.45 *CAPS scores:* • Avoidance (out of 56) *M* = 3.57 • Arousal (out of 40) *M* = 5.30 • Re-experiencing (out of 40) *M* = 3.36 *Symptoms trajectory:* N/R	A fear of dying and depressive symptoms were present among most participants. Greater negative beliefs about illness consequences (such as physical impairments) as well as negative emotional responses to the MI and stronger illness concerns were also reported, and which impacted the development of future post-MI PTSD. Younger and older participants exhibited differing illness perception, with younger people showing higher illness concerns and assumed a longer illness timeline. Authors suggest that assessments and interventions targeting illness perception post-MI may be valuable in decreasing risk of subsequent PTSD development.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#12] Roberge et al., (2010) Canada *Study design:* Longitudinal *Setting:* Coronary units *Sample:* *N* = 391, 76% male, mean age 59y, ethnicity not reported. *Named data source:* None cited	*Reference standard*: DSM-IV-TR *Evaluation tool*: SCID (ASD, PTSD, Past PTSD Modules), MPSS-SR *Evaluation method:* Interviewer-Administered, Self-report *Time since MI diagnosis:* Min. four days *Other psychological measures of distress or otherwise:* - Beck Depression Inventory - Life Events Stress Scale - Modified Medical Outcome Study Social Support Survey - Trauma Assessment (Adults)	*Classification:* Acute Stress Disorder, Post-traumatic Stress Disorder *Prevalence of ASD and PTSD:* 3.6% as having ASD (T1), 4.1% as having PTSD (T2) *SCID and MPSS-SR scores*: Mean scores not reported *Symptoms trajectory:* N/R	Intensity of traumatic stress symptoms following occurrence of MI predicted the development of further depressive symptoms in the months following an MI. The presence of depressive symptoms shortly after MI warrants further monitoring and possibly assessment for subsequent PTSD symptoms. Depressive symptoms were also associated a greater perceived threat to life during MI, as well as feelings of fear, helplessness, and horror. The strength of association between these two conditions does not seem to be attributable to their overlapping symptoms. Initial assessment and intervention when patients are in hospital shortly after an MI may limit negative psychological consequences. Avoidance symptoms were least prevalent, whilere-experiencing symptoms was most prevalent.

Citation	Traumatic Stress measurements	Relevant results	Summary
[#13] Wiedemar et al., (2008) Switzerland *Study design:* Cross-sectional *Setting:* Cardiology clinic *Sample:* *N* = 190, 87% male, mean age 60y, ethnicity not reported. *Named data source:* None cited	*Reference standard*: DSM-IV *Evaluation tool*: CAPS – German, PDS *Evaluation method:* Clinician-Administered, Self-report *Time since MI diagnosis:* Min. 24 days *Other psychological measures of distress or otherwise:* N/A	*Classification:* Post-traumatic Stress Disorder *Prevalence of PTSD:* 17.9% (via PDS), 4.7% (via CAPS), 3.2% - 44.7% (subthreshold) *CAPS scores:* *M* = 45 *PDS scores:* *M* = 28.9 (individual sub-scores not reported) *Symptoms trajectory:* N/A	Common symptoms reported included: fear of dying, helplessness, and high levels of perceived pain. Subjective perception of MI experience predicted subsequent PTSD symptoms. Authors suggest that clinical settings may warrant psychological interventions that are accessible and provided during or shortly after the MI.

Note. CAPS = Clinician-Administered PTSD Scale; DSM-IV –[TR] = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [Text Revised]; MPSS-SR = Modified PTSD Symptom Scale-Self Report; PDS = Posttraumatic Diagnostic Scale; PCL-C = PTSD Checklist; SASRQ = Stanford Acute Stress Reaction Questionnaire; SCID = Structured Clinical Interview for DSM-IV; N/R = Not Reported; N/A = Not Applicable.

Table 5

Quality Assessment of Included Studies, Noting Unmet Criteria Based on NIH Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies.

Citation	Criteria 1 - 5	Criteria 6 – 10 (*NB:* 8 and 10 not applicable for all)	Criteria 11 & 12	Criteria 13 & 14
[#1] Ayers et al., (2009)	Criterion 5: Not applicable.	Criteria 6 & 7: Exposure and outcome measured at same time. Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors not blinded.	Criterion 13: Not applicable. Criterion 14: Key potential confounding variables not controlled.
[#2] Bennett et al., (2001)	Criterion 2: Study population not adequately described. Criterion 5: Not applicable.	Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors not blinded.	Criterion 13: Loss to follow-up after baseline > 20%.
[#3] Bennett et al., (2002)	Criterion 5: Not applicable.	Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors not blinded.	
[#4] Castilla & Vazquez, (2011)	Criterion 5: Not applicable.	Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors not blinded.	Criterion 13: Loss to follow-up after baseline > 20%. Criterion 14: Confounding variables not reported.
[#5] Chung et al., (2008)	Criterion 2: Study population not adequately described. Criterion 4: Participants recruited from different populations. Criterion 5: Not applicable.	Criteria 6 & 7: Exposure and outcome measured at same time. Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors not blinded.	Criterion 13: Not applicable.

Citation	Criteria 1 - 5	Criteria 6 – 10 (*NB:* 8 and 10 not applicable for all)	Criteria 11 & 12	Criteria 13 & 14
[#6] Chung et al., (2011)	Criterion 2: Study population not adequately described. Criterion 5: Not applicable.	Criteria 6 & 7: Exposure and outcome measured at same time. Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors not blinded.	Criterion 13: Not applicable. Criterion 14: Key potential confounding variables not controlled.
[#7] Doerfler et al., (2005)	Criterion 3: Less than 50% participation rate. Criterion 5: Not applicable.	Criteria 6 & 7: Exposure and outcome measured at same time. Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors' blinded status cannot be determined.	Criterion 13: Not applicable. Criterion 14: Key potential confounding variables not controlled.
[#8] Ginzburg et al., (2003)	Criterion 5: Not applicable.	Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors' blinded status not reported.	Criterion 13: Loss to follow-up after baseline > 20%. Criterion 14: Key potential confounding variables not controlled.
[#9] Oflaz et al., (2014)	Criterion 5: Not applicable.	Criterion 9: Exposure measurements unclear.	Criterion 12: Outcome assessors' blinded status not reported.	Criterion 13: Loss to follow-up after baseline > 20%. Criterion 14: Key potential confounding variables not controlled.
[#10] Pedersen et al., (2003)		Criteria 6 & 7: Exposure and outcome measured at same time. Criterion 9: Exposure measurements unclear.	Criterion 11: Self-reported outcome measures. Criterion 12: Outcome assessors' blinded status cannot be determined.	Criterion 13: Not applicable.
[#11] Princip et al., (2018)	Criterion 5: Not applicable.	Criterion 9: Exposure measurements unclear.	Criterion 12: Outcome assessors' blinded status not reported.	Criterion 13: Loss to follow-up after baseline > 20%.

Citation	Criteria 1 - 5	Criteria 6 – 10 (*NB:* 8 and 10 not applicable for all)	Criteria 11 & 12	Criteria 13 & 14
[#12] Roberge et al., (2010)			Criterion 12: Outcome assessors' blinded status cannot be determined.	
[#13] Wiedemar et al., (2008)		Criteria 6 & 7: Exposure and outcome measured at same time. Criterion 9: Exposure measurements unclear.	Criterion 12: Outcome assessors not blinded.	Criterion 13: Not applicable.

Note: Methodological shortcomings of included studies noted against the NIH Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies (National Institutes of Health, 2014). Table notes unmet or non-applicable criteria; any criterion not listed was assessed as met for the respective study (see Appendix C).

Appendix A: Search protocol for individual databases

CINAHL: 1st Feb 2020 Traumatic Stress symptoms and Myocardial Infarction		
Reference #	**Search Terms**	**Results**
Myocardial Infarction		
1	MH (Myocardial Infarction OR Shock, Cardiogenic OR Coronary Vasospasm OR Coronary Thrombosis OR Coronary Stenosis OR Angina Pectoris OR Acute Coronary Syndrome or Cardiac Patients OR Myocardial Ischemia)	64629
2	TI ("Myocardial infarct*" OR "heart infarct*" OR "MI" OR "heart attack*" OR "ischemic heart disease" OR "acute coronary syndrome" OR angina OR "coronary disease*" OR "coronary thrombos*" OR "coronary occlusion*" OR "coronary spasm*" OR "coronary aneurysm*" OR "coronary artery disease*" OR "coronary stenosis" OR "myocardial ischemia" OR "cardiogenic shock" OR "cardiac patient*" OR "cardiac infarct*" OR "ischemic cardiomyopathy") OR AB ("Myocardial infarct*" OR "heart infarct*" OR "MI" OR "heart attack*" OR "ischemic heart disease" OR "acute coronary syndrome" OR angina OR "coronary disease*" OR "coronary thrombos*" OR "coronary occlusion*" OR "coronary spasm*" OR "coronary aneurysm*" OR "coronary artery disease*" OR "coronary stenosis" OR "myocardial ischemia" OR "cardiogenic shock" OR "cardiac patient*" OR "cardiac infarct*" OR "ischemic cardiomyopathy")	79319
3	#1 OR #2	107202
Traumatic stress		
4	MH (Stress Disorders, Post-Traumatic OR Psychological Trauma OR Psychological Stress OR Post-Trauma Response)	21371
5	TI ("Post-traumatic stress disorder" OR "ptsd" OR "posttraumatic stress" OR "post traumatic stress" OR "traumatic stress symptom*" OR "stress-related symptom*" OR "emotional trauma*" OR "stress reaction*" OR "post-traumatic diagnosis" OR "trauma-related disorder*" OR "psychosocial distress" OR "posttraumatic neuros*" OR "post traumatic neuros*" OR "psychological trauma" OR "acute stress disorder" OR "traumatic stress*" OR "adjustment disorder*" OR "post-trauma response*" OR "psychological stress*" OR "posttraumatic psychic syndrome" OR "posttraumatic psychosis" OR "psychotrauma") OR AB ("Post-traumatic stress disorder" OR "ptsd" OR "posttraumatic stress" OR "post traumatic stress" OR "traumatic stress symptom*" OR "stress-related symptom*" OR "emotional trauma*" OR "stress reaction*" OR "post-traumatic diagnosis" OR "trauma-related disorder*" OR "psychosocial distress" OR "posttraumatic neuros*" OR "post traumatic neuros*" OR "psychological trauma" OR "acute stress disorder" OR "traumatic stress*" OR "adjustment disorder*" OR "post-trauma response*" OR "psychological stress*" OR "posttraumatic psychic syndrome" OR "posttraumatic psychosis" OR "psychotrauma")	21513
6	#4 OR #5	29898
Combined Sets		
7.	#3 AND #6	323

Limits		
8	#7 AND Filters: Publication date from 2000/01/01	302
9	#8 AND Filters: English	**295**

Cochrane Library: 1st Feb 2020 Traumatic Stress symptoms and Myocardial Infarction		
Reference #	**Search Terms**	**Results**
Myocardial Infarction		
1	[mh "Myocardial Infarction"] OR [mh "Angina Pectoris"] OR [mh "Coronary Disease"] OR [mh "Acute Coronary Syndrome"] OR [mh ^"Myocardial Ischemia"]	27679
2	(Myocardial NEXT infarct* OR heart NEXT infarct* OR MI OR heart NEXT attack* OR "ischemic heart disease" OR "acute coronary Syndrome" OR angina OR coronary NEXT disease* OR coronary NEXT thrombos* OR coronary NEXT occlusion* OR coronary NEXT spasm* OR coronary NEXT aneurysm* OR coronary artery NEXT disease* OR "coronary stenosis" OR "myocardial ischemia" OR "cardiogenic shock" OR cardiac NEXT patient* OR cardiac NEXT infarct* OR "ischemic cardiomyopathy"):ti,ab,kw	70454
3	#1 OR #2	70649
Traumatic stress		
4	[mh "Stress Disorders, Post-Traumatic"] OR [mh "Psychological Trauma"] OR [mh "Stress Disorders, Traumatic, Acute"] OR [mh ^"Stress Disorders, Traumatic"]	784
5	("Post-traumatic stress disorder" OR ptsd OR "posttraumatic stress" OR "post traumatic stress" OR traumatic stress NEXT symptom* OR stress-related NEXT symptom* OR emotional NEXT trauma* OR stress NEXT reaction* OR "post-traumatic diagnosis" OR trauma-related NEXT disorder* OR "psychosocial distress" OR posttraumatic NEXT neuros* OR post traumatic NEXT neuros* OR "psychological trauma" OR "acute stress disorder" OR traumatic NEXT stress* OR adjustment NEXT disorder* OR post-trauma NEXT response* OR psychological NEXT stress* OR "posttraumatic psychic syndrome" OR "posttraumatic psychosis" OR psychotrauma):ti,ab,kw	7893
6	#4 OR #5	7958
Combined Sets		
7.	#3 AND #6	130
Limits		
8	#7 AND Filters: Publication date from 2000/01/01	**118**
9	#8 AND Filters: English	

Embase: 1st Feb 2020 Traumatic Stress symptoms and Myocardial Infarction		
Reference #	**Search Terms**	**Results**
Myocardial Infarction		
1	'heart infarction'/exp OR 'acute coronary syndrome'/exp OR 'coronary artery occlusion'/exp OR 'coronary artery obstruction'/exp OR 'cardiogenic shock'/exp OR 'angina pectoris'/exp OR 'coronary artery spasm'/exp OR 'coronary artery thrombosis'/exp OR 'ischemic cardiomyopathy'/exp OR 'silent myocardial ischemia'/exp OR 'coronary artery aneurysm'/exp OR 'ischemic heart disease'/exp	690 972
2	'Myocardial infarct*':ti,ab OR 'heart infarct*':ti,ab OR 'MI':ti,ab OR 'heart attack*':ti,ab OR 'ischemic heart disease':ti,ab OR 'acute coronary syndrome':ti,ab OR angina:ti,ab OR 'coronary disease*':ti,ab OR 'coronary thrombos*':ti,ab OR 'coronary occlusion*':ti,ab OR 'coronary spasm*':ti,ab OR 'coronary aneurysm*':ti,ab OR 'coronary artery disease*':ti,ab OR 'coronary stenosis':ti,ab OR 'myocardial ischemia':ti,ab OR 'cardiogenic shock':ti,ab OR 'cardiac patient':ti,ab OR 'cardiac infarct*':ti,ab OR 'ischemic cardiomyopathy':ti,ab	557 156
3	#1 OR #2	845 864
Traumatic stress		
4	'posttraumatic stress disorder'/exp OR 'acute stress disorder'/exp OR 'psychotrauma'/exp OR 'adjustment disorder'/exp	69 535
5	'Post-traumatic stress disorder':ti,ab OR 'ptsd':ti,ab OR 'posttraumatic stress':ti,ab OR 'post traumatic stress':ti,ab OR 'traumatic stress symptom*':ti,ab OR 'stress-related symptom*':ti,ab OR 'emotional trauma*':ti,ab OR 'stress reaction*':ti,ab OR 'post-traumatic diagnosis':ti,ab OR 'trauma-related disorder*':ti,ab OR 'psychosocial distress':ti,ab OR 'posttraumatic neuros*':ti,ab OR 'post traumatic neuros*':ti,ab OR 'psychological trauma':ti,ab OR 'acute stress disorder':ti,ab OR 'traumatic stress*':ti,ab OR 'adjustment disorder*':ti,ab OR 'post-trauma response*':ti,ab OR 'psychological stress':ti,ab OR 'posttraumatic psychic syndrome':ti,ab OR 'posttraumatic psychosis':ti,ab OR 'psychotrauma':ti,ab	65 457
6	#4 OR #5	90 843
Combined Sets		
7.	#3 AND #6	1 570
Limits		
8	#7 AND Filters: Publication date from 2000/01/01	1392
9	#8 AND Filters: English	**1318**

PsycINFO: 1st Feb 2020 Traumatic Stress symptoms and Myocardial Infarction		
Reference #	**Search Terms**	**Results**
Myocardial Infarction		
1	(myocardial infarctions OR coronary thromboses OR angina pectoris OR Heart disorders).sh	12183
2	(Myocardial infarct* OR heart infarct* OR MI OR heart attack* OR ischemic heart disease OR acute coronary Syndrome OR angina OR coronary disease* OR coronary thrombos* OR coronary occlusion* OR coronary spasm* OR coronary aneurysm* OR coronary artery disease* OR coronary stenosis OR myocardial ischemia OR cardiogenic shock OR cardiac patient* OR cardiac infarct* OR ischemic cardiomyopathy).ti,ab	13653
3	#1 OR #2	20994
Traumatic stress		
4	(Posttraumatic stress disorder OR PTSD OR Complex PTSD OR Acute Stress Disorder OR Adjustment Disorders OR Emotional Trauma OR Post-traumatic stress OR Stress Reactions OR Traumatic Neurosis OR Traumatic stress OR DESNOS).sh	53924
5	(Post-traumatic stress disorder OR ptsd OR posttraumatic stress OR post traumatic stress OR traumatic stress symptom* OR stress-related symptom* OR emotional trauma* OR stress reaction* OR post-traumatic diagnosis OR trauma-related disorder* OR psychosocial distress OR posttraumatic neuros* OR post traumatic neuros* OR psychological trauma OR acute stress disorder OR traumatic stress* OR adjustment disorder* OR post-trauma response* OR psychological stress* OR posttraumatic psychic syndrome OR posttraumatic psychosis OR psychotrauma).ti,ab	55233
6	#4 OR #5	75124
Combined Sets		
7.	#3 AND #6	644
Limits		
8	#7 AND Filters: Publication date from 2000/01/01	**467**
9	#8 AND Filters: English	

PTSDPubs: 1st Feb 2020 Traumatic Stress and Myocardial Infarction		
Reference #	**Search Terms**	**Results**
Myocardial Infarction		
1	MAINSUBJECT.EXACT.EXPLODE("Cardiovascular Diseases")	502
2	AB,TI(Myocardial infarct* OR heart infarct* OR MI OR heart attack* OR ischemic heart disease OR acute coronary Syndrome OR angina OR coronary disease* OR coronary thrombos* OR coronary occlusion* OR coronary spasm* OR coronary aneurysm* OR coronary artery disease* OR coronary stenosis OR myocardial ischemia OR cardiogenic shock OR cardiac patient* OR cardiac infarct* OR ischemic cardiomyopathy)	481
3	#1 OR #2	735
Traumatic stress		
4	MAINSUBJECT.EXACT.EXPLODE("PTSD") OR MAINSUBJECT.EXACT.EXPLODE("Acute Stress Disorder") OR MAINSUBJECT.EXACT("Stress Disorders")	40437
5	AB,TI(Post-traumatic stress disorder OR ptsd OR posttraumatic stress OR post traumatic stress OR traumatic stress symptom* OR stress-related symptom* OR emotional trauma* OR stress reaction* OR post-traumatic diagnosis OR trauma-related disorder* OR psychosocial distress OR posttraumatic neuros* OR post traumatic neuros* OR psychological trauma OR acute stress disorder OR traumatic stress* OR adjustment disorder* OR post-trauma response* OR psychological stress* OR posttraumatic psychic syndrome OR posttraumatic psychosis OR psychotrauma)	40562
6	#4 OR #5	46618
Combined Sets		
7.	#3 AND #6	631
Limits		
8	#7 AND Filters: Publication date from 2000/01/01	567
9	#8 AND Filters: English	553
10	#9 AND Filters: Peer-Reviewed	**446**

PubMed: 1st Feb 2020 Traumatic Stress and Myocardial Infarction		
Reference #	**Search Terms**	**Results**
Myocardial Infarction		
1	"myocardial infarction"[mh noexp] OR “myocardial ischemia”[mh:noexp] OR “acute coronary syndrome”[mh] OR “angina pectoris”[mh:noexp] OR “angina stable”[mh:noexp] OR “angina unstable”[mh:noexp] OR “coronary disease”[mh:noexp] OR “coronary aneurysm”[mh noexp] OR “coronary artery disease”[mh:noexp] OR “coronary stenosis”[mh:noexp] OR “coronary occlusion”[mh:noexp] OR “coronary thrombosis”[mh:noexp] OR “coronary vasospasm”[mh:noexp] OR “anterior Wall Myocardial Infarction”[mh noexp] OR “inferior Wall Myocardial infarction”[mh:noexp] OR “non-ST Elevated Myocardial infarction”[mh:noexp] OR “shock cardiogenic”[mh noexp] OR “st elevation myocardial infarction”[mh:noexp]	404553
2	Myocardial infarct*[tiab] OR heart infarct*[tiab] OR MI[tiab] OR heart attack*[tiab] OR ischemic heart disease[tiab] OR acute coronary Syndrome[tiab] OR angina[tiab] OR coronary disease*[tiab] OR coronary thrombos*[tiab] OR coronary occlusion*[tiab] OR coronary spasm*[tiab] OR coronary aneurysm*[tiab] OR coronary artery disease*[tiab] OR coronary stenosis[tiab] OR myocardial ischemia[tiab] OR cardiogenic shock[tiab] OR cardiac patient*[tiab] OR cardiac infarct*[tiab] OR ischemic cardiomyopathy[tiab]	387163
3	#1 OR #2	545727
Traumatic stress		
4	“Stress Disorders, Traumatic”[mh:noexp] OR "Stress Disorders, Post-Traumatic"[mh:noexp] OR “Stress Disorders, Traumatic, Acute”[mh:noexp] OR “Psychological Trauma”[mh noexp]	33031
5	Post-traumatic stress disorder[tiab] OR ptsd[tiab] OR posttraumatic stress[tiab] OR post traumatic stress[tiab] OR traumatic stress symptom*[tiab] OR stress-related symptom*[tiab] OR emotional trauma*[tiab] OR stress reaction*[tiab] OR post-traumatic diagnosis[tiab] OR trauma-related disorder*[tiab] OR psychosocial distress[tiab] OR posttraumatic neuros*[tiab] OR post traumatic neuros*[tiab] OR psychological trauma[tiab] OR acute stress disorder[tiab] OR traumatic stress*[tiab] OR adjustment disorder*[tiab] OR post-trauma response*[tiab] OR psychological stress*[tiab] OR posttraumatic psychic syndrome[tiab] OR posttraumatic psychosis[tiab] OR psychotrauma[tiab]	54599
6	#4 OR #5	63454
Combined Sets		
7.	#3 AND #6	849
Limits		
8	#7 AND Filters: Publication date from 2000/01/01	673
9	#8 AND Filters: English	**629**

Appendix B: Data Extraction Form

Study Overview			
Author(s):		Year:	
Study Design:		Country:	
Setting:			
Study Funding (if any)			
Conflicts of interest (if any)			
Population Characteristics			
Sample Size:		Sex Ratio: n(%)	
Mean Age (range):		Race/Ethnicity: n(%)	
Recruitment Method			
Inclusion Criteria			
Exclusion Criteria			
Other relevant socio-demographic:			
Methodology			
Description of data collection			

Participation Rate of eligible persons			
Follow-up? If yes, describe details	Y / N	Details:	
Attrition Rate n(%)			
Statistical Analyses Utilised			
Condition/Baseline Characteristics: Myocardial Infarction			
Reference Standard			
Evaluation tool (If N/A, list source of data)		Evaluation method	
Evaluating personnel		Length of time since initial diagnosis	
Outcome of interest: Traumatic Stress			
Reference Standard			
Evaluation tool (If N/A, list source of data)		Evaluation method	
Evaluating personnel		Length of time since initial diagnosis	
Results			
Classification of outcome			
Prevalence %(n)			
Symptoms features			

Symptoms Trajectory (if available)	
Confounding Variables	
Limitations	

Appendix C: Critical Appraisal Tool

Criteria	Yes	No	Other (CD, NR, NA)*
1. Was the research question or objective in this paper clearly stated?			
2. Was the study population clearly specified and defined?			
3. Was the participation rate of eligible persons at least 50%?			
4. Were all the subjects selected or recruited from the same or similar populations (including the same time period)? Were inclusion and exclusion criteria for being in the study prespecified and applied uniformly to all participants?			
5. Was a sample size justification, power description, or variance and effect estimates provided?			
6. For the analyses in this paper, were the exposure(s) of interest measured prior to the outcome(s) being measured?			
7. Was the timeframe sufficient so that one could reasonably expect to see an association between exposure and outcome if it existed?			
8. For exposures that can vary in amount or level, did the study examine different levels of the exposure as related to the outcome (e.g., categories of exposure, or exposure measured as continuous variable)?			
9. Were the exposure measures (independent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?			
10. Was the exposure(s) assessed more than once over time?			
11. Were the outcome measures (dependent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?			
12. Were the outcome assessors blinded to the exposure status of participants?			
13. Was loss to follow-up after baseline 20% or less?			
14. Were key potential confounding variables measured and adjusted statistically for their impact on the relationship between exposure(s) and outcome(s)?			

*CD, cannot determine; NA, not applicable; NR, not reported

Appendix D: Author Guidelines for the *Journal of Traumatic Stress*

Author Guidelines

To read the journal's position on open science practice, please find the full statement here.

Author Guidelines

1. Online Submissions: The *Journal of Traumatic Stress*accepts submission of manuscripts online at:

http://mc.manuscriptcentral.com/jots

Information about how to create an account or submit a manuscript may be found online on the Manuscript Central homepage in the "User Tutorials" section or, on the Author Dashboard, via the "Help" menu in the upper right corner of the screen. Personal assistance also is available by calling 434-964-4100.

2. Article Formats: Three article formats are accepted for consideration by JTS. All page counts should include references, tables, and figures. *Regular articles*(30 pages maximum, inclusive of all text, abstract, references, tables, and figures) include research studies, quantitative systematic reviews, and theoretical articles. Purely descriptive articles or narrative-based literature reviews are rarely accepted. In extraordinary circumstances, the editors may consider longer manuscripts that describe highly complex designs or statistical procedures but authors should seek approval prior to submitting manuscripts longer than 30 pages. *Brief reports* (18 pages maximum) are appropriate for pilot studies or uncontrolled trials of an intervention, preliminary data on a new problem or population, condensed findings from a study that does not merit a full article, or methodologically oriented papers that replicate findings in new populations or report preliminary data on new instruments. *Commentaries* (1,000 words or less) involve responses to previously published *JTS* Response commentaries, submitted no later than 8 weeks after the original article is published (12 weeks if outside the U.S.), must be content-directed and use tactful language. The original author is given the opportunity to respond to accepted commentaries.

3. Double-Blind Review: As of January 1, 2017, the Journal of Traumatic Stress utilizes a double-blind review process in which reviewers receive manuscripts with no authors' names or affiliations listed in order to ensure unbiased review. To facilitate blinded review, the title page should be uploaded as a separate document from the body of the manuscript, identified as "Title Page," and should include the title of the article, the running head (maximum 50 characters) in uppercase flush left, author(s) byline and institutional affiliation, and author note (see pp. 30-37 of the APA 7th manual). Within the main body of the manuscript, tables, and figures, authors should ensure that any identifying information (i.e., author names, affiliations, institutions where the work was performed, university whose ethics committee approved the project) is blinded; a simple way to accomplish this is by replacing the identifying text with the phrase "[edited out for blind review]". In addition, language should be used that avoids revealing the identity of the authors; e.g., rather than stating, "In other research by our lab (Bennett & Kerig, 2014), we found ..." use phrases such as, "In a previous study, Bennett and Kerig (2014) found ..." Please note that if you have uploaded the files correctly, you will **not** be able to view the title page in the PDF and HTML proofs of your manuscript; however, the Editor and JTS editorial office staff can view this information.

4. Preferred and Non-Preferred Reviewers: During the submission process, authors may suggest the names of preferred reviewers; authors also may request that specific individuals not be selected as reviewers.

5. Publication Style: JTS follows the style recommendations of the 2020 *Publication Manual of the American Psychological Association*(APA; 7th edition) and submitted manuscripts must conform to these formatting guidelines. Manuscripts should use non-sexist language. Manuscripts must be formatted using letter or A4 page size, with 1 inch (2.54 cm) margins on all sides, in an APA-approved font (i.e., 10-point Lucinda Sans Unicode or Computer Modern; 11-point Arial, Calibri, or Georgia; 12-point Times New Roman). All text within figures should be formatted in a sans serif font (e.g., Arial or Calibri) with a type size between 8 and 14 points. The title page, abstract, references, table title and notes, and figure title and notes should be double-spaced; text within tables and figures can be single or double spaced based on the layout of the information. Submit your manuscript in .doc or .docx format, ***not as a PDF***.

For assistance with APA style, in addition to consulting the manual itself, please note these helpful online sources that are freely available: https://apastyle.apa.org/style-grammar-guidelines/index and https://owl.english.purdue.edu/owl/section/2/10/

6. APA and JTS Style Pointers: In addition to consulting the APA 7th edition Publication Manual, the resources indexed above, and the JTS Style Sheet posted online, please consider these pointers when formatting each section of the manuscript:

1. **Tense:** Throughout the manuscript, please use past tense for everything that has already happened, including the collection and analyses of the data being reported.
2. **Abstract:** The Main Document of the manuscript should begin with an abstract no longer than 250 words, placed on a separate page. In addition, JTS house style requires the reporting of an effect size for each finding discussed in the abstract; if there are many findings, present the range.
3. **Participants:** Please include in this subsection of the Method section information on sample characteristics, subsample comparisons, and analyses that describe the sample but are not focused on testing the hypotheses that are the aims of your manuscript.

4. **Procedure:** Please describe the procedure in sufficient detail so that it could be comprehended and replicated by another investigator. Identify by name the IRB or ethics committee (edited out for blind review in the submitted manuscript) that approved the research, and the manner in which consent was obtained.
5. **Measures:** In addition to providing citations, psychometric, and validation data for each measure administered, please provide coefficient alpha from your data for each measure for which this is appropriate.
6. **Data Analysis:** Include a separate subsection with this header in the Method section in which you describe the analyses performed, the software program(s) used, and make an explicit statement about missing data in your data set. If there are no missing data, so state; otherwise describe the extent of missing data and how they were handled in the data analyses.
7. **Results** (and throughout): Present percentages to 1 decimal place, means and *SD*s to 2 decimal places, and exact *p* values to 3 decimal places except for any < .001. Include leading zeros (e.g., 0.92) when reporting any statistic that can be greater than 1.00 (or less than -1.00). For example, there is no leading zero used when reporting correlations, coefficient alphas, standardized betas, *p* values, or fit indices (e.g., *r* = .47, not 0.47). Report effect sizes for analyses conducted wherever possible and appropriate.
8. **References:** Format the references using APA 7th edition style: (a) begin the reference list on a new page following the text, (b) double-space, (c) use hanging indent format, (d) italicize the journal name or book title, and (e) list alphabetically by last name of first author. If a reference has a Digital Object Identifier (doi), it must be included as the last element of the reference

- ***Journal Article:***

Grady, J. S., Her, M., Moreno, G., Perez, C., & Yelinek, J. (2019). Emotions in storybooks: A comparison of storybooks that represent ethnic and racial groups in the United States? *Psychology of Popular Media Culture, 8*(3), 207–217. https://doi.org/10.1037/ppm0000185

- ***Book:***

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Erlbaum.

- ***Book Chapter:***

Meehl, P. E. (2006). The power of quantitative thinking. In N. G. Waller, L. J. Yonce, W. M. Grove, D. Faust, & M. F. Lenzenweger (Eds.), *Essays on the practice of scientific psychology* (pp. 433–444). Erlbaum.

Footnotes: Footnotes should be avoided. When their use is absolutely necessary, footnotes should be formatted in APA style and placed on a separate page after the reference list and before any tables.

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