International Psychogeriatrics (2011), 23:6, 858–868 © International Psychogeriatric Association 2011
doi:10.1017/S1041610211000366

REVIEW

PTSD in the elderly: the interaction between trauma and aging

Leann Kimberly Lapp,[1,2] Catherine Agbokou[1] and Florian Ferreri[1]

[1] *Service de Psychiatrie et de Psychologie Médicale de l'Adulte, Hôpital Saint-Antoine, Université Pierre et Marie Curie, Sorbonne Universités, Paris VI, Paris, France*
[2] *Université Paris 7 - Denis Diderot, Paris, France*

ABSTRACT

Background: Because an increasingly large cohort of individuals is approaching their elderly years, there is concern about how the healthcare system will cope with the greater demands placed upon it. One area of concern is the impact of trauma and post traumatic stress disorder (PTSD) in the aged. Although several reviews have highlighted the lack of knowledge and research on the topic, there still remain gaps in the literature. Nevertheless, some recent behavioral, endocrinological and neuroimaging studies may provide new insights into the discussion. The central aims of this paper are to summarize the etiological, epidemiological and clinical aspects of PTSD, trauma, and the elderly, and to integrate this knowledge with (i) what is known about PTSD in adults, and (ii) the behavioral, hormonal and cerebral changes associated with healthy aging.

Methods: A comprehensive search was performed with ISI Web of Science and PubMed for articles pertinent to the psychology and biology of PTSD, trauma, and the elderly.

Results: There exist both significant similarities and differences between adults and elderly with PTSD concerning cognitive and biological profile. Evidence suggests that PTSD in the elderly does not follow a simple clinical trajectory.

Conclusions: PTSD in the elderly must be considered within the context of normal aging. Strong claims about an interaction between PTSD and aging are difficult to make due to sample heterogeneity, but it is clear that PTSD in this age group presents unique aspects not seen in younger cohorts. Further research must integrate their studies with the biological, psychological, and social changes already associated with the aging process.

Key words: trauma, PTSD, elderly, aging

Introduction

With a large cohort of the population approaching their autumn years, it is imperative that the effect of health conditions on the elderly are better understood. In addition to the physical ailments that are often associated with aging, mental health disorders must also be considered. Unfortunately, research is lacking with respect to how certain mental health problems manifest themselves in the context of old age. One such disorder is post traumatic stress disorder (PTSD), which may emerge as a consequence of a traumatic event, even if the event occurred decades previously (Hiskey *et al.*, 2008). With more and more survivors of the various wars of the twentieth century entering old age, and – with longer lifespans – more individuals having occasions to be exposed to traumatic experiences, further understanding of PTSD in the elderly is necessary. Indeed, some have called the effects of trauma the "hidden variables" in the lives of older adults (Cook and O'Donnell, 2005).

While PTSD is well-described in the general population, the literature concerning PTSD in the elderly is patchy (Averill and Beck, 2000; Busuttil, 2004; Cook and O'Donnell, 2005). Nevertheless, there is evidence that PTSD is a significant issue for older adults. Elderly individuals with full and subthreshold PTSD suffer significant impairment in daily life, are less satisfied, receive less than optimum care, and report older subjective age, which is in itself related to greater health problems (van Zelst *et al.*, 2006; Solomon *et al.*, 2009). For instance, PTSD has been associated with an increased risk of coronary heart disease in older men

Correspondence should be addressed to: Leann K. Lapp, Service de Psychiatrie et de Psychologie Médicale de l'Adulte, Hôpital Saint-Antoine, Université Pierre et Marie Curie, Paris VI, 184 rue du Faubourg Saint-Antoine, 75012 Paris, France. Phone: +33 1 49 28 26 35; Fax +33 1 49 28 20 10. Email: leannklapp@gmail.com. Received 29 Sep 2010; revision requested 24 Nov 2010; revised version received 2 Feb 2011; accepted 2 Feb 2011. First published online 22 March 2011.

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

(Kubzansky *et al.*, 2007) and an increased risk of comorbidity such as depression and anxiety (Spitzer *et al.*, 2008). Most research has been conducted with Holocaust survivors, former prisoners of war (POWs) and combat veterans, but recent research has identified other types of potentially traumatic events experienced by the elderly such as the death of a loved one, physical injury, and serious disease (Elklit and O'Connor, 2005; Chung *et al.*, 2008; 2009).

There is evidence of similarities between PTSD in the general population and PTSD in the elderly, but there are also indications that PTSD in old age presents unique features. In the adult literature, PTSD is associated with various impairments including attention, memory, and other executive functions (Moore, 2009), as well as altered stress hormones levels and changes in brain structure (Yehuda, 2009). These are many of the same features that describe normal cognitive aging (Lupien *et al.*, 2007). In fact, some studies of PTSD in the elderly have suggested that this disorder represents accelerated aging (Golier *et al.*, 2002; Yehuda *et al.*, 2002; Yaffe *et al.*, 2010). Thus, it is imperative that in contrast to PTSD in the adult population, PTSD in the elderly be understood in developmental terms. That is, we must superimpose PTSD onto the background of the normal biological, psychological and social changes that are seen concurrently with aging. This review attempts to collate the existing prevalence and longitudinal studies, and touches upon risk factors for PTSD in the elderly. We then direct the focus of the paper to explore how PTSD can be understood within the context of cognitive aging. To this end, a comprehensive search was performed with ISI Web of Science and PubMed for articles pertinent to the psychology and biology of PTSD, trauma and the elderly. The results of this search are presented below, followed by a discussion of caveats and further directions to be considered in this field of research.

Results

Presentation

PTSD is traditionally classified as an anxiety disorder in DSM-IV, but is also conceptualized as a disorder of memory: one of the cardinal features is the repeated reliving of the traumatic event (American Psychiatric Association, 1994). In order for a clinical diagnosis to be made, the individual must have experienced a traumatic event during which they felt intense fear, helplessness or horror. Subsequently, survivors may present three distinct clusters of symptoms that characterize PTSD: re-experiencing of the traumatic event (e.g. nightmares, intrusive thoughts), numbing and avoidance of stimuli associated with the trauma, and hyperarousal (e.g. problems with sleeping, exaggerated startle response). Formal diagnosis is only made after symptoms have been experienced for at least one month and have caused significant distress and functional impairment in daily living (American Psychiatric Association, 1994).

The elderly may be at more risk, or at least may present additional complexity, regarding the development of psychopathology following a traumatic event. Compared with the general population, the elderly more often suffer from chronic physical and mental disorders; social networks tend to narrow with time; and financial means are often reduced. All of these factors, in addition to the social *zeitgeist* of the time and particular generation (Averill and Beck, 2000), must be taken into account when considering PTSD in this population. Nevertheless, most research into PTSD excludes older adults (Spitzer *et al.*, 2008).

PTSD in the elderly is noteworthy in that it can emerge and develop in several ways. Several possible forms of presentation have been postulated (Busuttil, 2004). First is *de novo*, due to a trauma experienced in old age. Second is chronic, where the trauma is experienced much earlier in life, and leads to PTSD that follows a chronic course with possible periods of fluctuation with respect to symptom severity. Third, PTSD can manifest itself with delayed onset; after years of absent, low or well-controlled symptoms, full-blown PTSD surfaces in old age. This last characterization is not without controversy, however. Some maintain that PTSD with delayed onset is in fact a misnomer – the apparent absence of PTSD is actually due to under-reporting, delayed referral, exacerbation of subclinical PTSD, or misdiagnosis (Hiskey *et al.*, 2008). Fourth is chronic PTSD with enduring personality changes, and lastly, is chronic complex PTSD with multiple psychiatric disturbances.

Several hypotheses regarding PTSD in the elderly have been advanced, and evidence can be found for each. The opposing hypotheses can be described as (i) the inoculation hypothesis, whereby older adults are more resistant because prior experience of traumatic events is protective (Eysenck, 1983), and (ii) the vulnerability hypothesis, which states that the accumulation of previous traumatic experience, combined with the additional stresses of aging, may render older people more vulnerable when faced with a novel stressful event (Solomon and Ginzburg, 1998). The crisis resolution hypothesis ostensibly accounts for the discrepancy between the aforementioned theories: previous exposure to extreme stress can

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

either predispose or protect the older adult against subsequent traumatic experience depending on how that prior experienced was resolved (Hantman and Solomon, 2007). A cognitive aging hypothesis has been put forward to describe the re-emergence of PTSD that can occur in old age (Floyd *et al.*, 2002). In this explanation, the age-related decreases in attention and memory function account for the possible recurrence of PTSD (Floyd *et al.*, 2002). Likewise, neurodegenerative underpinnings of aging have been implicated in explaining the recurrence of PTSD in old age (Ruzich *et al.*, 2005).

Possible explanations for delayed onset PTSD include (i) the fact that, because of decreased physical and mental resilience over time, the elderly become less able to "ward off" PTSD (Aarts and Op den Velde, 1996); (ii) age-related stressors such as retirement or bereavement may induce the presentation of PTSD; (iii) in old age, people start to reflect more on their lives, often in a search for life meaning, and may increase their focus on traumatic memories (Erikson, 1966); and (iv) the psychodynamic view that re-emergence of PTSD is due to a failure of repression (Horowitz, 1974). It must be noted that several different terms can be found in the literature, including "delayed onset", "re-emergent", or "re-activated" PTSD (Port *et al.*, 2001; Bramsen *et al.*, 2006; Hiskey *et al.*, 2008). As Hiskey and colleagues note, a clear definition of what is meant by recurrent or delayed onset PTSD must be constructed in order for accurate prevalence rates to be measured and the underlying mechanisms to be discovered (Hiskey *et al.*, 2008).

Another unclear concept in the literature is the definition of trauma itself with respect to the elderly population. An aged individual will generally have experienced a lifetime of stressful events, more so than their younger counterparts. Does this change the threshold for which an event is experienced as traumatic? Are the same events experienced as traumatic for the elderly compared with younger individuals? Methodologically, it must be agreed upon as to how "trauma" is operationalized: do we include all potentially negative events as traumatic, or simply those that are beyond the realm of typical experience? This question is not new within the adult PTSD literature (for example, see McNally, 2003); however, there is potentially an additional dimension to this discussion when the context of aging is introduced.

Prevalence and course

Epidemiological studies indicate significant lifetime prevalence rates for trauma exposure in the general population, with men consistently at higher risk than women (Kessler *et al.*, 1995). Lifetime prevalence rates for PTSD from South America (Zlotnick *et al.*, 2006), North America (Kessler *et al.*, 1995; Norris *et al.*, 2003; van Ameringen *et al.*, 2008), and Europe (Frans *et al.*, 2005; de Vries and Olff, 2009) have reported rates between 4.4% and 11.2%. Most of the prevalence studies have not examined elderly individuals; however, a handful of recent studies has begun to address this gap. A six-month prevalence study found rates of 0.9% for full PTSD and 13.1% for subthreshold PTSD in a group of older adults (van Zelst *et al.*, 2003). More recently, a community study comprising three groups (aged $\leq$44, 45–64, $\geq$65 years) found that lifetime prevalence of PTSD in the elderly was 3.1% and did not significantly differ from the other age groups (Spitzer *et al.*, 2008). Aged individuals who endorse a diagnosis of PTSD were found to meet mild or moderate severity rather than extreme symptom severity (Yehuda *et al.*, 1997). Overall, these results suggest that older adults present lower rates of PTSD than the general population. However, this conclusion should be interpreted with care. Some have cautioned that these rates may be artificially low due to under-reporting or because the elderly may attribute or experience their PTSD symptoms as somatic rather than psychological (van Zelst *et al.*, 2003; Owens *et al.*, 2005). Additionally, changes in response to stress with age may affect both the experience and presentation of PTSD, and older adults may attribute their symptoms to aging itself rather than to trauma (Owens *et al.*, 2005).

The paucity of cohort studies conducted after a traumatic event makes it premature to conclude that the elderly are at a greater risk of PTSD. Still, the extant literature seems to suggest that, compared with younger adults, they are not (however, for contrary findings, see Lee *et al.*, 2006; Liu *et al.*, 2006). In community samples comprised of younger, middle aged, and elderly individuals, there was no significant difference in PTSD symptoms between groups after technological disasters (Chung *et al.*, 2004), natural disaster (Kohn *et al.*, 2005), and interpersonal violence (Acierno *et al.*, 2002). A community prevalence study that measured trauma exposure across the lifespan uncovered a differential pattern between men and women (Creamer and Parslow, 2008). Traumatic exposure over the lifespan followed an inverted-U pattern for women and a linear pattern for men, the difference being explained by male combat experience. Overall, this study found that the net prevalence of PTSD declined with age (Creamer and Parslow, 2008).

PTSD prevalence has also been studied specifically in older adults after particular types of

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

events. These include after myocardial infarction (Chung *et al.*, 2006), spinal cord injury (Chung *et al.*, 2006), bereavement (Elklit and O'Connor, 2005), and falls (Chung *et al.*, 2009), and these studies advocate the consideration of these events as being potentially traumatic in the elderly. Another study has investigated the possibility of reactivation of PTSD after a secondary trauma (Bramsen *et al.*, 2006). Survivors of World War II who experienced a fireworks disaster in the Netherlands were compared with WWII survivors who did not, and were assessed both before and after the fireworks disaster. PTSD symptoms as well as war-related re-experiencing increased in the former group, and supported the conclusion that reactivation of previous traumatic events can occur. Additionally, there was no evidence for delayed onset PTSD, consistent with several other studies that do not support an association between recent life events and an increase in PTSD (Dirkzwager *et al.*, 2001; van Achterberg *et al.*, 2001; Port *et al.*, 2002; but see Op den Velde *et al.*, 1993; Macleod, 1994; Hiskey *et al.*, 2008).

The handful of longitudinal studies reveals that the course of PTSD into old age may not necessarily be summarized by a simple trajectory. Rather, different trajectories may exist depending on the type of trauma experienced, duration of trauma, and other factors. There is evidence for symptom stability (Dirkzwager *et al.*, 2001), exacerbation (Port *et al.*, 2001; Joffe *et al.*, 2003; Solomon and Mikulincer, 2006; Trappler *et al.*, 2007), reduction (Kluznick *et al.*, 1986; Tennant *et al.*, 1997), and fluctuation (Dirkzwager *et al.*, 2001); in short, the literature is unsettled. In trying to make sense of such equivocal data, Yehuda and colleagues suggested that an array of trajectories may be possible, related to particular factors or individual differences (Yehuda *et al.*, 2009). Their ten-year longitudinal study of Holocaust survivors supported this: there was an overall reduction in symptoms with time, but, more accurately, those who endorsed PTSD at time 1 experienced a reduction in symptom severity by time 2. Meanwhile those without PTSD at time 1 showed an increase in symptom severity by time 2. A small portion of the sample presented delayed onset PTSD at time 2; however this subset did have higher symptom severity at time 1 than the others who did not develop PTSD at time 2. The authors emphasize that the possibility of delayed onset PTSD complicates any projection of a simple trajectory for PTSD in this population. Furthermore, delayed onset PTSD may be understood as a redistribution of PTSD symptoms rather than a dichotomous measure of the presence or lack of PTSD symptoms *per se* (Yehuda *et al.*, 2009).

A final note of caution is suggested when interpreting the aforementioned prevalence studies because of the possibility of sample bias. It is conceivable that the elderly populations investigated in the handful of epidemiological studies performed are more resilient in some respect (for example, biological, psychological, attachment style, etc.) compared with their younger counterparts, for presumably there are many individuals who have not fared as well as they have over the years. This potentially significant factor should be kept in mind when considering PTSD in this population, as well as when comparing findings with younger cohorts.

Risk factors

PTSD is not an inevitable consequence of a traumatic experience; most people in fact recover. The mystery remains as to why some people who are exposed to traumatic stress develop PTSD while others do not, and much research has sought to identify the particular factors that may explain the discrepant reactions. Risk factors associated with the development of PTSD are well described in the general PTSD literature (Brewin *et al.*, 2000; Ozer *et al.*, 2003). Overall, peri- and post-traumatic factors seem to be stronger predictors of PTSD than pre-traumatic factors. Factors more proximal to the trauma, such as perceived life threat, perceived support, trauma severity, peri-traumatic emotionality, and peri-traumatic dissociation, are stronger predictors than more distal factors such as prior adjustment, prior history of trauma, and family history of psychopathology (Brewin *et al.*, 2000; Ozer *et al.*, 2003). PTSD severity has also been related to particular cognitive decrements such as problems with episodic memory, working memory, verbal memory, and negative trauma-related and self-related appraisals (Ehring *et al.*, 2008; Field *et al.*, 2008), and there is evidence to suggest these features might represent premorbid traits (Moore, 2009). Several prospective studies suggest that negative cognitions directed towards the self prior to or immediately after the trauma may predict PTSD severity (Bryant and Guthrie, 2007; Ehring *et al.*, 2008).

As in the general PTSD literature, several risk factors have been specifically associated with PTSD in the elderly after an array of trauma types. These include being female (e.g. van Zelst *et al.*, 2003; Liu *et al.*, 2006) although gender is not always significant, for example (Brodaty *et al.*, 2004); adverse events in childhood (van Zelst *et al.*, 2003), low self-efficacy (van Zelst *et al.*, 2003), having lower IQ (e.g. Hart *et al.*, 2008), lower social support (Acierno *et al.*, 2002; Alpass *et al.*, 2004), current level of PTSD symptoms of spouse

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

(Bramsen *et al.*, 2002), use of immature defense mechanisms (Brodaty *et al.*, 2004), and personality factors such as neuroticism and antagonism (van Zelst *et al.*, 2003; Brodaty *et al.*, 2004; Chung *et al.*, 2006). Current cognitive status may also represent a noteworthy risk factor in the elderly population, especially with respect to the emergence or re-emergence of PTSD in old age after years of absent or well-controlled symptoms (Johnston, 2000; Mittal *et al.*, 2001; van Achterberg *et al.*, 2001; Floyd *et al.*, 2002; Cook *et al.*, 2003; Alpass *et al.*, 2004; Ruzich *et al.*, 2005).

An additional vulnerability when considering trauma across the lifespan is attachment style. Although discussion of attachment theory and PTSD in the literature is sparse, there is a basis for thinking about the reaction to traumatic events across the lifespan within the context of attachment (Cassidy and Mohr, 2001). Attachment theory was initially conceived of to explain, in both psychological and biological terms, how individuals respond to danger and threat (Bowlby, 1973). Later research has examined how early experiences with fear and separation relate to how adults cope with trauma later in life (e.g. Diamond and Fagundes, 2010). Differential attachment patterns established in childhood, adolescence, and adulthood may explain some of the individual variability in reactions to trauma in the elderly years, in terms of both vulnerability and resilience. In fact, recent research has considered dementia and aging in terms of the attachment styles established earlier in life as well as in the later years (Browne and Shlosberg, 2006). Other investigators have begun to study the relationship between attachment style and the functioning of the hypothalatmic-pituitary-adrenocortical axis, which is integrally affected in trauma, stress and PTSD (and will be discussed in later sections below) (Diamond and Fagundes, 2010).

PTSD and the interaction with healthy aging

PTSD, as with normal aging, is associated with an array of biological alterations and, at times, cognitive impairment. Certain biological features are similar between the elderly and younger samples with PTSD, while others deviate and seem more similar to the biological changes associated with normal aging. These findings have led investigators to postulate that there is a superimposition of aging and PTSD in certain respects, and that, in fact, the interaction between aging and PTSD may lead to accelerated aging in particular domains (e.g. Yaffe *et al.*, 2010). Although this hypothesis is by no means proven (e.g. Pitman, 2010), it is clear that PTSD in the elderly must nevertheless be considered against the backdrop of the expected behavioral, hormonal, and cerebral changes associated with aging. This discussion mainly concerns chronic PTSD, or PTSD that stems from a traumatic event experienced many years earlier, since *de novo* PTSD seems to be associated with similar or even lower prevalence levels with respect to younger cohorts, as expounded in the preceding sections.

Case studies

The first indications that PTSD and aging may have a particular relationship came from several case series. In general, they described how select individuals, after decades of absent or well-controlled symptoms, developed PTSD in old age that coincided with the onset of cognitive decline or neurological illness. Mittal *et al.* (2001) documented three cases of war-related PTSD that was under control until the development of Alzheimer's disease, vascular, and alcohol-related dementia, respectively. The authors highlight the fact that these patients, despite undergoing significant cognitive decline, experienced intense recollections of their traumatic event, and hypothesize that the aging process may disinhibit the neural pathways and lead to a worsening of traumatic memories. Similar cases have been noted in Holocaust survivors (Grossman *et al.*, 2004) and combat veterans (Johnston, 2000). The cases of a Holocaust survivor, a World War II veteran, and a survivor of the *Titanic*, who all showed mild symptoms during the majority of their lives, only to exhibit significant worsening of PTSD symptoms with the onset of dementia have been described by van Achterberg *et al.* (2001). In none of these cases can the psychosocial stressors associated with aging, such as retirement or death of a loved one, account for the aggravation of PTSD symptoms. These results suggest that trauma-related memories are perhaps more resistant to the effects of dementia, and that the dementing process may interfere with cognitive strategies or defense mechanisms previously employed to keep traumatic memories in check (van Achterberg *et al.*, 2001). However, in 15 elderly combat veterans who reported delayed onset PTSD, their disorder coincided with the onset of unrelated medical problems, psychosocial stress related to aging, and/or mild cognitive impairment (Ruzich *et al.*, 2005). The authors suggest that age-related psychosocial stress and neurodegeneration interact, and that this occurs in the context of past traumatic experience, which may account for the possible re-emergence of symptoms in later life (Ruzich *et al.*, 2005).

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

Behavioral studies

Concerning the cognitive profile associated with PTSD, research in adult cohorts has revealed relatively consistent episodic memory impairment (e.g. Moore, 2009). Episodic, or declarative, memory refers to the effortful and conscious recollection of events and facts (Tulving, 1985). Research with aged Holocaust survivors has revealed both content independent and dependent episodic memory impairment (Golier *et al.*, 2002; 2003). In fact, 36% of Holocaust survivors performed at a level of frank cognitive impairment (Golier *et al.*, 2002). Further studies were conducted with both aged combat veterans (Yehuda *et al.*, 2005a) and Holocaust survivors (Yehuda *et al.*, 2004) using the California Verbal Learning Test (CVLT), which can parse apart the different aspects of episodic learning and memory (Delis *et al.*, 1987). Similar to younger veterans, but unlike Holocaust survivors with PTSD, these elderly combat veterans had impaired delayed free recall (Yehuda *et al.*, 2005a). However unlike the younger veterans and similar to the Holocaust survivors, these aged veterans showed reduced total learning, indicating problems with encoding and consolidation in both elderly samples (Yehuda *et al.*, 2004; 2005a). This suggests that a reduced rate of total learning may be attributable to an interaction between PTSD and aging, while the reduced delayed free recall is a particularity of combat veterans with PTSD. In addition, in all of these studies, age was negatively correlated with memory performance only in the PTSD groups, while no such correlation was uncovered in the exposed and healthy control groups (Golier *et al.*, 2002; 2003; Yehuda *et al.*, 2004; 2005a). In a longitudinal study of Holocaust survivors with PTSD, compared with controls, a decrease in symptom severity, and decrease in paired word association performance was found, while there was an improvement in performance on the California Verbal Learning Test (CVLT) that correlated with symptom improvements (Yehuda *et al.*, 2006). Paired learning is influenced by age but not symptom severity, suggesting an interaction between PTSD and age, while the CVLT is associated with severity and not age. This is consistent with the idea that a trauma-related biological change at an earlier time can be related to the trajectory of memory changes (Yehuda *et al.*, 2006). It seems that the effects of aging on explicit memory are greater in the PTSD group, which again is consistent with the idea of accelerated aging. Overall, the memory deficits are more substantial and generalized in the elderly with PTSD compared with younger individuals. Investigators have thus hypothesized that aging and PTSD interact and can lead to accelerated, age-related cognitive decline in some aspects.

Endocrinological studies

Endocrinological studies with aged PTSD samples have also suggested an interaction. In the general PTSD population, there seems to be a pattern of increased type II glucocorticoid receptor responsiveness, and lower or normal ambient cortisol levels (e.g. Yehuda, 2009). In elderly Holocaust survivors with PTSD, a flatter circadian rhythm of cortisol release was found, which is similar to that seen in healthy aging, and in contrast to younger samples (Yehuda *et al.*, 2005b). Yehuda *et al.* (2005b) interpret this pattern as reflecting a superimposition of PTSD and aging. In a subsequent study, Yehuda *et al.* (2005c) found a negative correlation between cortisol concentrations and episodic memory performance, even though the aged PTSD sample had lower ambient cortisol levels overall. This finding can seem paradoxical at face value given the cognitive impairment associated with PTSD, and especially in face of the glucocorticoid cascade hypothesis, which holds that stress is associated with elevated cortisol (Sapolsky *et al.*, 1986). However, cortisol is presumed to relate to cognitive ability in terms of an inverted-U function: either too low or too high levels can induce cognitive impairment (Lupien and McEwen, 1997; Erickson *et al.*, 2003). More recently, cortisol was administered to aging combat veterans and healthy controls (Yehuda *et al.*, 2007a). In both groups, episodic memory was enhanced, while working memory was enhanced only in the PTSD group, which is in contrast to younger samples where working memory was impaired (Grossman *et al.*, 2006; Yehuda *et al.*, 2007a). The older sample had worse working memory performance at baseline than did the younger sample in the previous study, which the authors interpret as evidence for accelerated age-related decline (Yehuda *et al.*, 2007a).

Neuroimaging studies

Given that the cognitive impairment that has been typically associated with PTSD is assumed to be dependent on the medial temporal lobe (MTL), combined with the centrality of memory to the disorder (i.e. intrusive memories, flashbacks), much neuroimaging research has been conducted on this region (e.g. Shin *et al.*, 2006). In the bulk of these studies, PTSD has been associated with smaller hippocampal volume compared with healthy controls. Still, it is difficult to attribute this to glucocorticoid toxicity since PTSD is not associated with elevated ambient cortisol levels

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

(Yehuda *et al.*, 2007b). However, the results are not unequivocal. In fact, a minority of studies have found cognitive impairment in the absence of hippocampal abnormality, or vice versa. In the handful of neuroimaging studies conducted with aged PTSD samples of Holocaust survivors, POWs and combat veterans, respectively, no difference was found in hippocampal volumes compared with controls (Golier *et al.*, 2005; Freeman *et al.*, 2006; Yehuda *et al.*, 2007b).

The discrepant findings concerning hippocampal volume between young and elderly PTSD samples and controls could be explained by several not necessarily mutually exclusive hypotheses. First, it could be that the atrophy that occurs with normal aging may mask the reduced hippocampal volume associated with PTSD in adults, such that in the elderly, the differences are no longer significant. Second, perhaps a lifetime of lower ambient cortisol levels manifested itself as cognitive impairment, but at the same time, protected the hippocampus from stress-related atrophy (Yehuda *et al.*, 2005c). Third, Freeman *et al.* (2006) hypothesized that since POWs have significantly higher morbidity than civilians, high attrition over time could have resulted in an exceptionally robust and healthy sample that lack reduced hippocampal volume (Freeman *et al.*, 2006). Fourth, it has been proposed that smaller hippocampal volume represents a predisposing factor rather than a consequence of PTSD itself. By interpreting the glucocorticoid and hippocampal characteristics as pre-trauma traits, researchers are able to explain why only a minor subset of individuals experience higher arousal and distress as a response to traumatic stress and go on to develop PTSD, and why PTSD is not always associated with smaller hippocampal volumes or cognitive impairment (Gilbertson *et al.*, 2002; Moore, 2009; Yehuda *et al.*, 2009). That no difference in hippocampal volume was found in the Holocaust survivors with PTSD could be because, in this unique sample, the main risk factors were being Jewish in a particular time and place (Yehuda *et al.*, 2009), which trumps every other predisposing risk factor. Furthermore, in the aged sample of combat veterans studied by Yehuda *et al.* (2007b), smaller left hippocampal volume was only found in those who developed PTSD after their first traumatic exposure, compared to those who went on to develop PTSD after subsequent traumatic events, suggesting that the latter individuals were more resilient. Finally, Yehuda *et al.* (2006) suggest that biological alterations present at one point during the lifespan can be related to particular trajectories of changes at a later time, and furthermore, that these alterations do not need to be present at that later time. They parallel the finding that increased heart rate immediately subsequent to traumatic exposure can be predictive of later PTSD, but that increased heart rate is no longer present when the PTSD is finally diagnosed (Yehuda *et al.*, 2006).

Discussion

Any clear relationship between PTSD and aging is challenging since aging is associated with significant heterogeneity. For instance, although aging is associated with cognitive decline, the elderly exhibit wide variation in cognitive function. Some individuals enjoy stable cognitive function with age while others experience severe decline. Lupien *et al.* (2005; 2007) have shown that there is much heterogeneity within the elderly population in terms of cortisol levels and diurnal patterns. They identified three subgroups of elderly people: two with increasing cortisol levels over time, with either currently high, or currently moderate levels, and a third group with decreasing cortisol levels over time with currently moderate levels. In the first group, hippocampal volume is 14% smaller than the other groups and is also associated with episodic memory impairment. Lupien *et al.* (2007) have pointed out that while the hippocampus is generally smaller and atrophies at a faster rate with age, there is significant overlap between the mean hippocampal volumes between old and young cohorts. Thus, because the cerebral and endocrinological findings associated both with healthy aging and with PTSD in the general population are highly heterogeneous and complex, making claims about the interaction between aging and PTSD is difficult. But it is clear from recent findings that PTSD in the elderly is associated with some unique characteristics, probably in part due to age.

In general, the epidemiological studies concerning PTSD in the elderly suggest a complex situation. The elderly seem to be at no worse risk than the rest of the population for developing PTSD after experiencing a traumatic event, although the evidence is not incontrovertible. Regarding PTSD that has its roots in a trauma experienced many decades previously, the research suggests that PTSD symptoms may persist significantly for some individuals. The course of PTSD over the lifespan can wax and wane, can remain stable, and can even seemingly disappear for decades only to reappear in old age. The fact of the matter is that the elderly represent a heterogeneous population that has experienced myriad types of traumas at various points during the lifespan. The trajectory of PTSD throughout life seems highly variable, and probably depends on a multitude of factors. Only additional,

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

well-designed longitudinal studies will reveal which factors are involved.

Overall, it seems that PTSD and the associated cognitive profile and biological features are not necessarily static over the lifespan. In certain respects, the trajectory of PTSD-related changes apparently interact with the normal aging process, perhaps leading to accelerated, age-related changes. Although it is difficult to elucidate clearly what these changes are, and how we may predict them, it is clear that the aging process in combination with PTSD presents a unique situation. The enhanced cognitive impairment in the elderly samples must be taken into consideration in the development and administration of treatment (Cook and O'Donnell, 2005). Again, more longitudinal studies focusing on the biological and cognitive changes over time are required in order to verify how PTSD interacts with age.

Conflict of interest

None.

Description of authors' roles

FF and LL conceived the topic and scope of paper. LL wrote the paper, and CA and FF read and commented on the final draft.

Acknowledgment

We would like to thank Dr Jonathan Lapp and Dr Serge Gauthier for their careful reading of the manuscript.

References

Aarts, P. G. H. and Op den Velde, W. (1996). Prior traumatization and the process of aging: theory and clinical implications. In B.A. van der Kolk, A.C. McFarlane and L. Weisaeth (eds.), *Traumatic Stress: The Effects of Overwhelming Experience on Mind, Body, and Society* (pp. 359–377). New York: Guilford.

Acierno, R., Brady, K., Gray, M., Kilpatrick, D., Resnick, D. and Best, C. (2002). Psychopathology following interpersonal violence: a comparison of risk factors in older and younger adults. *Journal of Clinical Geropsychology*, 8, 13–23.

Alpass, F., Long, N., Blakey, J. and Pachana, N. (2004). Post-traumatic stress disorder, social support and cognitive status in community-based older veterans. *Australasian Journal on Aging*, 23, 97–99.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC: American Psychiatric Association.

Averill, P. M. and Beck, J. G. (2000). Posttraumatic stress disorder in older adults: a conceptual review. *Journal of Anxiety Disorders*, 14, 133–156.

Bowlby, J. (1973). *Attachment and Loss. Vol. 2. Separation: Anxiety and Anger.* New York: Basic Books.

Bramsen, I., van der Ploeg, H. M. and Twisk, J. W. R. (2002). Secondary traumatization in Dutch couples of World War II survivors. *Journal of Consulting and Clinical Psychology*, 70, 241–245.

Bramsen, I., van der Ploeg, H. M. and Boers, M. (2006). Posttraumatic stress in aging World War II survivors after a fireworks disaster: a controlled prospective study. *Journal of Traumatic Stress*, 19, 291–300.

Brewin, C. R., Andrews, B. and Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, 68, 748–766.

Brodaty, H., Joffe, C., Luscombe, G. and Thompson, C. (2004). Vulnerability to post-traumatic stress disorder and psychological morbidity in aged holocaust survivors. *International Journal of Geriatric Psychiatry*, 19, 968–979.

Browne, C. J. and Shlosberg, E. (2006). Attachment theory, aging and dementia: a review of the literature. *Aging and Mental Health*, 10, 134–142.

Bryant, R. A. and Guthrie, R. M. (2007). Maladaptive self-appraisals before trauma exposure predict posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 75, 812–815.

Busuttil, W. (2004). Presentations and management of Post Traumatic Stress Disorder and the elderly: a need for investigation. *International Journal of Geriatric Psychiatry*, 19, 429–439.

Cassidy, J. and Mohr, J. J. (2001). Unsolvable fear, trauma, and psychopathology: theory, research, and clinical considerations related to disorganized attachment across the life span. *Clinical Psychology: Science and Practice*, 8, 275–298.

Chung, M. C., Werrett, J., Easthope, Y. and Farmer, S. (2004). Coping with post-traumatic stress: young, middle-aged and elderly comparisons. *International Journal of Geriatric Psychiatry*, 19, 333–343.

Chung, M. C., Berger, Z, Jones, R. and Rudd, H. (2006). Posttraumatic stress disorder and general health problems following myocardial infarction (post-MI PTSD) among older patients: the role of personality. *International Journal of Geriatric Psychiatry*, 21, 1163–1174.

Chung, M. C., Preveza, E., Papandreou, K. and Prevezas, N. (2006). The relationship between posttraumatic stress disorder following spinal cord injury and locus of control. *Journal of Affective Disorders*, 93, 229–232.

Chung, M. C., Berger, Z, Jones, R. and Rudd, H. (2008). Posttraumatic stress and co-morbidity following myocardial infarction among older patients: the role of coping. *Aging and Mental Health*, 12, 124–133.

Chung, M. C. *et al.* (2009). Posttraumatic Stress Disorder in older people after a fall. *International Journal of Geriatric Psychiatry*, 24, 955–964.

Cook, J. M. and O'Donnell, C. (2005). Assessment and psychological treatment of posttraumatic stress disorder in older adults. *Journal of Geriatric Psychiatry and Neurology*, 18, 61–71.

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

Cook, J. M., Ruzek, J. I. and Cassidy, E. (2003). Possible association of posttraumatic stress disorder with cognitive impairment among older adults. *Psychiatric Services*, 54, 1223–1225.

Creamer, M. and Parslow, R. (2008). Trauma exposure and posttraumatic stress disorder in the elderly: a community prevalence study. *American Journal of Geriatric Psychiatry*, 16, 853–856.

de Vries, G. J. and Olff, M. (2009). The lifetime prevalence of traumatic events and posttraumatic stress disorder in the Netherlands. *Journal of Traumatic Stress*, 22, 259–267.

Delis, D. C., Kramer, J. H., Kaplan, E. and Ober, B. A. (1987). *The California Verbal Learning Test*. San Antonio, TX: Psychological Corporation.

Diamond, L. M., and Fagundes, C. P. (2010). Psychobiological research on attachment. *Journal of Social and Personal Relationships*, 27, 218–225.

Dirkzwager, A. J. E., Bramsen, I. and van der Ploeg, H. M. (2001). The longitudinal course of posttraumatic stress disorder symptoms among aging military veterans. *Journal of Nervous and Mental Disease*, 189, 846–853.

Ehring, T., Ehlers, A. and Glucksman, E. (2008). Do cognitive models help in predicting the severity of posttraumatic stress disorder, phobia, and depression after motor vehicle accidents? A prospective longitudinal study. *Journal of Consulting and Clinical Psychology*, 76, 219–230.

Elklit, A. and O'Connor, M. (2005). Post-traumatic stress disorder in a Danish population of elderly bereaved. *Scandinavian Journal of Psychology*, 46, 439–445.

Erickson, K., Drevets, W. and Schulkin, J. (2003). Glucocorticoid regulation of diverse cognitive functions in normal and pathological emotional states. *Neuroscience and Biobehavioral Reviews*, 27, 233–246.

Erikson, E. H. (1966). Eight ages of man. *International Journal of Psychiatry*, 2, 281–307.

Eysenck, H. J. (1983). Stress, disease, and personality: the inoculation effect. In C. L. Cooper (ed.), *Stress Research* (pp. 121–146) New York: Wiley.

Field, E. L., Norman, P. and Barton, J. (2008). Cross-sectional and prospective associations between cognitive appraisals and posttraumatic stress disorder symptoms following stroke. *Behaviour Research and Therapy*, 46, 62–70.

Floyd, M., Rice, J. and Black, S. (2002). Recurrence of posttraumatic stress disorder in late life: a cognitive aging perspective. *Journal of Clinical Geropsychology*, 8, 303–11.

Frans, O., Rimmo, P. A., Aberg, L. and Fredrikson, M. (2005). Trauma exposure and post-traumatic stress disorder in the general population. *Acta Psychiatrica Scandinavica*, 111, 291–299.

Freeman, T. *et al.* (2006). Evidence of resilience: neuroimaging in former prisoners of war. *Psychiatry Research-Neuroimaging*, 146, 59–64.

Gilbertson, M. W. *et al.* (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience*, 5, 1242–1247.

Golier, J. A., Yehuda, R., Lupien, S. J., Harvey, P. D., Grossman, R. and Elkin, A. (2002). Memory performance in Holocaust survivors with posttraumatic stress disorder. *American Journal of Psychiatry*, 159, 1682–1688.

Golier, J. A., Yehuda, R., Lupien, S. J. and Harvey, P. D. (2003). Memory for trauma-related information in Holocaust survivors with PTSD. *Psychiatry Research*, 121, 133–143.

Golier, J. A., Yehuda, R., De Santi, S., Segal, S., Dolan, S. and de Leon, M. J. (2005). Absence of hippocampal volume differences in survivors of the Nazi Holocaust with and without posttraumatic stress disorder. *Psychiatry Research – Neuroimaging*, 139, 53–64.

Grossman, A. B., Levin, B. E., Katzen, H. L. and Lechner, S. (2004). PTSD symptoms and onset of neurologic disease in elderly trauma survivors. *Journal of Clinical and Experimental Neuropsychology*, 26, 698–705.

Grossman, R., Yehuda, R., Golier, J. A., McEwen, B. S., Harvey, P. D. and Maria, N. S. (2006). Cognitive effects of intravenous hydrocortisone in subjects with PTSD and healthy control subjects. *Annals of the New York Academy of Sciences*, 1071, 410–421.

Hantman, S. and Solomon, Z. (2007). Recurrent trauma: Holocaust survivors cope with aging and cancer. *Social Psychiatry and Psychiatric Epidemiology*, 42, 396–402.

Hart, J. *et al.* (2008). Cognitive dysfunctions associated with PTSD: evidence from World War II prisoners of war. *Journal of Neuropsychiatry and Clinical Neurosciences*, 20, 309–316.

Hiskey, S., Luckie, M., Davies, S. and Brewin, C. R. (2008). The emergence of posttraumatic distress in later life: a review. *Journal of Geriatric Psychiatry and Neurology*, 21, 232–241.

Horowitz, M. (1974). Stress response syndromes: character style and dynamic psychotherapy. *Archives of General Psychiatry*, 31, 768–781.

Joffe, C., Brodaty, H., Luscombe, G. and Ehrlich, F. (2003). The Sydney Holocaust Study: posttraumatic stress disorder and other psychosocial morbidity in an aged community sample. *Journal of Traumatic Stress*, 16, 39–47.

Johnston, D. (2000). A series of cases of dementia presenting with PTSD symptoms in World War II combat veterans. *Journal of the American Geriatrics Society*, 48, 70–72.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M. and Nelson, C. B. (1995). Posttraumatic-stress disorder in the national comorbidity survey. *Archives of General Psychiatry*, 52, 1048–1060.

Kluznick, J. C., Speed, N., van Valkenburg, C. and Magraw, R. (1986). Forty-year follow-up of United States prisoners of war. *American Journal of Psychiatry*, 143, 1443–1446.

Kohn, R., Levav, I., Garcia, I.D., Machuca, M. E. and Tamashiro, R. (2005). Prevalence, risk factors and aging vulnerability for psychopathology following a natural disaster in a developing country. *International Journal of Geriatric Psychiatry*, 20, 835–841.

Kubzansky, L. D., Koenen, K. C., Spiro, A., Vokonas, P. S. and Sparrow, S. (2007). Prospective study of posttraumatic stress disorder symptoms and coronary heart disease in the normative aging study. *Archives of General Psychiatry*, 64, 109–116.

Lee, T. M. C., Chi, I., Chung, L. W. M. and Chou, K. L. (2006). Aging and psychological response during the post-SARS period. *Aging and Mental Health*, 10, 303–311.

Liu, A. Z. *et al.* (2006). An epidemiologic study of posttraumatic stress disorder in flood victims in Hunan

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

China. *Canadian Journal of Psychiatry – Revue Canadienne de Psychiatrie*, 51, 350–354.

Lupien, S. J. and McEwen, B. S. (1997). The acute effects of corticosteroids on cognition: integration of animal and human model studies. *Brain Research Review*, 24, 1–27.

Lupien, S. J. *et al.* (2005). Stress hormones and human memory function across the lifespan. *Psychoneuroendocrinology*, 30, 225–242.

Lupien, S. J. *et al.* (2007). Hippocampal volume is as variable in young as in older adults: implications for the notion of hippocampal atrophy in humans. *Neuroimage*, 34, 479–485.

Macleod, A. D. (1994). The reactivation of posttraumatic-stress disorder in later life. *Australian and New Zealand Journal of Psychiatry*, 28, 625–634.

McNally, R. J. (2003). Progress and controversy in the study of posttraumatic stress disorder. *Annual Review of Psychology*, 54, 229–252.

Mittal, D., Torres, R., Abashidze, A. and Jimerson, N. (2001). Worsening of post-traumatic stress disorder symptoms with cognitive decline: case series. *Journal of Geriatric Psychiatry and Neurology*, 14, 17–20.

Moore, S. A. (2009). Cognitive abnormalities in posttraumatic stress disorder. *Current Opinion in Psychiatry*, 22, 19–24.

Norris, F. H., Murphy, A. D., Baker, C. K., Perilla, J. L., Rodriguez, F. G. and Rodriguez, J. D. (2003). Epidemiology of trauma and posttraumatic stress disorder in Mexico. *Journal of Abnormal Psychology*, 112, 646–656. Op den

Velde, W. *et al.* (1993). Posttraumatic stress disorder in Dutch resistance veterans from World War II. In J. P. Wilson and B. Raphael (eds.), *International Handbook of Traumatic Stress Syndromes* (pp. 219–230). New York: Plenum.

Owens, G. P., Baker, D. J., Kasckow, J., Ciesla, J. A. and Mohamed, S. (2005). Review of assessment and treatment of PTSD among elderly American armed forces veterans. *International Journal of Geriatric Psychiatry*, 20, 1118–1130.

Ozer, E. J., Best, S., R., Lipsey, T. L. and Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychological Bulletin*, 129, 52–73.

Pitman, R. K. (2010). Posttraumatic stress disorder and dementia. What is the origin of the association? *JAMA*, 303, 2287–2288.

Port, C. L., Engdahl, B. and Frazier, P. (2001). A longitudinal and retrospective study of PTSD among older prisoners of war. *American Journal of Psychiatry*, 158, 1474–1479.

Port, C. L., Engdahl, B., Frazier, P. and Eberly, R. E. (2002). Factors related to the long-term course of PTSD in older ex-prisoners of war. *Journal of Clinical Geropsychology*, 8, 203–214.

Ruzich, M. J., Looi, J. C. L. and Robertson, M. D. (2005). Delayed onset of posttraumatic stress disorder among male combat veterans: a case series. *American Journal of Geriatric Psychiatry*, 13, 424–427.

Sapolsky, R. M., Krey, L. C. and McEwen, B. S. (1986). The neuroendocrinology of stress and aging: the glucocorticoid cascade hypothesis. *Endocrine Reviews*, 7, 284–301.

Shin, L. M., Rauch, S. L. and Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Science*, 1071, 67–79.

Solomon, Z. and Ginzburg, K. (1998). War trauma and the aged: an Israeli perspective. *Handbook of Aging and Mental Health*, 135–152.

Solomon, Z. and Mikulincer, M. (2006). Trajectories of PTSD: a 20-year longitudinal study. *American Journal of Psychiatry*, 163, 659–666.

Solomon, Z., Helvitz, H. and Zerach, G. (2009). Subjective age, PTSD and physical health among war veterans. *Aging and Mental Health*, 13, 405–413.

Spitzer, C., Barnow, S., Volzke, H., John, U., Freyberger, H. J. and Grabe, H. J. (2008). Trauma and posttraumatic stress disorder in the elderly: findings from a German community study. *Journal of Clinical Psychiatry*, 69, 693–700.

Tennant, C., Fairley, M. J., Dent, O. F., Sulway, M. and Broe, G. A. (1997). Declining prevalence of psychiatric disorder in older former prisoners of war. *Journal of Nervous and Mental Disease*, 185, 686–89.

Trappler, B., Cohen, C. I. and Tulloo, R. (2007). Impact of early lifetime trauma in later life: depression among Holocaust survivors 60 years after the liberation of Auschwitz. *American Journal of Geriatric Psychiatry*, 15, 79–83.

Tulving, E. (1985). How many memory systems are there? *American Psychologist*, 40, 385–398.

van Achterberg, M. E., Rohrbaugh, R. M. and Southwick, S. M. (2001). Emergence of PTSD in trauma survivors with dementia. *Journal of Clinical Psychiatry*, 62, 206–207.

van Ameringen, M., Mancini, C., Patterson, B. and Boyle, M. H. (2008). Post-traumatic stress disorder in Canada. *CNS Neuroscience and Therapeutics*, 14, 171–181.

van Zelst, W. H., de Beurs, E., Beekman, A. T. F., Deeg, D. J. H. and van Dyck, R. (2003). Prevalence and risk factors of posttraumatic stress disorder in older adults. *Psychotherapy and Psychosomatics*, 72, 333–342.

van Zelst, W. H., de Beurs, E., Beekman, A. T. F., van Dyck, R. and Deeg, D. D. H. (2006). Well-being, physical functioning, and use of health services in the elderly with PTSD and subthreshold PTSD. *International Journal of Geriatric Psychiatry*, 21, 180–188.

Yaffe, K. *et al.* (2010). Posttraumatic stress disorder and risk of dementia among US veterans. *Archives of General Psychiatry*, 67, 608–613.

Yehuda, R. (2009). Status of glucocorticoid alterations in post-traumatic stress disorder. *Annals of the New York Academy of Sciences*, 1179, 56–69.

Yehuda, R., Schmeidler, J., Siever, L. J., Binder-Brynes, K. and Elkin, A. (1997). Individual differences in posttraumatic stress disorder symptom profiles in Holocaust survivors in concentration camps or in hiding. *Journal of Traumatic Stress*, 10, 453–463.

Yehuda, R., Halligan, S. L., Grossman, R., Golier, J. A. and Wong, C. (2002). The cortisol and glucocorticoid receptor response to low dose dexamethasone administration in aging combat veterans and Holocaust survivors with and without posttraumatic stress disorder. *Biological Psychiatry*, 52, 393–403.

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press

Yehuda, R., Golier, J. A., Halligan, S. L. and Harvey, P. D. (2004). Learning and memory in Holocaust survivors with posttraumatic stress disorder. *Biological Psychiatry*, 55, 291–295.

Yehuda, R., Golier, J. A., Tischler, L., Stavitsky, K. and Harvey, P. D. (2005a). Learning and memory in aging combat veterans with PTSD. *Journal of Clinical and Experimental Neuropsychology*, 27, 504–515.

Yehuda, R., Golier, J. A. and Kaufman, S. (2005b). Circadian rhythm of salivary cortisol in holocaust survivors with and without PTSD. *American Journal of Psychiatry*, 162, 998–1000.

Yehuda, R. *et al.* (2005c). Relationship between cortisol and age-retated memory impairments in Holocaust survivors with PTSD. *Psychoneuroendocrinology*, 30, 678–687.

Yehuda, R. *et al.* (2006). Longitudinal assessment of cognitive performance in Holocaust survivors with and without PTSD. *Biological Psychiatry*, 60, 714–721.

Yehuda, R., Harvey, P. D., Buchsbaum, M., Tischler, L. and Schmeidler, J. (2007a). Enhanced effects of cortisol administration on episodic and working memory in aging veterans with PTSD. *Neuropsychopharmacology*, 32, 2581–2591.

Yehuda, R. *et al.* (2007b). Hippocampal volume in aging combat veterans with and without post-traumatic stress disorder: relation to risk and resilience factors. *Journal of Psychiatric Research*, 41, 435–445.

Yehuda, R. *et al.* (2009). Ten-year follow-up study of PTSD diagnosis, symptom severity and psychosocial indices in aging holocaust survivors. *Acta Psychiatrica Scandinavia*, 119, 25–34.

Zlotnick, C., Johnson, J., Kohn, R., Vicente, B., Rioseco, P. and Saldivia, S. (2006). Epidemiology of trauma, post-traumatic stress disorder (PTSD) and co-morbid disorders in Chile. *Psychological Medicine*, 36, 1523–1533.

https://doi.org/10.1017/S1041610211000366 Published online by Cambridge University Press