



Investigating the separate and interactive associations of trauma and depression on neurocognition in urban dwelling adults



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ARTICLE INFO

Article history:

Received 4 October 2016

Received in revised form

6 January 2017

Accepted 12 January 2017

Keywords:

Trauma

Depression

Neurocognition

Aging

Learning

Memory

ABSTRACT

Background: Trauma and depression have each been associated with neurocognitive alterations, but their combined effect on neurocognition is unclear. We investigated the separate and interactive associations of trauma and depression on neurocognition in a sample of ethnically diverse urban dwellers, and explored the impact of age on these effects.

Methods: 284 adults aged 30–89 were divided into groups based on their current depression and trauma history. Individuals meeting DSM-IV criteria for depression were considered Depressed (D+) and individuals rated through diagnostic interview as having trauma history were considered positive for Trauma (T+). Resulting Ns were 73 D+T+, 56 D+T-, 68 D-T+, and 87 D-T-. A principal component analysis of neuropsychological scores resulted in a 3-factor solution representing verbal learning/memory/recognition (VERBAL-LMR), visual learning/memory/recognition, and speeded attention/cognitive flexibility accounting for 70.21% of the variance.

Results: Multivariable linear regressions adjusting for age revealed that Trauma, regardless of Depression, is associated with worse VERBAL-LMR performance. This Trauma association was driven by verbal list and prose passages learning and memory, but not recognition memory. Age-stratified (<60 versus ≥60 years) regressions revealed the Trauma association was only significant for older adults. No main or interactive effects for Depression were observed.

Conclusions: Trauma, regardless of Depression, is associated with worse verbal learning and memory, but not recognition performance. These results suggest that trauma exposure may negatively impact neurocognition. Clinicians working with adults in urban settings should query for trauma in addition to depression when considering subjective and objective measures of neurocognitive functioning, particularly in older adults.

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1. Introduction

An estimated 89% of urban dwellers have experienced traumatic events (Breslau et al., 2004). Experiencing traumatic events such as exposure to actual or threatened death, serious injury or sexual violation puts one at risk for psychological disturbances including depressive disorders, regardless of genetic/familial factors that may contribute to mood disorders (Brown et al., 2014). Later life

depression is a known risk factor for neurocognitive decline (Dotson et al., 2008) and dementia (Diniz et al., 2013). Though trauma is independently related to both depression and alterations in neurocognitive functioning (Hedges and Woon, 2010), the role of trauma in the depression-neurocognition association is rarely explored.

While neurocognitive alterations seen in depression include episodic memory/recognition, visuospatial and/or psychomotor deficits, reduced executive functioning and information processing are the most commonly occurring impairments (Elderkin-Thompson et al., 2011; Morimoto and Alexopoulos, 2013; Rock et al., 2013; Snyder, 2013); often persisting into remitted states

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(Hasselbalch et al., 2011). Depressed older adults are particularly vulnerable to alterations of memory, executive functioning and information processing given these deficits are more common (Sexton et al., 2012) and more pronounced (Dotson et al., 2008) in older compared to younger adults and are present in minor and major depression (Elderkin-Thompson et al., 2003). Furthermore, meta-analytic longitudinal findings suggest that the neurocognitive deficits seen in late life depression predict the development of dementia (Potter et al., 2013). This may be due, in part, to the fact that many of the neurocognitive deficits associated with depression, e.g., memory and executive dysfunction, are also early signs of dementia in older adults (Butters et al., 2008).

In some, but not all (Flaks et al., 2014; Leskin and White, 2007) studies, trauma history is also independently associated with neurocognitive dysfunction (Hedges and Woon, 2010), particularly in older adults (Petkus et al., 2012; Ritchie et al., 2012; Yehuda et al., 2005). For example, early-life traumas are associated with decline in verbal fluency, verbal learning, visual and verbal memory, and executive functioning in older adults (Ritchie et al., 2012). While the literature addressing the relationship between trauma and neurocognitive performance in adults regardless of age is almost exclusive to early-life trauma, studies of specific trauma (e.g. interpersonal violence in adulthood) provide insight into the impact of trauma experienced across the lifespan. For example, older adults with a history of multiple sexual assaults in adulthood, experienced executive dysfunction in later life (Petkus et al., 2012). In addition, individuals with trauma history experienced in adulthood exhibit worse performance on tasks of visuoconstruction and visual memory, sustained attention, and executive functioning as adults (Stein et al., 2002), as well as verbal learning as older adults (Yehuda et al., 2005), than non-exposed control groups. Together, these findings suggest that trauma exposure across the lifespan is associated with neurocognitive alterations in older adults.

There is literature to suggest that depression and trauma history in combination are associated with poorer affective outcomes (Bernet and Stein, 1999), with recent literature extending these findings to include neurocognition. This may be due, in part to the fact that there are similarities in the long-term neurocognitive profiles associated with trauma history and those associated with depression on tasks of information processing and executive functioning (Petkus et al., 2012; Yehuda et al., 2004, 2005; Stein et al., 2002; Navalta et al., 2006; Majer et al., 2010; Gould et al., 2012). In fact, a recent study in adults with depression showed that processing speed worsens with increasing early-life trauma scores (Saleh et al., 2016). Additionally, compared to healthy controls, adults with depression and trauma history exhibited worse executive functioning, delayed recall, and recognition memory (Parlar et al., 2016). In considering these similarities, however, one must also consider that the literature investigating how depression and trauma individually contribute to neurocognition do not control for the presence of the other, i.e., depression studies do not control for trauma or vice versa. In fact, many early-life trauma studies fail to control for, or measure past and/or present mood disorders (Yehuda et al., 2004, 2005; Stein et al., 2002; Navalta et al., 2006; Gould et al., 2012). This is particularly important given the high rate of comorbidity between trauma and depression (Afzali et al., 2016). While a recent PTSD study attempted to address this statistically, reporting depressive symptoms mediated the relationship between PTSD and executive functioning (Olf et al., 2014), incorporating a trauma-exposed group free of formal psychiatric diagnoses including PTSD, and a depressed group free of trauma history would help to further elucidate how depression and trauma independently contribute to neurocognition in aging.

The current study examined the separate and interactive associations of trauma and depression on neurocognitive functioning in

a diverse urban sample. We focused on an urban population given the high trauma exposure in metropolitan cities (Breslau et al., 2004). We hypothesized that depression alone would be negatively associated with information processing speed and executive functioning given that these domains are the most commonly and profoundly influenced in depression, regardless of remitted states (Rock et al., 2013; Snyder, 2013). We hypothesized that trauma alone would be negatively associated with information processing speed and executive functioning, as well as learning and memory. We further hypothesized that together, trauma and depression would exacerbate deficits in information processing speed, executive functioning, learning, and memory when compared to either one alone. Given that the aforementioned neurocognitive alterations associated with depression (Dotson et al., 2008) and trauma independently (Stein et al., 2002; Navalta et al., 2006; Majer et al., 2010) are worse in older adults (Yehuda et al., 2004, 2005; Petkus et al., 2012; Ritchie et al., 2012), we also explored the role of age on the associations between trauma/depression and neurocognition.

2. Methods

This study leveraged cross-sectional data from investigations of depression and diabetes conducted in a diverse urban population at the Department of Psychiatry, University of Illinois at Chicago (UIC). Participants include 319 adults >30 years old recruited through community outreach, fliers, and research registries. The study was approved by the UIC Institutional Review Board and conducted in accordance with the Declaration of Helsinki.

All participants underwent a preliminary telephone screen. Exclusion criteria consisted of current or past history of an Axis I disorder other than major depression or anxiety (i.e. manic episodes, psychotic disorders, PTSD), current substance abuse/dependence, neurological disorders (e.g. stroke, dementia, seizure, etc.), prior head injury with or without loss of consciousness, and/or current psychotropic medication use including anti-depressants. Thus, all study participants, including those diagnosed with major depression were free of anti-depressant medication for at least two weeks in order to study depressed mood during an untreated state. Participants were not excluded for chronic medical conditions (e.g. hypertension) or a past history of substance abuse >5 years prior to study entry; a past history of substance dependence was an exclusion criterion.

After passing the telephone screen, participants were scheduled for a more detailed evaluation that included neurocognitive, the Mini Mental State Examination (MMSE; Folstein et al., 1975), and affective, the Structured Clinical Interview for the DSM-IV-TR Disorders (SCID; Spitzer et al., 1992), screens for final inclusion/exclusion determination. Screening measures were administered by a trained research assistant followed by an evaluation by a board certified (AK) or board eligible (OA) psychiatrist who completed the 17-item Hamilton Depression Rating Scale (HDRS; Hamilton, 1960). All raters were blind to telephone screen information.

Final inclusion criteria for adults with Depression included a current diagnosis of major depressive disorder based on the SCID and HDRS scores >15. The SCID also assessed for trauma history (Spitzer et al., 1992) as it is a valid assessment of trauma that exhibits high positive predictive power compared to the Stressful Life Event Questionnaire (Elhai et al., 2008). Individuals with Trauma had to meet both traumatic event Criterion A:1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to physical integrity of self or others, and 2) the person's response involved intense fear, helplessness, or horror. Inclusion criteria for non-depressed participants included an absence of current and

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