



The impact of self-efficacy, alexithymia and multiple traumas on posttraumatic stress disorder and psychiatric co-morbidity following epileptic seizures: A moderated mediation analysis

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ABSTRACT

This study investigated the incidence of posttraumatic stress disorder (PTSD) and psychiatric co-morbidity following epileptic seizure, whether alexithymia mediated the relationship between self-efficacy and psychiatric outcomes, and whether the mediational effect was moderated by the severity of PTSD from other traumas. Seventy-one ($M=31$, $F=40$) people with a diagnosis of epilepsy recruited from support groups in the United Kingdom completed the Posttraumatic Stress Diagnostic Scale, the Hospital Anxiety and Depression Scale, the Toronto Alexithymia Scale-20 and the Generalized Self-Efficacy Scale. They were compared with 71 people ($M=29$, $F=42$) without epilepsy. For people with epilepsy, 51% and 22% met the diagnostic criteria for post-epileptic seizure PTSD and for PTSD following one other traumatic life event respectively. For the control group, 24% met the diagnostic criteria for PTSD following other traumatic life events. The epilepsy group reported significantly more anxiety and depression than the control. Partial least squares (PLS) analysis showed that self-efficacy was significantly correlated with alexithymia, post-epileptic seizure PTSD and psychiatric co-morbidity. Alexithymia was also significantly correlated with post-epileptic seizure PTSD and psychiatric co-morbidity. Mediation analyses confirmed that alexithymia mediated the path between self-efficacy and post-epileptic seizure PTSD and psychiatric co-morbidity. Moderated mediation also confirmed that self-efficacy and PTSD from one other trauma moderated the effect of alexithymia on outcomes. To conclude, people can develop posttraumatic stress disorder symptoms and psychiatric co-morbidity following epileptic seizure. These psychiatric outcomes are closely linked with their belief in personal competence to deal with stressful situations and regulate their own functioning, to process rather than defend against distressing emotions, and with the degree of PTSD from other traumas.

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1. Introduction

Following epileptic seizure, people can develop psychiatric co-morbid symptoms such as depression and anxiety (e.g. Piazzini et al., 2001; Manchanda, 2002; Gilliam et al., 2003; Gaitatzis et al., 2004; Bevenburg et al., 2005; Cramer et al., 2005; Jones et al., 2005; de Souza and Salgado, 2006; Kimiskidis et al., 2007; López-Gómez et al., 2008; Sperling et al., 2008; Endermann and Zimmermann, 2009; Kanner, 2009; Thapar et al., 2009). These symptoms are more severe than in people who are free from seizures or epilepsy (e.g. Mensah et al., 2006; Hessen et al., 2008; Seminario et al., 2009; Tellez-Zenteno et al., 2007).

Little is known about the possible emergence of posttraumatic stress disorder (PTSD) following epileptic seizures (post-epileptic

seizure PTSD), despite the fact that epileptic seizure is an experience involving threat to one's physical integrity, fear, helplessness or horror (APA, DSM-IV, 1994). Research into this psychological syndrome and associated factors has been very limited. This study aimed to help to fill these gaps in knowledge. It is worth noting that post-epileptic seizure PTSD is different from "posttraumatic epilepsy" which is a recurrent seizure disorder following brain injury (e.g. Temkin, 2009; Raymont et al., 2010).

In selecting the psychological factors for our investigation, we are drawn to a recent review suggesting that individuals with a high level of self-efficacy can regulate their own functioning, buffer against the adverse effects of trauma and thus foster recovery. These individuals have the ability to exercise control over traumatic adversity and to mend their lives rather than feeling overwhelmed by adverse circumstances which control their lives. Hence such challenges foster increased resilience to future adversity. Self-efficacy is an enabling and protective process which helps people to control what they think, and to regulate

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their emotions, feelings or affects. They can consequently confront the effects of trauma with confidence, leading to emotional well-being and the alleviation of posttraumatic stress and anxiety.

A low level of self-efficacy and inability to control one's thoughts, on the other hand, leads to the exacerbation of distress and to on-going traumatic thoughts intruding into consciousness. When threats are felt to be unmanageable, their severity is magnified. This leads to maladaptive coping, difficulty in regulating emotions, increased anxiety and distress and impaired functioning. It is not the frequency of aversive cognitions per se which generates the anxiety but rather the sense of powerlessness in controlling these cognitions (Bandura, 1997; Benight and Bandura, 2004).

People with epilepsy tend to have a low level of self-efficacy which has been associated with depression (Dilorio et al., 2004; Robinson et al., 2008; Ridsdale, 2009; Lee et al., 2010) and anxiety (Dilorio et al., 1996). This can affect their ability to manage their medication (Dilorio et al., 1992, 2004; Kobau and Dilorio, 2003; Chen et al., 2010) and to adopt an appropriate lifestyle for handling their chronic illness (Dilorio et al., 1994; Robinson et al., 2008; Begley et al., 2010) and reducing the occurrence of seizures (Dilorio et al., 1992; Wagner et al., 2010). We therefore hypothesize that for people with epilepsy, low self-efficacy would be associated with high levels of PTSD and psychiatric co-morbidity.

Whilst this implies a direct path between self-efficacy and psychiatric outcomes, there is another construct that needs to be considered. As was mentioned earlier, low self-efficacy leads to a lack of ability to regulate emotional processes. One way to conceptualize this difficulty is in terms of alexithymia. Low self-efficacy, we therefore hypothesized, would be related to high alexithymia. Alexithymia is a deficit in the cognitive processing and regulation of emotional states characterized by difficulty in identifying feelings, distinguishing between feelings and bodily sensations, and in describing feelings. It also involves restricted imaginal processing with a poverty of symbolization in fantasy and imagination and a tendency towards focusing on external events rather than internal experiences (Taylor, 1994). People with epilepsy have been shown to have a higher degree of alexithymia than those without (Tojek et al., 2000). In particular, they have difficulty in the domain of identifying feelings (Bewley et al., 2005). This in turn influences psychological co-morbid symptoms (Kalinin et al., 2010).

Alexithymia has been shown to relate to PTSD and psychiatric co-morbid symptoms. When confronting distress from a traumatic event, in this case, epileptic seizure, alexithymia acts as a defence mechanism (Wise et al., 1991; Helmes et al., 2008) against the distressing emotions associated with the event. Having difficulty processing emotions alongside dissociation between subjective feelings and physiological sensations (Taylor, 1984; Alonzo and Reynolds, 1998) minimizes emotional involvement and protect the self (McDougall, 1985; Thome, 1990; Ihilevich and Gleser, 1993). Hence, alexithymia has been conceptualized as the emotionally numbing aspect of PTSD following a traumatic event (Badura, 2003).

Given that alexithymia acts as a defence mechanism against distressing emotions associated with a specific traumatic event such as epileptic seizure, one can assume that it would also act as a defence against the distress from other traumas. In other words, for people with epilepsy, multiple traumas could be related to alexithymia. The self-trauma theory (Briere, 1996) describes how multiple traumas alter self-capacities characterized partly by difficulty in regulating affect or emotion. Multiple traumas can affect self-regulatory ability and increase symptom complexity in adults (Cloitre et al., 2009).

In sum, for people with epilepsy, low self-efficacy would be associated with high levels of post-epileptic seizure PTSD and

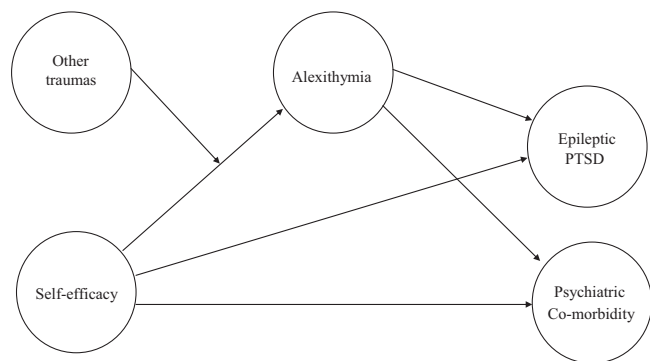


Fig. 1. The path model describing the interrelationship between self-efficacy, alexithymia, traumatic events other than epileptic seizure, post-epileptic seizure PTSD and psychiatric co-morbidity.

psychiatric co-morbidity. Low self-efficacy would also be associated with a high level of alexithymia which, in turn, would lead to a high level of post-epileptic seizure PTSD and co-morbid psychiatric symptoms. Alexithymia, in other words, would be a mediator which is, at the same time, moderated by the degree of PTSD following trauma other than epileptic seizure. This sums up the novelty of this study. It aimed to address a gap in trauma literature, namely, PTSD following epileptic seizure. It also intended to explore how the interface of self-efficacy and alexithymia alongside multiple traumas could impact on post-epileptic seizure PTSD and psychiatric co-morbidity. This is another gap in literature. The findings from this study could potentially open up a new way of understanding the traumatic reactions of individuals who suffer from epilepsy.

Thus, the present study aimed to investigate (1) the incidence of post-epileptic seizure PTSD and psychiatric co-morbidity and (2) the interrelationship between these outcomes and self-efficacy, alexithymia and multiple traumas. We hypothesized that

- 1) a proportion of people with epileptic seizure experience would meet the diagnostic criteria for PTSD and report a higher level of psychiatric co-morbidity than those without epilepsy.
- 2) Self-efficacy would be negatively associated with PTSD and psychiatric co-morbidity and negatively correlated with alexithymia which in turn would be positively correlated with post-epileptic seizure PTSD and psychiatric co-morbidity. We also hypothesized a mediational and moderated mediational effect in that
- 3) alexithymia would mediate the path between self-efficacy and outcomes and this mediational effect would be moderated by the severity of PTSD from other traumas.

This hypothesized model is shown in Fig. 1.

2. Method

2.1. Participants

The participants comprised 71 people ($M=31$, $F=40$) who had previously been given a medical diagnosis of some types of epilepsy. On average, they were 39 (mean=39.12, S.D.=12.86) years old ranging from 18 to 86 years with almost half ($n=35$, 49%) married and about a third ($n=23$, 32%) single. All were Caucasian. Most ($n=59$, 83%) were in the low income category which was defined according to occupation, and most ($n=69$, 97%) had not received higher or university education. On average, the onset of epilepsy was 21 (mean=21.39, S.D.=13.89) years prior to the study. Tonic Clonic epilepsy was the most common ($n=60$, 85%) whilst the rest of the sample had complex partial epilepsy. The average estimated number of seizures experienced in the year prior to the study was 63 (mean=63.14, S.D.=64.42). All participants were taking medication to control seizures at the time

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