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EC in SAD and MDD

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Abstract

Event centrality is defined by the extent to which a memory of an event has become central to an individual's identity and life story. Previous research predominantly focused on the link between event centrality and trauma-related symptomatology. Nevertheless, it can be argued that the perception of (adverse) events as central to one's self is not exclusive to Posttraumatic Stress Disorder (PTSD). Other disorders where adverse events are linked to the onset of symptoms might also be related to event centrality. This study examined the relevance of event centrality for Social Anxiety Disorder (SAD) and for Major Depressive Disorder (MDD) separately. Moreover, we examined which cognitive and emotion regulation variables (i.e., trait anxiety, rumination, worry, intrusions and avoidance, and posttraumatic cognitions) mediated these relationships. No significant correlation was found between event centrality and social anxiety. However, a significant positive correlation was found between event centrality and depression. In a combined group, this relation was mediated by all cognitive and emotion regulation variables except for worry.

Keywords: Autobiographical memory; event centrality; MDD; depression; SAD; social anxiety; maladaptive cognitions; emotion regulation

Introduction

Event centrality is defined by the extent to which a memory of an event has become central to the individual's identity and life story. Berntsen and Rubin (2006) proposed that the memory of a traumatic event can form a cognitive reference point in autobiographical knowledge and thereby influence the interpretation of past experiences and expectations for future events. Conceptually, event centrality consists of three aspects: 1) the event is seen as a turning point in life; 2) the event is seen as a reference point for understanding other everyday experiences; 3) the event has become part of the life-story and identity. The majority of research on event centrality has centered on its association with trauma-related symptomatology, revealing strong positive correlations with intrusions, avoidance, posttraumatic cognitions, and rumination (Berntsen & Rubin, 2006, 2007; Boals, 2010; Brown et al., 2010; Gehrt et al., 2018; Vermeulen et al., 2019). Moreover, the relation between event centrality and Posttraumatic Stress Disorder (PTSD) symptoms may be mediated by these variables (Boelen, 2012b; Lancaster et al., 2011; Vermeulen et al., 2019).

Onset-event-related disorders and Event Centrality

From a broader theoretical perspective, it is proposed that the perception of (adverse) events as central to one's identity is not confined solely to PTSD. Other psychological disorders, in which adverse events have been associated to the onset of symptoms might also be related to event centrality. For example, experiences of negative life events (in combination with feelings of shame; Magee, 1999), recurrent experiences of negative social situations (e.g., bullying, rejection, humiliation, or exclusion by significant others), and the experience of childhood trauma (De Venter et al., 2017) have a significant effect on the development and maintenance of Social Anxiety Disorder (SAD; Rapee & Spence, 2004). Moreover, there is a substantial causal relationship between stressful life events and the onset of Major Depressive Disorder (MDD; Kendler et al., 1999; Kessler, 1997; McCutcheon et al.,

2009). More specifically, a large body of research has shown a strong relationship between childhood abuse and MDD (Maniglio, 2010; Wiersma et al., 2009). Taken together, while the diagnosis of PTSD presupposes a traumatic experience, other psychological disorders like SAD and MDD do not necessarily require a specific traumatic event for classification based on DSM criteria. It is crucial, however, to differentiate between the classification criteria outlined in the DSM and the etiology of a disorder. In the case of PTSD, the diagnosis is explicitly linked to the experience of trauma, whereas for disorders such as SAD and MDD, the origins of symptoms may be more varied, involving a person's history and the evaluation of key learning moments including adverse events (for an overview, see Beck, 2008; Rapee & Spence, 2004). This broader perspective suggests that adverse events can still exert a significant impact on symptom development across various psychological disorders.

Therefore, it seems plausible that event centrality of adverse events would also be related to the onset of SAD and MDD. When the memories of these adverse events become central to the individual, such memories could become a turning point in life, a reference point for understanding neutral, everyday experiences, and part of the life story and identity. This, in turn, could have a continuous impact on how individuals see themself and the world.

SAD and event centrality

Cognitive models of SAD (Clark & Wells, 1995; Hofmann, 2007; Rapee & Heimberg, 1997; Rapee & Spence, 2004) converge on the idea that there is a bi-directional relationship between maladaptive cognitions and negative social experiences: The individual underestimates its social abilities and has a biased negative self-representation (Hofmann, 2007). This can lead to a feeling of underachieving in social situations, which, on the one hand, is potentially reinforced by an attentional bias towards negative feedback (Clark & Wells, 1995). On the other hand, it could also lead to an interpretation bias, where neutral, non-threatening social situations are interpreted as threatening. This, in turn, could evoke

behavioral changes, e.g., avoidance of certain social situations and post-event rumination (Hofmann, 2007). Experiencing recurrent negative social events like bullying, rejection, humiliation, or exclusion will reinforce or strengthen these maladaptive cognitions (Rapee & Spence, 2004). Also, the engagement in avoidance and safety behaviors, in combination with rumination, can be expected to lead to a vicious circle, ultimately maintaining and further exacerbating social anxiety symptoms (Hofmann, 2007).

The negative social events might be seen as a reference point for other experiences, where the memory is appraised as an anchoring event. The memory of the negative social event colors neutral memories in hindsight and can continuously impact the expectations for future situations. Also, the negative social event may become part of the individual's identity; e.g., the individual appraises themselves as someone who does not perform well in social situations. In turn, these appraisals can lead to lower social performance, thereby maintaining social anxiety. Thus, when the individual appraises the memory of a negative social (onset) event as central, this could fuel the development of SAD.

MDD and Event Centrality

Following Beck's cognitive model of depression (Beck, 1967), the occurrence of negative events can lead to the development of depressive self-referential schemas and dysfunctional attitudes. Negative events, such as the loss of a significant person, or underperforming at certain key moments, act as catalysts in the development of maladaptive self-referential schemas. In these schemas, the meaning attributed to the event becomes deeply ingrained as stable attitudes, integrated in personal identity. For example, beliefs like "*if I lose an important person, I'll be helpless*", or "*if I fail at something, it means I'm a total failure*" become established. Once these schemas take root, they dynamically interact with an individual's perception of subsequent events, predisposing them to interpret new situations in alignment with their negative attitudes, serving as a reference point for everyday inferences.

The activation of a maladaptive self-referential schema can contribute to an increased vulnerability to depression, acting as a filter through which individuals perceive and evaluate the world. Moreover, the impact of these maladaptive schemas extends beyond the immediate activation by external cues. Appraising the negative event as central to identity could contribute to these maladaptive self-referential schemas: If memories associated with these schemas are easily accessible in autobiographical memory, they can exert a continuous influence even in the absence of explicit external triggers. This persistent influence underscores the enduring nature of maladaptive self-referential schemas and their role in perpetuating and intensifying depressive symptoms over time (Beck, 1967, 2002).

Empirical findings

As discussed, SAD and MDD have been linked to specific onset events (Kendler et al., 1999; Kessler, 1997; Magee, 1999; McCutcheon et al., 2009; Rapee & Spence, 2004). Moreover, empirical research has found that both disorders are related to overlapping cognitive and emotion regulation variables which are linked to event centrality, including trait anxiety, rumination and worry, intrusions and avoidance, and posttraumatic cognitions (Ehring et al., 2006; Gehrt et al., 2018; Muris et al., 2005; Vermeulen et al., 2019). A small positive association between event centrality and SAD has been reported before (Matos et al., 2013). Also, event centrality levels decreased throughout treatment of social anxiety (O'Toole et al., 2018), hinting towards a positive association between event centrality and SAD symptoms. Research on the link between event centrality and depression has, however, provided mixed results**.** Significant positive correlations were found between event centrality and the amount and severity of depression symptoms (Berntsen & Rubin, 2006, 2007; Boals, 2010, 2014; Boelen, 2009, 2012a; Robinaugh & McNally, 2011; Vermeulen et al., 2020), over and above anxiety, avoidance, and neuroticism (Boelen, 2012a), and repetitive negative thinking (Allbaugh et al., 2016). However, other studies could not find a link between event

centrality and currently depressed, recovered, and never-depressed groups (Newby & Moulds, 2011). In addition, the link between event centrality and depression was not consistently found among different cultures in large cross-cultural studies in community samples (Zaragoza Scherman et al., 2015). It has been suggested that depression is affected by the lack of centrality of positive events instead of increased centrality of negative events (Janssen et al., 2015). Moreover, event centrality might have an indirect relation with depression mediated by PTSD symptoms (Vermeulen et al., 2020), negative cognitions, rumination, and avoidance (Boelen, 2012b). This suggests a positive relationship between event centrality and depression; however, this link might be mediated by several maladaptive emotion regulation variables.

Current Study

Most of the above-mentioned studies have been conducted in student or community samples; it is unclear how the relation between event centrality and symptoms manifests in clinical samples. Therefore, the first aim of this study was to explore the association between event centrality and social anxiety/depressive symptoms in a SAD patient sample and a MDD patient sample, where we expected significant positive relations between event centrality and the disorder specific symptoms. Our second aim was to explore potential mediators (i.e., trait anxiety, rumination, worry, intrusions and avoidance, and posttraumatic cognitions) of this relationship within both groups. Based on the results from PTSD samples, we expected each variable to mediate the relationship between event centrality and disorder-specific symptoms individually.

Method

Participants and Design

The current study employed a cross-sectional design, collecting data from two patient groups: a SAD patient group and an MDD patient group. A total of 59 participants were

recruited. Sample size was based on resource limitations (number of patients willing to participate within the timeframe allocated by the participating mental healthcare facilities for this study). Patients descriptives of both groups can be found in Table 1. The SAD group consisted of 38 patients (23 females; 61%) from an outpatient center for specialized mental healthcare in the Netherlands. Participants had a current primary diagnosis of SAD, based on DSM-5 criteria (American Psychiatric Association, 2013), as ascertained by a trained psychologist or psychiatrist (or trainee under supervision) from the participating mental healthcare center as part of their regular intake procedure. Patients with comorbid PTSD, substance abuse, or psychosis were excluded.

The MDD group consisted of 21 patients (12 females; 57%) from a university hospital in Belgium (n = 19) and an outpatient center for specialized mental healthcare in the Netherlands (n = 2). All participants had a current primary diagnosis of MDD based on DSM-IV-TR criteria (American Psychiatric Association, 2000), as ascertained by a trained psychologist or psychiatrist (or trainee under supervision) participating mental healthcare center as part of their regular intake procedure. Patients with comorbid bipolar disorder, PTSD, SAD, substance use, or psychosis were excluded.

Measures

Episode duration and perceived onset event. Participants were asked for the duration of their current episode of SAD or MDD and were asked whether they had experienced an adverse event which was in their opinion related to the onset of their disorder, and if so, to write down the memory of this event.

Centrality of Event Scale (CES; Berntsen & Rubin, 2006; Vermeulen et al., 2020). The abbreviated 7-item version of the Dutch CES (Vermeulen et al., 2020) was used to measure the centrality of the perceived onset event. The questionnaire consists of 7 self-report items, with Likert scale answer options ranging from 1 (*totally disagree*) to 5 (*totally agree*).

Total scores on the CES range between 5-35. The internal consistency in the current samples was α = .79 in the SAD group and α = .84 in the MDD group. Because the abbreviated version of the Dutch CES has been shows a similar level of validity as the 20-item version (Vermeulen et al., 2023) and need for parsimony, we chose to administer the abbreviated (and thus shorter) 7-item version of the CES.

Beck Depression Inventory-II-NL (BDI-II-NL; Beck et al., 1996, 2002). The Dutch translation of the BDI-II-NL (Beck et al., 2002) was used to measure the severity of depressive symptoms. The questionnaire consists of 21 self-report items (score 0–3). Total scores on the BDI-II-NL range from 0 to 63. The internal consistency in our samples was for the SAD group: α = .89; and MDD group α = .82.

Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987; Van Vliet, 1999). The Dutch translation of the LSAS (Van Vliet, 1999) was used to measure symptoms of social anxiety. The questionnaire consists of 24 items, 11 items on social situations, and 13 items on performance anxiety. All items are rated twice: once on level of anxiety, ranging from *none* (0) to *severe* (3), and once on level of avoidance, ranging from *never* (0) to *usually* (3). Total score range between 0 and 144. The internal consistency in the current SAD sample was α = .95 (not assessed in the MDD sample).

Impact of Event Scale (IES; Brom & Kleber, 1985; Horowitz et al., 1979). The Dutch version of the IES (Brom & Kleber, 1985) was used to measure symptoms of intrusions and avoidance related to the memory of the perceived onset event. The questionnaire consists of 15 self-report items, eight items in the intrusion subscale, and seven in the avoidance subscale. Items are rated on a 5-point Likert scale, ranging from 0 (*not at all*) to 4 (*extremely*). Total score range between 0 - 28 (Intrusions; 7 items) and 0 - 32 (Avoidance; 8 items). The internal consistency in the current samples was SAD group: intrusions α = .91; avoidance α = .89; and the MDD group: intrusions α = .88; avoidance α = .56.

Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999; van Emmerik et al., 2006). The Dutch version of the PTCI (van Emmerik et al., 2006) was used to assess posttraumatic cognitions. Participants rate 33 self-report items on a 7-point Likert scale ranging from 1 (*totally disagree*) to 7 (*totally agree*). Total score ranges between 33 and 231. The internal consistency in this sample was SAD group: α = .95; and MDD group α = .91.

State-Trait Anxiety Inventory (STAI-T; Spielberger et al., 1970; Van der Ploeg, 1980). The Dutch version of the STAI-T (Van der Ploeg, 1980) was used to measure trait anxiety. The questionnaire consists of 20 statements, scored on a 4-point Likert scale, ranging from 1 *(almost never)* to 4 *(almost always).* Participants indicate how they feel in general regarding each statement. Total score range between 20 and 80. The internal consistency in this sample was SAD group: α = .87; and MDD group α = .86.

Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990; Van Rijsoort et al., 1999). The Dutch version of the PSWQ (Van Rijsoort et al., 1999) was used to measure pathological worry. The questionnaire consists of 16 self-report items scored on a 5-point Likert scale, ranging from 1 *(not at all)* to 5 *(very)*. Total score range between 16 and 80. The internal consistency in this sample was SAD group: α = .85; and MDD group α = .79.

Ruminative Response Scale (RRS; Raes et al., 2009; Raes & Hermans, 2007; Treynor et al., 2003). The Dutch version of the RRS (Raes & Hermans, 2007) was used to measure trait rumination. The questionnaire consists of 22 self-report items scored on a 4-point Likert scale, ranging from 1 *(almost never)* to 4 *(almost always)*. The total score range between 22 and 88. The internal consistency in this sample was SAD group: α = .90; and MDD group α = .85.

Procedure

Approval for this study was obtained from *[Edited out for blind review in the submitted manuscript]*.

After completing the informed consent form, participants completed in this order the demographic variables (age, sex, nationality, academic degree, and marital status), the disorder-specific questionnaire (SAD group: LSAS; MDD group: BDI-II-NL), a question about episode duration and the perceived onset event, CES, IES, PTCI, BDI-II-NL (added in SAD group), STAI-T, PSWQ, and the RRS. The questionnaires were completed individually (without help of the therapist), using paper and pencil.

The SAD group completed the questionnaires as part of their intake process, i.e., before the start of treatment. In the MDD sample, the questionnaires were distributed to patients who were currently in treatment. Patients could choose when and where to complete the questionnaires and returned the questionnaires in a closed envelope to their therapist, who then delivered the anonymous questionnaires to the researcher.

Statistical analysis

All analyses were conducted using R studio. Missingness in the data was handled using multiple imputations. Data on all missing cases were computed using the predictive mean matching approach. The multiple imputation process was carried out using the MICE package (v3.5.0) in R (Van Buuren & Groothuis-Oudshoorn, 2011) to impute 100 datasets.

Given the relatively small sample size, Bayesian analyses with weakly informative priors for obtaining reliable results were conducted to answer our research questions (McNeish, 2016). Correlational analyses were conducted within patient groups using Bayesian First Aid (v0.1) package in R (Bååth, 2014), which uses wide, uninformative priors. Correlation coefficients with 95% highest-density intervals (HDI) are reported (Kruschke, 2018). To reject the null value of a parameter, the results need to express that the null value excludes the posterior 95% HDI. Thus, we considered a correlation "significant" when the 95% HDI interval did not include zero. The total iterations for the correlations were set at 20000.

Mediation analyses were conducted. Within each patient group, mediation models were tested with CES as independent variable, the disorder-specific symptoms (LSAS or BDI-II) as dependent variable, and IES (intrusions and avoidance), PTCI, RRS, PSWQ, STAIT, and BDI-II-NL (only SAD group) as potential mediators. Because of potential power issues, the depression symptom analysis was additionally conducted in the total (SAD + MDD) sample. The priors for the intercept, the coefficients, and sigma (sd*sd) were set to t-distribution (with *df* =3) to penalize the estimates for a small sample size. Total iterations for the mediation analysis was set to 5000. The significance of indirect effects was evaluated by constructing 95% HDI intervals around them. The indirect effect was interpreted as credible if the 95% HDI's did not include zero. Completely standardized indirect effects were calculated (Preacher & Kelley, 2011), as these effects can be used to compare the different effects sizes across populations or different metrics in populations (Cheung, 2009). The mediation models were fitted using the brms package (v2.9.0) in R (Bürkner, 2017). Model components (e.g., coefficients, intercepts, indirect effects) are interpreted as they typically are in mediation models. As typical for Bayesian analysis, no adjustment for multiple testing was made (Gelman et al., 2012). Coefficients of the indirect effects were reported by *a*b.* We report how we determined our sample size, all data exclusions (if any), all manipulations, and all measures in the study. Data, materials, and analysis code for this study are available by e-mailing the corresponding author.

Perceived onset events were scored ad hoc (as suggested by one of our reviewers) on PTSD criterion A (American Psychiatric Association, 2013, p. 271), general life event (e.g., divorce, surgery, moving), or social/relational events (e.g., social conflict, bullying) by two independent raters. It was possible for descriptions to be scored in multiple categories as some contained multiple events. Interrater reliability was calculated with Cohen’s Kappa.

Disagreements (n = 8) were resolved through discussion. Differences in reporting each category of events between the SAD and MDD groups were tested with Chi-Square tests.

Results

Descriptive statistics

Means and SDs of the descriptive variables per patient group and possible significant differences between the two groups can be found in Table 1. A significant age difference between the two patient groups was found. Regarding the perceived onset event, 26 patients (68.4%) in the SAD group and 18 patients (85.7%) in de MDD group indicated that they had experienced an adverse event which was in their perception related to the onset of their disorder. Differences in participants' descriptives between those who reported a perceived onset event and those who did not can be found in Table 2, showing no significant difference between these two groups. Missingness analyses showed that 37 (62.7%) participants completed all items across measures. Overall, 8% of missingness was registered.

Correlations

Correlations within the patient groups can be found in Table 3 (the top half [above the diagonal] shows the correlations within the SAD group, the bottom half [under the diagonal] shows the correlations within the MDD group). All correlations were positive, ranging between .10 and .79 in the SAD group and between .29 and .74 in the MDD group. In the SAD group, the results showed that CES scores were significantly correlated, in descending order from large to moderate, with IES-avoidance, IES-intrusions, and the PTCI (posttraumatic cognitions) only.

In the MDD group, the CES was significantly and positively correlated, in descending order, from large to moderate, with PTCI (posttraumatic cognitions), IES-intrusions, RRS (rumination), STAI-T (trait-anxiety), IES-avoidance, and BDI-II (depressive symptoms), but not to the PSWQ (worry).

Mediation analyses

Results of the within-group mediation analyses can be found in Figures 1 and 2 and Tables 4 and 5. In the within-group mediation analyses of the SAD group (Table 4 and Figure 1), the results showed that none of the variables (PTCI, IES-intrusions, IES-avoidance, RRS , STAI-T, PSWQ, and BDI-II) mediated the relation between the CES and LSAS; neither significant direct effect nor significant indirect effects could be found. In the MDD group (Figure 2 and Table 5), a significant direct effect was found between the CES and BDI-II-NL with PSWQ as mediating variable, indicating no effect of worry as mediator. Significant indirect effects were found in the analysis with PTCI and STAI-T as mediators, indicating the relation between event centrality and depressive symptoms was mediated by posttraumatic cognitions and trait anxiety.

Based on the correlations reported in Table 3, additional significant mediation effects were expected, indicating a possible statistical power issue. Therefore, both patient groups were combined to increase the sample size and, thereby, power. Results can be found in Figure 3 and Table 6. A significant direct effect between the CES and BDI-II-NL was again found for PSWQ as mediator, again indicating that worry did not mediate the relation between event centrality and depressive symptoms. Significant indirect effects were found in all other analyses with as mediator; IES-intrusions, IES-avoidance, PTCI, RRS, and STAI-T. This indicates that the relation between the event centrality and depressive symptoms was mediated by intrusions, avoidance, posttraumatic cognitions, rumination, and trait anxiety.

Ad hoc analyses on perceived onset memory content

Interrater reliability of the scored events was high, Cohen's $\kappa = 0.84$. The MDD group reported significantly more PTSD A criterion events than the SAD group, $\chi^2(1) = 7.52$, $p = .006$, whereas the groups did not differ in the number of reported general events, $\chi^2(1) = 2.68$,

p = .10. Finally, significantly more social/relational events were reported in the SAD group than the MDD group $\chi^2(1) = 4.39$, p = .036.

Discussion

Our aim was to study the relation between event centrality and depressive and social anxiety symptoms, and their mediators (i.e., intrusions, avoidance, posttraumatic cognitions, rumination, trait anxiety, and worry) in two clinical groups of patients diagnosed with SAD or MDD.

Event Centrality in SAD

Although event centrality was moderate to strong and positively correlated with intrusions, avoidance, and posttraumatic cognitions within the SAD group, contrasting to our hypothesis, event centrality scores were not correlated with social anxiety symptoms. However, the size of the correlations was similar to those reported by Gehrt et al. (2018), where they reached significance, probably due to a larger sample size. In addition, a substantial portion (46%) of individuals in the SAD group did not report a perceived onset event, for which their most negative life event might (which they had in mind while completing the CES) might not have been related to their disorder specific symptoms. The lack of a significant correlation between event centrality and social anxiety symptoms might also be explained by certain characteristics of SAD. Event centrality represents cognitions and appraisals about a specific (negative) event (Berntsen & Rubin, 2006), whereas SAD is characterized by maladaptive cognitions about the self in more general terms (Hofmann, 2007), such as low self-esteem (Rapee & Heimberg, 1997). SAD typically develops more gradually during adolescence and might be linked to more general schemas related to less salient but common occurrences (e.g., implicit messages that the child is not wanted, Young, 1999). Event centrality appraisals such as "this event changed my life" (Berntsen & Rubin,

2006) might not be of specific impact on the development and maintenance of SAD as it is in PTSD. Indeed, research has shown that the development of SAD is more often preceded by events of more subtle emotional messages of, for example, shame (Magee, 1999; Rapee & Spence, 2004), whereas MDD and PTSD are more often linked with intense emotional, physical, and/or sexual (childhood) abuse (Maniglio, 2010; Wiersma et al., 2009). This aligns with the results of our content analyses of the reported perceived onset events, where the MDD group reported more traumatic events (PTSD criterion A) and the SAD group reported more non-traumatic social/relational events (e.g., social conflict, bullying) as perceived onset events of their symptoms.

Event Centrality in MDD

Within the MDD group, event centrality and depressive symptoms were moderately and positively correlated, aligning with our expectations. Event centrality was significantly correlated with all variables except worry. Posttraumatic cognitions and trait anxiety mediated the relation between event centrality and depression in the MDD sample, consistent with the conceptualization of MDD as a post-trauma disorder (Radell et al., 2020) with a high comorbidity with PTSD (Angelakis & Nixon, 2015). A remarkable result was the surprisingly low internal consistency of avoidance symptoms as measured with the IES in the MDD group ($\alpha = .56$). As we are unaware of other studies that reported similar results, we expect this problem to be caused by the small sample size.

In the combined group mediation analysis, all variables except worry were significant mediators of the relationship between event centrality and depression symptoms, indicating preliminary evidence for a transdiagnostic link between event centrality and depressive symptoms. The absence of worry as a mediator indicates that event centrality is typically more focused on the past than the future, challenging the theoretical assumption of event centrality (Berntsen & Rubin, 2006) stating that central event form a cognitive reference

point, and thereby influence the interpretation of both past experiences and future expectations. This highlights an avenue for future research to explore this aspect more comprehensively.

In sum, our findings confirm the results of earlier studies supporting a direct relation between event centrality and depressive symptoms (Allbaugh et al., 2016; Berntsen & Rubin, 2006, 2007; Boals, 2010, 2014; Boelen, 2009, 2012a, 2012b; Robinaugh & McNally, 2011; Vermeulen et al., 2020). Our findings also align with Beck's cognitive model of depression (Beck, 1967), providing a nuanced understanding of how event centrality contributes to depressive symptomatology: The observed direct association between event centrality and depressive symptoms resonates with Beck's conceptualization that negative events serve as catalysts for the development of maladaptive self-referential schemas and dysfunctional attitudes.

The results of the mediation analysis are consistent with earlier research (Boelen, 2012b; Vermeulen et al., 2020), which demonstrated that the relationship between event centrality and depressive symptoms is mediated by PTSD symptoms, negative cognitions, and rumination. Furthermore, our mediation analysis provides concrete evidence that supports the cognitive processes delineated in Beck's model (Beck, 1967, 2002). The presence of mediating variables, including PTSD symptoms, negative cognitions, and rumination, signifies a pathway through which event centrality influences depressive symptoms. This mediation pathway echoes Beck's proposition that maladaptive self-referential schemas dynamically interact with perceptions of subsequent events, predisposing individuals to interpret new situations in a manner consistent with their negative attitudes. Generally, the impact of event centrality appears to be more pronounced in PTSD (Boals & Murrell, 2016; Gehrt et al., 2018) and MDD than in other disorders such as SAD (as observed in the current study).

Clinical implications

Our findings hold clinical implications. First, there are notable similarities in the correlations of event centrality between PTSD and MDD, and both disorders can develop following a traumatic experience (Angelakis & Nixon, 2015; Radell et al., 2020). This suggests that specific negative events may contribute to or trigger the onset of both PTSD and MDD, potentially through the mechanism of event centrality. In the case of PTSD, a clear (traumatic) onset event is even a requirement for diagnostic classification. One could argue that a similar criterion should be considered for MDD. However, establishing the causal role of any perceived onset event is highly challenging in MDD. Additionally, many MDD patients do not perceive a specific onset event for their episode. Therefore, we do not recommend including a central onset event in the classification criteria for MDD. Nevertheless, in a more comprehensive diagnostic interview, it might be valuable to inquire about perceived onset events and event centrality as this could guide treatment direction (e.g., identifying key negative cognitions). We did not find any specific evidence to support this approach for SAD, so we will not make these specific recommendations for SAD treatment.

Limitations

Several limitations have to be taken into account when interpreting the results of the current study. The main limitation of our study was the small sample size of the two groups suggesting that some existing correlations (Gehrt et al., 2018) might not have been detected in our study due to lack of statistical power (type-II error). Furthermore, there was a large difference in age range between the patient groups (i.e., *SAD-Mage* = 30.66; *MDD-Mage* = 49.48). These age ranges fit with the prevalence of these specific disorders thereby reflecting typical patient samples. However, age may have been a confounding factor in the combined group for data-analysis. For example, age is a relevant factor in trauma-related psychopathology, where a younger age might be an increased vulnerability of PTSD

(Kongshøj & Berntsen, 2022). Thus, if possible, future research should control for age range differences by matching groups on age. Moreover, 46% of patients in the SAD group did not report an event linked to the onset of their disorder. This makes it unclear what event they had in mind while answering the questionnaires. We suggest that future studies record a brief description of the specific memory which they had in mind while completing the questionnaires, in addition to requesting a description of their identified onset event. Furthermore, we did not include event centrality assessments for neutral events, positive events, or negative events not perceived as related to the onset of the disorder. Consequently, it is not feasible to determine whether the observed event centrality relations are exclusive to the perceived onset events or if they represent a more general bias towards event centrality bias within individual participants. Future research should include this and control for this possible bias. As finial limitation, the cross-sectional design of this study limits our ability to draw causal inferences from the mediation analyses. Future research should employ a longitudinal design to examine the potential causal effects and the temporal ordering of the hypothesized mediation processes.

Conclusion

In sum, we found that event centrality and social anxiety symptoms were not significantly correlated. A positive strong correlation was found between event centrality and depressive symptoms in the sample of MDD patients. Posttraumatic cognitions and trait anxiety mediated the role between event centrality and depressive symptoms in this group. In the combined transdiagnostic sample, we found that intrusions, avoidance, posttraumatic cognitions, rumination, and trait anxiety mediated the relation between event centrality and depressive symptoms. Our results indicate that event centrality may play a larger role in MDD (or trauma-related disorders such as PTSD and MDD) than SAD. When treating MDD (but not SAD), it might be important to ask whether the client associates the onset of their

symptoms with a particular event, and if so, target cognitions of event centrality in treatment. The supported mediators (i.e., intrusions, avoidance, posttraumatic cognitions, rumination, and trait anxiety) might be important for future exploration in understanding the persistence of depression.

Compliance with Ethical Standards

Conflict of interest: All authors have stated that they have no potential conflict of interest pertaining to this submission to Memory

Ethical Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent: Informed consent was obtained from all individual participants included in the study.

Animal Rights: This article does not contain any studies with animals performed by any of the authors.

Open data: The data, materials, and analysis code that support the findings of this study are available from the corresponding author *[corresponding author's email address]* upon reasonable request.

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Appendix

Table 1:

Means and Standard deviations of all variables in the two patient groups and differences between the two groups.

	SAD *M (SD)*	MDD *M* (*SD*)	*test*	*value*	Cohen's *d*
Number of participants	38	21			
Age (years)	30.66 (10.94)	49.48 (14.37).	*t*	5.65***	0.36
Gender (M/F)	15/23	9/12	χ^2	0.06	
Reported onset (yes/no)	26/12	18/3	χ^2	2.13	
Disorder duration (months)	141.94 (120.51)	128.20 (154.26)	*T*	0.37	0.28
Education (primary/secondary/higher)	1/22/16	2/15/4	χ^2	3.41	
CES	25.27 (4.93)	28.04 (5.39)	*t*	1.99[a]	0.54
LSAS	72.68 (24.65)	-			
BDI-II	25.02 (4.93)	32.50 (8.56)	*t*	2.71**	0.78
IES-I	14.43 (6.68)	23.58 (6.81)	*t*	4.82***	1.36
IES-A	16.14 (7.44)	22.00 (5.09)	*t*	2.86**	0.92
PTCI	127.54 (34.44)	123.75 (28.75)	*t*	.38	0.12
RRS	51.71 (10.56)	61.35 (9.76)	*t*	3.33**	0.95
STAI-T	59.91 (8.18)	63.00 (8.28)	*t*	1.32	0.37
PSWQ	66.31 (9.50)	61.12 (10.21)	*t*	1.81[a]	0.53

Note: CES = Centrality of Event Scale; LSAS = Liebowitz Social Anxiety Scale; BDI-II-NL= Beck Depression Inventory-II; IES-I = Impact of Event Scale – Intrusions; IES-A = Impact of Event Scale –Avoidance; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative

Response Scale; STAI-T = State-Trait Anxiety Inventory - Trait; PSWQ = Penn State Worry Questionnaire. Standard deviations are noted between brackets.

[a] <.10; * < .05; ** < .01; *** < .001

Table 2:

Participant's descriptives and between group differences of participants who did or not reported an onset event

	Onset - Yes *M* *(SD)*	Onset - No *M* (*SD*)	*test*	*value*
Number of participants	44	15		
Group (MDD/SAD)	18/26	3/12	χ^2	2.13
Gender (M/F)	17/27	7/8	χ^2	0.30
Age (years)	35.80 (16.80)	37.89 (14.74).	*t*	0.46
Disorder duration (months)	131.42 (128.23)	153.86 (147.66)	*t*	0.55

Note: Standard deviations are noted between brackets. None of the differences were significant.

Table 3:

Correlations between all variables within the two patient groups. The top half (above the diagonal) shows the results of the SAD group, the bottom half (under the diagonal) shows the results of the MDD group. Values in bold show significant correlations.

Variables	CES	LSAS	BDI-II	IES-I	IES-A	PTCI	RRS	STAI-T	PSWQ
CES	1.00	0.10 [-0.26, 0.44]	0.17 [-0.17, 0.49]	**0.53 [0.26, 0.76]**	**0.54 [0.28, 0.77]**	**0.36 [0.02, 0.63]**	0.28 [-0.05, 0.59	0.25 [-0.10, 0.56]	0.33 [0.00, 0.62]
LSAS	-	1.00	**0.54 [0.25, 0.78]**	0.20 [-0.16, 0.54]	0.28 [-0.07, 0.58]	**0.41 [0.10, 0.71]**	**0.50 [0.20, 0.77]**	**0.46 [0.14, 0.73]**	**0.42 [0.10, 0.70]**
BDI-II	**0.51 [0.13, 0.80]**	-	1.00	**0.46 [0.17, 0.72]**	**0.59 [0.33, 0.80]**	**0.57 [0.29, 0.78]**	**0.59 [0.32, 0.80]**	**0.68 [0.47, 0.86]**	**0.38 [0.06, 0.67]**
IES-Int	**0.65 [0.32, 0.87]**	-	**0.53 [0.15, 0.82]**	1.00	**0.79 [0.57, 0.93]**	**0.55 [0.28, 0.76]**	0.51 [0.23, 0.756]	**0.48 [0.19, 0.73]**	0.31 [-0.03, 0.59]
IES-avoi	**0.53 [0.16, 0.85**]	-	**0.71 [0.38, 0.91]**	**0.49 [0.03, 0.83]**	1.00	**0.70 [0.50, 0.85]**	**0.50 [0.22, 0.74]**	**0.56 [0.29, 0.77]**	0.25 [-0.09, 0.56]
PTCI	**0.67 [0.32, 0.91]**	-	**0.64 [0.28, 0.89]**	**0.63 [0.22, 0.89]**	**0.55 [0.13, 0.86]**	1.00	**0.55 [0.28, 0.77]**	**0.60 [0.34, 0.80]**	**0.77 [0.60, 0.90]**
RRS	**0.60 [0.26, 0.85]**	-	**0.55 [0.20, 0.83]**	**0.65 [0.35, 0.88]**	**0.53 [0.11, 0.85]**	**0.63 [0.26, 0.90]**	1.00	**0.36 [0.02, 0.64]**	**0.57 [0.30, 0.79]**
STAI-T	**0.54 [0.14, 0.84]**	-	**0.74 [0.48, 0.91]**	**0.63 [0.28, 0.88]**	**0.62 [0.25, 0.89]**	**0.58 [0.19, 0.87]**	**0.65 [0.32, 0.88]**	1.00	**0.61 [0.35, 0.81]**
PSWQ	0.35 [-0.14, 0.77]	-	**0.57 [0.17, 0.86]**	0.34 [-0.16, 0.74]	**0.53 [0.06, 0.86]**	0.30 [-0.21, 0.75]	0.29 [-0.18, 0.72]	**0.59 [0.20, 0.87]**	1.00

Note: CES = Centrality of Event Scale; LSAS = Liebowitz Social Anxiety Scale; BDI-II-NL= Beck Depression Inventory-II; IES = Impact of Event Scale; PTCI = Posttraumatic Cognitions Inventory; RRS = ruminative response scale; STAI-T = State trait anxiety inventory - trait; PSWQ = Penn State Worry Questionnaire. The table's top half (above the diagonal) shows the correlations within the SAD group, and the bottom half (under the diagonal) shows the correlations within the MDD group. HDI credibility intervals are shown between square/box brackets.

Table 4:

Within group mediation analysis for the relation between event centrality and SAD.

Mediator	*A*		*B*		*C*		*Indirect effect*		
	β_1 (S.E.)	95% CI	β_2 (SE)	95% CI	β_3 (SE)	95% CI	*a*b*	Standardized	95% CI
IES-I	.740 (.196)	.347, 1.136	.869 (.778)	-.689, 2.426	-.005 (1.025)	-2.070, 2.036	.598	.120	-.538, 1.974
IES-A	.911 (.216)	.477, 1.347	1.428 (.682)	.042, 2.777	-.665 (1.029)	-2.719, 1.394	1.244	.249	-.023, 2.799
PTCI	2.618 (1.115)	.288, 4.873	.283 (.124)	.034, .530	-.082 (.849)	-1.802, 1.608	.679	.136	-.066, 1.735
RRS	.737 (.349)	.022, 1.422	1.239 (.383)	.453, 1.999	-.267 (.799)	-1.884, 1.328	.841	.168	-.050, 2.018
STAI-T	.415 (.285)	-.156, .991	1.624 (.432)	-.749, 2.491	-.042 (.746)	-1.544, 1.453	.632	.126	-.302, 1.758
PSWQ	.671 (.306)	.059, 1.289	1.153 (.439)	.273, 2.035	-.134 (.834)	-1.803, ,1.550	.712	.142	-.058, 1.774
BDI-II	.395 (.366)	-.334, 1.128	1.117 (.386)	.384, 1.925	-.184 (.755)	-1.337, 1.715	.411	.082	-.441, 1.469

Note: IES = Impact of Event Scale; PTCI = Posttraumatic Cognitions Inventory; RRS = ruminative response scale; STAI-T = State trait anxiety inventory - trait; PSWQ = Penn State Worry Questionnaire; BDI-II-NL= Beck Depression Inventory II. Equations: Mediator = β_0^m * Intercept + β_1 * Centrality (*A*); SAD = β_0^c * Intercept + β_2 * Mediator (*B*) + β_3 * Centrality (*C*). Sample size = 59; Multiply imputed data (*m* = 100); Priors = T-distribution for the intercept, the coefficients, and sigma. Significant indirect effects are displayed in bold.

Table 5:

Within group mediation analysis for the relation between event centrality and MDD.

Mediator	*A*		*B*		*C*		*Indirect effect*		
	β_1 (S.E.)	95% CI	β_2 (S.E.)	95% CI	β_3 (S.E.)	95% CI	*a*b*	Standardized	95% CI
IES-I	.831 (.220)	.375, 1.294	.330 (.359)	-.409, 1.055	.573 (.432)	-.316, 1.458	.254	.160	-.362, .955
IES-A	.504 (.189)	.110, .900	.864 (.443)	-.052, 1.734	.417 (.353)	-.307, 1.154	.396	.249	-.075, .1.024
PTCI	**3.665 (.905)**	**1.793, 5.533**	**.179 (.080)**	**.014, .342**	**.197 (.405)**	**-.662, 1.038**	**.630**	**.397**	**.003, 1.388**
RRS	1.123 (.335)	.428, 1.808	.338 (.221)	-.117, .793	.471 (.390)	-.332, 1.127	.351	.221	-.159, 1.001
STAI-T	**.885 (.295)**	**.280 1.490**	**.691 (.217)**	**.226, 1.140**	**.239 (.316)**	**-.421, .901**	**.579**	**.365**	**.061, 1.209**
PSWQ	.510 (.428)	-.367, 1.394	.334 (.175)	-.026, .690	.678 (.309)	.044, 1.315	.141	.088	-.150, .605

Note: IES = Impact of Event Scale; PTCI = Posttraumatic Cognitions Inventory; RRS = ruminative response scale; STAI-T = State trait anxiety inventory - trait; PSWQ = Penn State Worry Questionnaire. Equations: Mediator = β_0^m * Intercept + β_1 * Centrality (*A*); MDD = β_0^c * Intercept + β_2 * Mediator (*B*) + β_3 * Centrality (*C*). Sample size = 59; Multiply imputed data (*m* = 100); Priors = T-distribution for the intercept, the coefficients, and sigma. Significant indirect effects are displayed in bold.

Table 6:

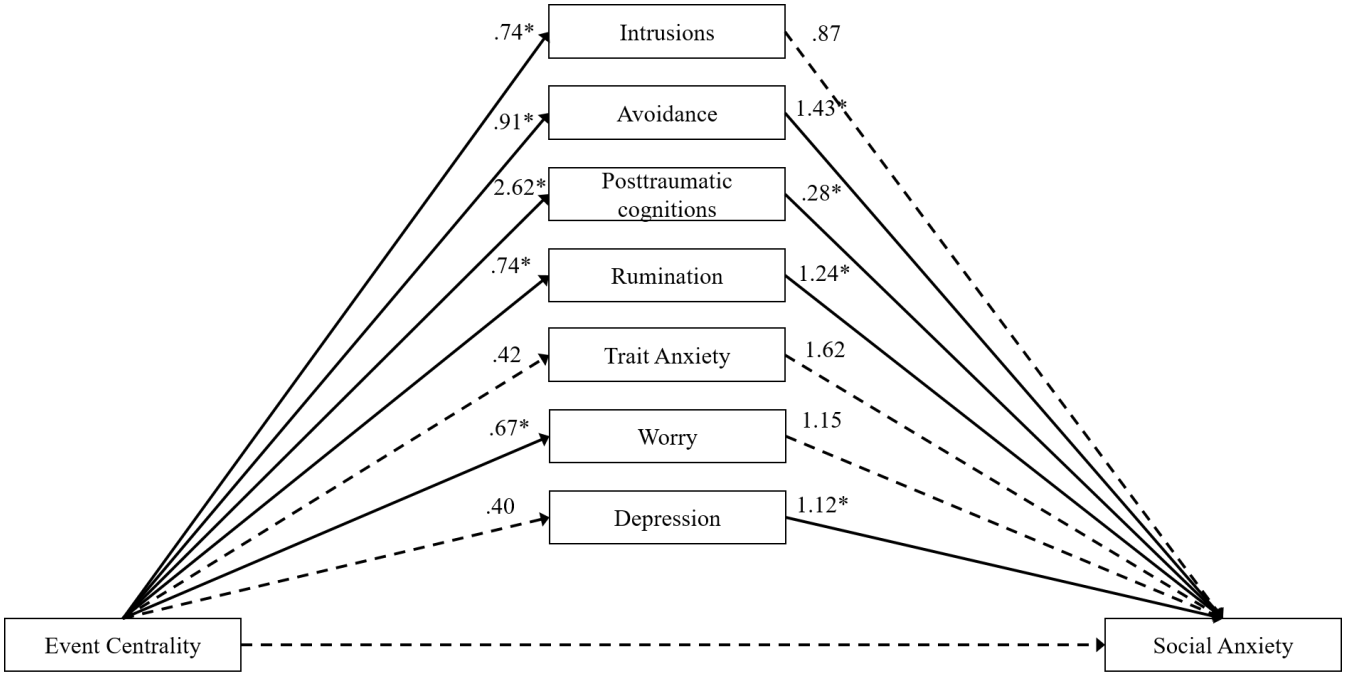
Combined sample mediation analysis for the relation between event centrality (CES) and MDD.

Mediator	*A*		*B*		*C*		*Indirect effect*		
	β_1 (S.E.)	95% CI	β_2 (S.E.)	95% CI	β_3 (S.E.)	95% CI	*a*b*	Standardized	95% CI
IES-I	**.949 (.162)**	**.623, 1.169**	**.702 (.191)**	**.321, 1.080**	**.049 (.287)**	**-.522, .6118**	**.652**	**.328**	**.253, 1.100**
IES-A	**.836 (.158)**	**.523, 1.157**	**1.025 (.174)**	**.684, 1.373**	**-.143 (.244)**	**-.629, .341**	**.844**	**.425**	**.440, .1.295**
PTCI	**2.903 (.742)**	**1.420, 4.377**	**.171 (.040)**	**.091, .251**	**.224 (.249)**	**-.273, .718**	**.482**	**.243**	**.171, .854**
RRS	**1.042 (.249)**	**.544, 1.534**	**.582 (.116)**	**.351, .872**	**.108 (.239)**	**-.368, .584**	**.593**	**.299**	**.249, .998**
STAI-T	**.642 (.200)**	**.244, 1.039**	**.855 (.139)**	**.597, 1.110**	**.165 (.204)**	**-.242, .572**	**.539**	**.271**	**.180, .937**
PSWQ	.503 (.252)	.006, 1.009	.210 (.137)	-.063, .481	.609 (.257)	.098, 1.118	.090	.045	-.049, .310

Note: IES = Impact of Event Scale; PTCI = Posttraumatic Cognitions Inventory; RRS = ruminative response scale; STAI-T = State trait anxiety inventory - trait; PSWQ = Penn State Worry Questionnaire. Equations: Mediator = β_0^m * Intercept + β_1 * Centrality (*A*); MDD = β_0^c * Intercept + β_2 * Mediator (*B*) + β_3 * Centrality (*C*). Sample size = 59; Multiply imputed data (*m* = 100); Priors = T-distribution for the intercept, the coefficients, and sigma. Significant indirect effects are displayed in bold.

Figure 1

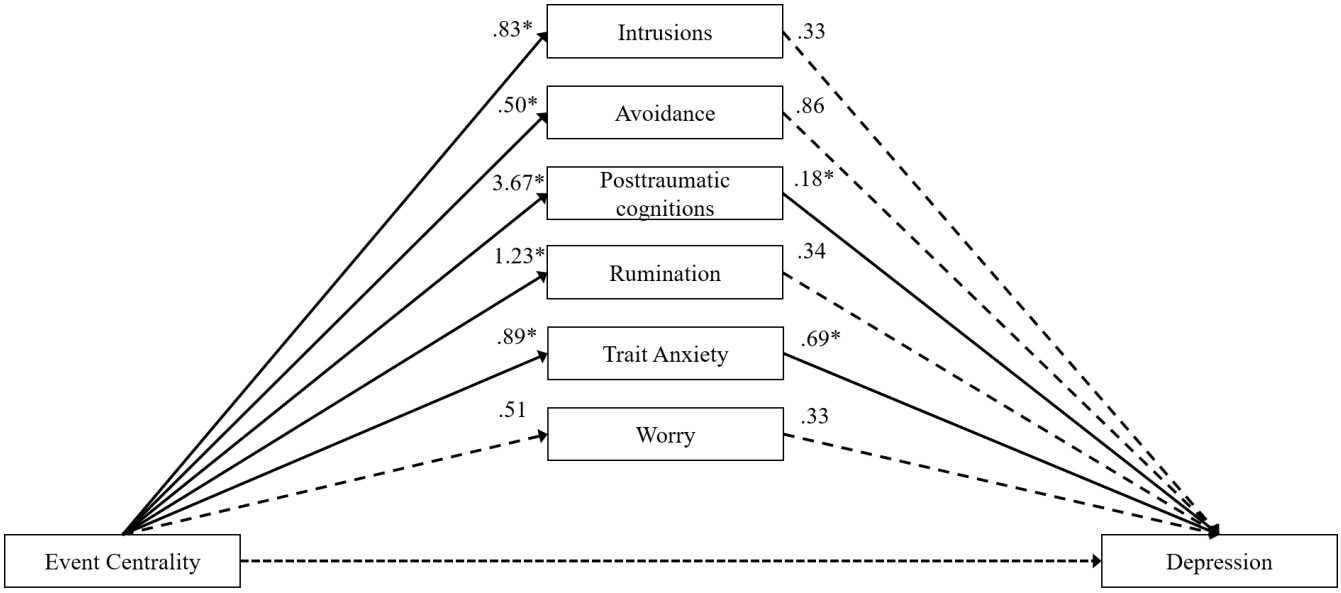
Mediation analyses of the effect of event centrality on social anxiety symptoms through the different variables in the SAD sample



Note. None of the variables significantly mediated the link between event centrality and social anxiety symptoms. Dotted lines represent non-significant betas. Total results of the Bayesian mediation analysis can be found in Table 4.

Figure 2

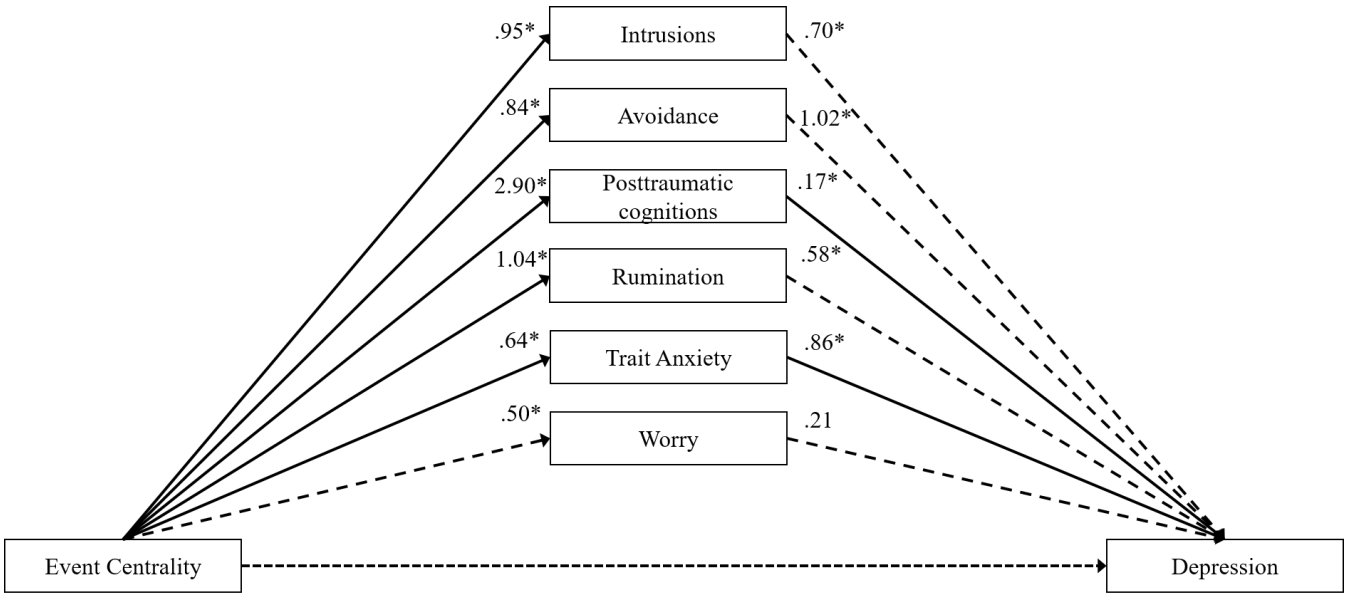
Mediation analyses of the effect of event centrality on depressive symptoms through the different variables in the MDD sample



Note. Posttraumatic cognitions and Trait Anxiety mediated the link between event centrality and depressive symptoms. Dotted lines represent non-significant betas. Full results of the Bayesian mediation analysis can be found in Table 5.

Figure 3

Mediation analyses of the effect of event centrality on depressive symptoms through the different variables in the combined sample



Note. All variables, except worry, mediated the relation between event centrality and depressive symptoms. Dotted lines represent non-significant betas. Full results of the Bayesian mediation analysis can be found in Table 6.