Review article

Cognitive Reactivity to Sad Mood as a Risk Factor for Depressive Recurrence

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Abstract

According to cognitive theories of depression, vulnerability for onset and recurrence lies in some kind of cognitive dysfunction, or maladaptive cognitive information processing. This literary review paper aims to clarify the role of cognitive reactivity to sad mood as a risk factor to depressive recurrence, mechanism and measurement in order to provide tailor treatment and prevention for depression. Comprehensive searches of PsycInfo, PubMed and Web of Science were conducted. Teasdale's differential activation hypothesis suggests that the initial depressive episode establishes specific dysfunctional patterns of processing that lie latent after recovery but that can be reactivated by depressed mood. This activation would in turn strengthen the dysfunctional processing patterns and thereby create a vicious loop of depression recurrence. Teasdale calls this concept cognitive reactivity. Cognitive Reactivity (CR) is the extent to which an individual experiences a negative shift in cognitive content and processes during a sad mood. Finding from our review suggests that CR can be conceptualized as a risk factor that is present in vulnerable individuals before depression-onset, that distinguishes between vulnerable and non-vulnerable groups even when in remission, and that predicts depression relapse.There also research evidence that the duration of the first depressive episode, regardless of its intensity, is of crucial importance in the formation of cognitive reactivity to sad mood.Based on findings from different countries we conclude that Index for Depression sensitivity, LEIDS-R is a promising test for measuring cognitive reactivity to sad mood (CR). The results obtained from previous research indicate the importance of an interactive approach when examining the factors that contribute to the recurrent course of depression.

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Introduction

Major Depressive Disorder (MDD) is a heavy burden, for vulnerable individuals and for society as a whole.The prognosis of affective disorders, including depression, is generally good, and a large number of patients achieve satisfactory or complete remission. One of the disabling aspects of major depressive disorder (MDD) is its recurrent nature (1). In about a third of patients, MDD becomes recurrent (2) and the risk of recurrences increases after each episode (3). Recurrent depressive disorder often results in complete remission, but cases with persistent limiting dysfunctional symptomatology are also common. Impairment and suffering result both from the initial episode and later recurrence, and identifying risk factors for depressive recurence is therefore pivotal for decreasing its detrimental effects through effective personalized treatment and prevention strategies.

During the last decades, research has been presented that focused on various demographic risk factors for the occurrence, but not recurrence of depression (marital status, gender, negative life events, and Socio-Economic Status (SES) (4). On the other hand, another group of factors, such as : age of onset, number of episodes, and episode severity, together with comorbidity and family history of psychopathology are all implicated in the recurrence of depression (4). But what they all have in common is that these risk factors are either unchangeable or hard to modify, and it may be difficult to tailor treatment and prevention efforts according these variables.

According to cognitive theories of depression, vulnerability for onset and recurrence lies in some kind of cognitive dysfunction, maladaptive cognitive information processing.

This literary review paper aims to clarify the role of cognitive reactivity to sad mood as a risk factor to depressive recurrence, mechanism and measurement in order to provide tailored treatment and prevention for depression.

Method: Comprehensive searches of PsycInfo, PubMed and Web of Science were conducted. We decided on a broader search to not miss out on any potentially eligible studies by frequent use of "or" rather than strict search terms. The keywords searched were "Cognitive AND (reactivity OR dysfunction*) AND depress* AND (predict* OR relapse OR recur*).

Defining cognitive reactivity to sad mood (CR)

Decades of research into the role of cognitive factors in depression have provided support for many aspects of cognitive theories of depression (5). The theoretical-empirical framework from which we started when postulating the problem of this review paper is the paradigm of maladaptive information processing. Despite the revolution in the theory, research, and treatment of depression, existing theories have been insufficient to explain how cognitive patterns, which are latent in nature, contribute to the recurrent course of the disorder. In the last decades, research has focused on examining the role of dysfunctional thought patterns in the development of depression, and a number of findings have resulted in contradictory interpretations of the status of cognitive factors as causative factors or consequences of their depressive state (5,6,7). It became clear that the mechanisms responsible for maintaining depression should not be sought at the level of content, but at the level of processes. In response to these criticisms, studies and models followed that emphasized less "what" and more began to consider "how" those contents become active.

Different theories and hypothesis have been proposed to explaine activation of cognitive schemas. We would like to emphasise the hypothesis of differential activation (8) by which Teasdale, in addition to the stressor as an activator of the schema, proposes another way how the schema can be activated.

He builds on Beck's cognitive model (9) but proposes that the presence of negative affectivity that existed at times when the depressive schema was active can reactivate the schema.

The mentioned hypothesis assumes that during the early episodes of depression, certain patterns of processing are formed, thus forming a link between dysphoria and negative cognitions. It refers to the interaction of affect and cognition and their role as mechanisms that explain the recurrent course of depressive disorder. Teasdale's differential activation hypothesis (10) suggests that the initial depressive episode establishes specific dysfunctional patterns of processing that lie latent after recovery but that can be reactivated by depressed mood. This activation would in turn strengthen the dysfunctional processing patterns and thereby create a vicious loop of depression recurrence (11).

Teasdale calls this concept cognitive reactivity (12). Cognitive Reactivity is defined as the negative change in cognitive processes and content during a sad mood state (CR) (13). Cognitive Reactivity (CR) is the extent to which an individual experiences a negative shift in cognitive content and processes during a sad mood.

The differential activation hypothesis has been tested by correlational studies as well as studies with the induction of depressed affect in depressed patients in remission (14). Also, affect-induction experiments confirm the existence of latent depressive patterns in individuals who have gone through a depressive episode, but it remains unclear how after the depressive episode, the pattern becomes latent again. It was assumed that with the change of the conditions in which the person is, the depressive cognitions withdraw and eventually become inactive again, thus enabling a return to a normal emotional state.

The mechanisms of the recurrent course of depression

Contemporary cognitive-behavioral theories have explained these findings by arguing that dysfunctional cognitions do not disappear during the remission of a depressive episode, but rather remain "latently present" and that they can easily be activated by a sad/dysphoric mood that occurs on the occasion of an unfavorable event (15). An important dilemma in the field of cognitive vulnerability to depression concerns whether such vulnerability is related to the initial episode of depression and/or to the recurrent course of the disorder. Behind the notion of cognitive vulnerability, there is an implicit idea that cognitive vulnerability participates both in the development of the first symptoms and in the development of repeated episodes.

Research on cognitive reactivity to sadness arises as a direct consequence of negative research findings on the stability hypothesis of dysfunctional attitudes and beliefs. According to the stability hypothesis, dysfunctional attitudes and beliefs represent a stable cognitive trait, so it is logical to expect that in vulnerable persons they are registered before the episode of depression, as well as after the symptoms of acute depression disappear. However, the results of research on these hypotheses show that individuals who later entered an episode of clinical depression did not have more dysfunctional attitudes before the episode, than people who did not enter a depressive episode. Endorsement of dysfunctional attitudes declines after depressed individuals enter remission (16).

The above-mentioned research indicated the existence of cognitive reactivity, defined as a person's tendency to react to sadness by producing negative thoughts that are characteristic of depression.

Cognitive reactivity scores are higher in patients treated with antidepressants compared to those treated with cognitive therapy. Furthermore, high cognitive reactivity predicted depressive relapse, independent of prior treatment modality, and a low level of cognitive reactivity to sad mood and a high level of decentering ability was associated with the lowest recurrence rate during the 18-month follow-up (17).

While the contribution of cognitive reactivity to depressive relapses has been empirically investigated, little is still known about the origins and generation of this reactivity. Some authors (18) suggest that it is a remnant of previous depression, which is activated by the

sad/dysphoric mood. In people who are not prone to depression, the cognitive-affective activity quickly subsides, but in people who are vulnerable to depression, the negative spiral continues, in which there is a wider and more elaborate processing of depressive thoughts and intensification of symptoms characteristic of clinical depression.

In this regard, the duration of the first depressive episode, regardless of its intensity, is of crucial importance in the formation of cognitive reactivity to sad mood (16).The summary of meta-analysis conducted by Duaas Nymoen (11) suggests that CR can be conceptualized as a risk factor that is present in vulnerable individuals before depression-onset, that distinguishes between vulnerable and non-vulnerable groups even when in remission, and that predicts depression relapse. The 23 experiments that she analyzed in the meta-analyses on cross-sectional studies vary widely in methodological as well as participant variables, strengthening the generalizability of the findings and supporting the conclusion that there is a moderate CR difference between euthymic ND and FD participants. The 7 longitudinal studies as a part of this meta-analysis, suggested that CR is predictive of depression relapse, with no significant moderators (11).

Measuring Cognitive Reactivity (CR) to sad mood

Cognitive reactivity as a central term of the differential activation hypothesis refers to the idea that once established, negative thought patterns can be easily reactivated through very small triggers, such as subtle changes in mood. Dysfunctional attitudes are considered important causal and maintaining factors of depression, and for that reason most research to date investigates the potential relationship between cognitive reactivity and first episode or depressive relapse based on the Dysfunctional Attitudes Scale (DAS) (19).

The operationalization of cognitive reactivity offered by Van der Does (20), which actually followed Teasdale's formulation of cognitive reactivity, replaced the DAS, which was the "golden standard" until then. Using the Depression Sensitivity Index-Revised (LEIDS-R), respondents report how their typical behaviors and cognitions change while experiencing negative mood.

Research has shown that the LEIDS-R predicts the onset of first symptoms (21), and that it discriminates well between subjects with a prior history of a depressive episode from those without a prior history of depressive symptoms (22). The findings of Sokić, (22) suggest that general cognitive reactivity could be a good predictor of the first episode, but also that specific cognitive reactivity (eg, rumination, dysfunctional attitudes) are related to the recurrent course of the disorder. The results support the concept of cognitive reactivity at a more specific level, in the form of negative automatic thoughts and ruminations in response to current negative mood.

Numerous findings support the notion of vulnerability through the cognitive stress diathesis, yet empirical studies that directly examine whether it actually contributes to the onset, recurrence, or recurrent course of symptoms are rare. In one such study that examined cognitive reactivity, operationalized through Dysfunctional Attitudes Scales (DAS), to negative mood induction as a predictor of depressive relapse, Segal et al. (23) showed that the level of cognitive reactivity at baseline successfully predicted relapse 13-48 months later. These findings were replicated in a similar but methodologically more rigorous study (24).

Comparing whether dysfunctional beliefs (DAS) or the reactivity of such beliefs to mild dysphoric states (i.e., cognitive reactivity assessed with the LEIDS-R) represent a key factor predicting relapse, a group of researchers (25) showed that cognitive reactivity, especially the items related to rumination - predict depressive relapse in the long term. The main finding in the research conducted by Figueroa et al. (25) is that cognitive reactivity (CR) is a risk factor of depressive recurrence. They state that the current measurement of CR, by assessing change on the Dysfunctional Attitudes Scale (DAS) after mood-induction, is not reliable and that the Leiden

Index Depression Sensitivity-Revised (LEIDS-R) is an alternative CR measure. In contrast to mood-induction, it reliably assesses depression vulnerability.

From 2003 until today, a large number of researchers in Europe and other continents have translated and validated this instrument. For example, Senín-Calderón et al. (26) on a Spanish population extracted only 5 factors and subscales with satisfactory psychometric characteristics, similar to Solis et al. (27).

Himle et al. (28) on a Norwegian population confirmed the validity of 6 factor subscales, but also proposed a better psychometric model with only five subscales. Ostovar et al. (29) on a population from Iran confirmed the psychometric characteristics of the LEIDS-R, and 6 separate factors (subscales), with a satisfactory reliability coefficient. The Japanese version of the LEIDS-R was shown to have reasonable reliability and validity (30). The modified Chinese version of the Leiden Index of Depression Sensitivity (LEIDS-RR-CV) is a 26-item self-report measure of CR to sad mood, which contains 5 subscales, including hopelessness/suicidality, acceptance coping, aggression, control/perfectionism, and avoidant coping (31).

Participants with recurrent major depressive episodes showed more repetitive thoughts about negative issues and avoidance from internal and external aversive events when depressive mood was induced, compared to participants with only a single episode of depression. These results suggest that the characteristics of cognitive reactivity are important considerations for preventing relapse of depression. Currently, we are working on validation of LEIDS-R on non-clinical and clinical population in Republic of North Macedonia.

Conclusion and future directions

In recent years, a promising line of research has highlighted the role of CR in the development, maintenance, and relapse/recurrence of depressive symptoms or clinical depression (32). Thus, the relevance of CR to depression should be further explored. Our paper reveals enough evidence to support our hypothesis that cognitive reactivity to sad mood is a risk factor for depressive reccurence. There is also research evidence for the importance of the first depressive episode. While the contribution of cognitive reactivity to depressive relapses has been empirically investigated, little is still known about the origins and generation of this reactivity. The literature review confirmed the lack of standardization of methodologies applied in the research and their great diversity. Even though, DAS proves its good psychometric characteristics in many research studies, according to the psychometric characteristics of the Index for Depression sensitivity, LEIDS-R in various countries in Europe, and countries (Japanes, China, Iran etc.) from other continents, in which it has been translated and validated, we conclude that it is a promising test for measuring cognitive reactivity to sad mood (CR).

The results obtained from previous research indicate the importance of an interactive approach when examining the factors that contribute to the recurrent course of depression. Furthermore, the elaboration of the answer to these questions, through research that takes into account the mentioned aspects, would contribute to understanding the situational, dispositional, emotional and cognitive mechanisms within the framework of recurrent depressive symptoms. Additionaly, understanding the neural mechanism responsible for the biased processing of emotional stimuli in depression might bring important clinical benefits, including predicting, detecting and treating depression (33).

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Author contribution. Single author article

Kognitivna reaktivnost na tužno raspoloženje kao čimbenik rizika za ponovno pojavljivanje depresije

Sažetak

Prema kognitivnim teorijama depresije, ranjivost za nastanak i ponavljanje depresije leži u određenoj vrsti kognitivne disfunkcije ili neprilagođenoj obradi kognitivnih informacija. Ovaj pregledni rad ima za cilj razjasniti ulogu kognitivne reaktivnosti na tužno raspoloženje kao čimbenika rizika za ponovno pojavljivanje depresije, njezin mehanizam i mjerenje, kako bi se omogućilo prilagođeno liječenje i prevencija depresije. Provedena je sveobuhvatna pretraga baza podataka PsycInfo, PubMed i Web of Science. Teasdaleova hipoteza diferencijalne aktivacije sugerira da prvi depresivni epizoda uspostavlja specifične disfunkcionalne obrasce obrade informacija koji ostaju latentni nakon oporavka, ali se mogu ponovno aktivirati depresivnim raspoloženjem. Ta aktivacija dodatno jača disfunkcionalne obrasce obrade i tako stvara začarani krug ponavljanja depresije. Teasdale taj koncept naziva kognitivna reaktivnost.

Kognitivna reaktivnost (KR) definira se kao razina do koje pojedinac doživljava negativnu promjenu u kognitivnom sadržaju i procesima tijekom tužnog raspoloženja. Nalazi našeg pregleda sugeriraju da se KR može konceptualizirati kao čimbenik rizika prisutan kod ranjivih osoba prije pojave depresije, koji razlikuje ranjive od neranjivih skupina čak i u remisiji, te koji predviđa povratak depresije. Također postoje istraživački dokazi da je trajanje prve depresivne epizode, bez obzira na njezin intenzitet, od ključne važnosti za formiranje kognitivne reaktivnosti na tužno raspoloženje.

Na temelju rezultata iz različitih zemalja zaključujemo da je Indeks osjetljivosti na depresiju, LEIDS-R, obećavajući test za mjerenje kognitivne reaktivnosti na tužno raspoloženje (KR). Rezultati prethodnih istraživanja ukazuju na važnost interaktivnog pristupa pri ispitivanju čimbenika koji doprinose ponavljajućem tijeku depresije.