Heliyon 7 (2021) e07218

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon

Review article

A narrative review of emotion regulation process in stress and recovery phases

Pablo Ezequiel Flores-Kanter [a,b,*], Luciana Moretti [a,c], Leonardo Adrián Medrano [a,c]

[a] *Universidad Siglo 21, Argentina*
[b] *Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), Argentina*
[c] *Pontificia Universidad Católica Madre y Maestra, República Dominicana*

ARTICLE INFO

Keywords:
Emotion regulation
Stress
Review

ABSTRACT

The difficulty in studying the relationship between stress and emotional regulation is due to the need to contemplate a dynamic perspective that analyzes the moderating role of stress. In fact, stress involves different phases or stages, and the neurocognitive processes involved in emotion regulation differ significantly between these phases. The period of anticipation of stressful events can be fundamental to understand the process of stress regulation; however, surprisingly few works have analyzed the differential activation of brain networks involved in cognitive regulation during the phases of stress and recovery. Taking this into consideration, within this study we propose to analyze in an integrated way the psychological and neurobiological processes during the phase of stress and recovery, with the aim of improving our understanding of the mechanisms that underlie successful and unsuccessful stress regulation. We consider that from the present review we contribute to achieve a better understanding of the mechanisms underlying successful and unsuccessful stress regulation would contribute to the improvement of prevention and treatment interventions for mental disorders.

1. Introduction

We all experience stress at some point in our lives, which affects our mood, behavior and well-being. The inability to regulate daily stress is associated with the development of a wide range of psychological disorders such as major depression, substance abuse, and anxiety disorders (Pulopulos et al., 2020). Emotion regulation (ER) is the ability to influence (automatically or voluntarily) our emotions (including stress) in order to maintain our emotional balance and to achieve our goals (Aldao et al., 2015).

The relationship between stress and emotional regulation is complex and results from the interaction of biological, psychological and environmental factors (Gotlib et al., 2008; De Raedt and Koster, 2010). A better understanding of the underlying mechanisms of successful and unsuccessful stress regulation would contribute to improve the strategies to prevent and treat mental disorders (Nasso et al., 2019).

The difficulty in studying the relationship between stress and emotional regulation is due to the need to contemplate a dynamic perspective that analyzes the moderating role of stress. In fact, stress involves different phases or stages, and the neurocognitive processes involved in emotion regulation differ significantly between these phases. In general terms, the process of stress involves at least three phases: anticipation, confrontation with the stressor (stress) and recovery.

Only recently has the need to look at each phase differently been pointed out. In this line, Ottaviani (2018) and Nasso et al. (2019) state that the study of the neurocognitive processes involved during the period of anticipation of stressful events can be fundamental for understanding the process of stress regulation. In this framework, De Raedt and Hooley (2016) have proposed the Neurocognitive framework for Regulation Expectation (NFRE), a framework in which stress anticipation plays a central role in the process of stress regulation and the development of depression and other stress-related psychopathologies.

The results obtained indicate that activation of the dorsolateral prefrontal cortex (dlPFC) during the stress anticipation phase reduces the response of the HPA axis to stress through indirect and inhibitory connections with the amygdala (Nasso et al., 2019). These investigations have made possible the development of different psychological interventions aimed at improving the regulation of stress. For example, Salzmann et al. (2018) observed that during the anticipation phase, strategies focused on distraction and personal control decrease the physiological response to a stress task. Similarly, Nasso et al. (2019)

---

* Corresponding author.
E-mail addresses: ezequielfk@gmail.com, pablo.floreskanter@ues21.edu.ar, pablo.floreskanter@conicet.gov.ar (P.E. Flores-Kanter).

https://doi.org/10.1016/j.heliyon.2021.e07218
Received 18 November 2019; Received in revised form 13 March 2020; Accepted 2 June 2021

observed that the use of cognitive reappraisal during anticipation of stress results in a muffled response to stress.

The period of anticipation of stressful events can be fundamental to understand the process of stress regulation; however, surprisingly few works have analyzed the differential activation of brain networks involved in cognitive regulation during the phases of stress and recovery (Figure 1). Taking this into consideration, within this study we propose to analyze in an integrated way the psychological and neurobiological processes during the phase of stress and recovery, with the aim of improving our understanding of the mechanisms that underlie successful and unsuccessful stress regulation.

2. Balance, stress and recovery

Stress can be defined as an uncomfortable emotional experience accompanied by biochemical, physiological, and behavioral changes that occur during difficult or demanding situations (Nasso et al., 2020). Chronic stress can have a negative impact on our health and contribute to the appearance of mental disorders (Chaby et al., 2015; Gomez-Bernal et al., 2019). However, not all people facing stressful circumstances develop psychopathology. Adaptive emotion regulation during periods of post-stress recovery is key to overcoming aversive emotional experiences and gaining a sense of control after exposure to stress (Miklósi et al., 2014).

Adaptive recovery from acute and chronic stressors is crucial to maintain healthy cognitive and affective functioning (Murray et al., 2021). Post-stress recovery periods allow for a return to homeostatic physiological states, through allostatic processes (Karatsoreos and McEwen, 2011). The inability to employ appropriate emotional regulation strategies during this stage can lead to prolonged negative moods and persistent states of arousal, increasing the likelihood of developing mental disorders such as depression and anxiety (Disner et al., 2011; Jordan et al., 2018; Martin and Dahlen, 2005).

Studies in affective neuroscience have recently begun to provide clearer neuroanatomical and neurofunctional data on the brain processes involved in responses to stressful events (Tobia et al., 2017). Based on these approaches it is proposed to differentiate between brain functioning in stress and recovery situations (see Figure 2; Bornas et al., 2013; Flores-Kanter 2020; Flores-Kanter and Medrano, 2020; McNaughton, 2019; Tobia et al., 2017).

3. Differential activation of brain networks involved in emotion regulation in stress situations

With regard to the brain regions involved in emotion regulation and stress response, two differential processes are activated: bottom-up and top-down regulation processes (Beauchaine and Zisner, 2017; Belden et al., 2014; Phillips et al., 2008). Bottom-up processes are usually referred to as automatic or reactive, since they involve cognitive responses automatically induced by an emotional stimulus generated or triggered in subcortical structures. Top-down processes, on the other hand, involve a deliberate and reflexive cognitive effort and take place in higher cortex structures. Evidence so far suggests that bottom-up and top-down responses are distinct processes, activate different cognitive-emotion regulation (CERs), and interact bi-directionally with each other (Beauchaine and Zisner, 2017; Belden et al., 2014; Park et al., 2018; Whittle et al., 2006).

The ascending signals of the limbic subcortical structures (e.g. hypothalamus and amygdala) influence the activity of the upper cortex structures through their connections to the medial prefrontal cortex (mPFC; Weis et al., 2019). It is this activation that triggers self-referential repetitive negative thinking (RNT: perseverative, stable, general, self-focused, negative ways of thinking; Epel et al., 2018; Hervas and Vazquez, 2011; Luca, 2019; Paulus, 2015) and is the basis for prolonged processing of negative information in working memory (WM) (Stout et al., 2018; Whittle et al., 2006) This two-way connection between limbic structures such as the amygdala and the mPFC results in the intensification and prolongation of the negative emotional response in its affective and expressive components (Park et al., 2019; Zald, 2003) and involves two regions of the brain: a) the connections of the amygdala with the hippocampus and the caudate and putamen nucleus, which explain the bias towards the negative components of events produced in memory (both in the formation and in the recovery of memories; see Stout et al., 2018); and b) the activation of another upper cortical structure, the right dorsolateral prefrontal cortex (right dlPFC), which plays a central role in the anticipation of negative emotional stimulus, directing attention and cognitive resources towards it (Park et al., 2019; Siegle et al., 2003; Whittle et al., 2006).

Emotional responses produced through the ascending signals of the limbic subcortical structures are modulated by the intensification of negative affect on superior cortex structures (Park et al., 2019; Whittle

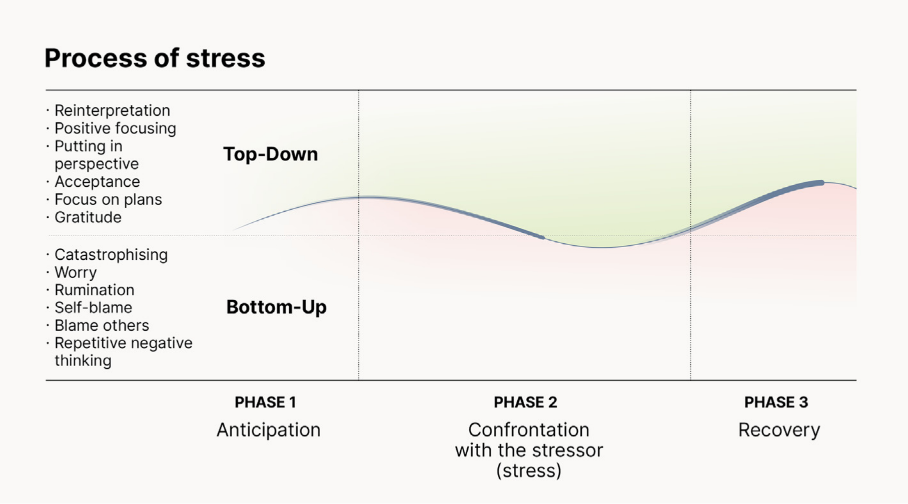

Figure 1. Process of Stress and Emotion Regulation. *Note.* Schematic representation of the dynamic relationship between the process of stress and cognitive emotion regulation responses more characteristic in each phase.

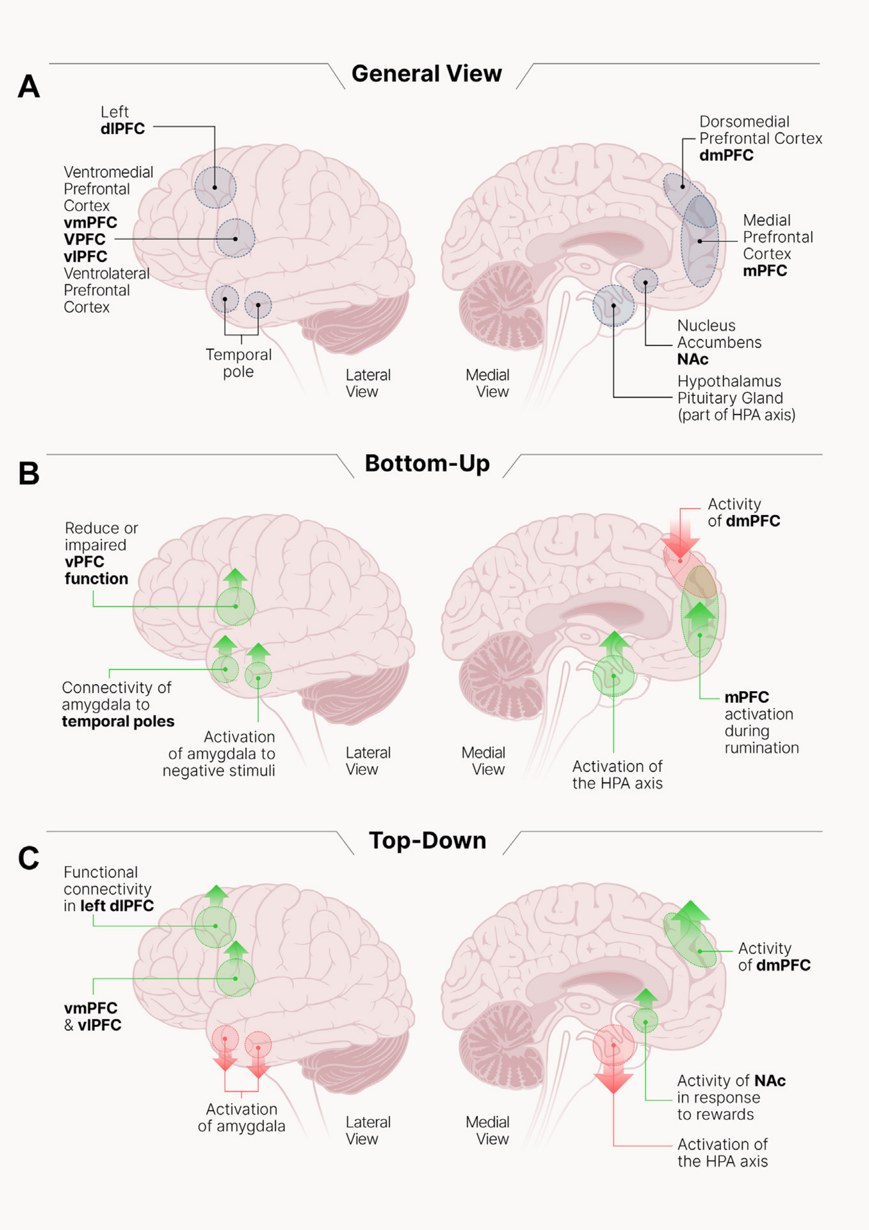

Figure 2. Schematic representation of brain structures implicated in emotion generation and regulation. *Note.* Schematic representation of subcortical structures (e.g. amygdala) implicated in emotion generation and cortical regions implicated in emotion regulation. Panel A (top side of the figure) = General structures view; Panel B (middle part of the figure) = Bottom-up structures; Panel C (lower side of the figure) = Top-down view. mPFC = Medial Prefrontal Cortex; vPFC = Ventral Prefrontal Cortex; vlPFC = Ventrolateral Prefrontal Cortex; vmPFC = Ventromedial Prefrontal Cortex; dmPFC = Dorsomedial Prefrontal Cortex; NAc = Nucleus Accumbens.

et al., 2006) that trigger the RNT (Crosswell et al., 2019; Epel et al., 2018; Garland et al., 2010; Koval et al., 2012; Taylor and Liberzon, 2007). Based on both neurobiological models (Stout et al., 2018) and neuropsychological approaches (Coifman et al., 2019) it has been proposed that the effect produced by the RNT (e.g., worry and rumination) is due to the difficulty of filtering all the negative information that constantly occupies the WM, due to its threat-related component, which requires recruiting resources from other higher structures of cognitive control.

In the case of downstream processes, one of the structures involved is the orbital frontal cortex (OFC), which is linked to the processing of information and the modulation of behavior in the face of positive reinforcement stimuli (Whittle et al., 2006). Descending ER signals also involve areas of the prefrontal cortex, such as the left dorsolateral prefrontal cortex (left dlPFC), which is associated with the reorientation of attention towards the negative event (see Stout et al., 2018, on the relationship of these regions with working memory). Together with the anterior cingulate cortex (ACC), these structures have been linked to the regulation of the positive affect generated by mesolimbic structures, including the nucleus accumbens (NAc) (Beauchaine and Zisner, 2017). A reciprocal functional relationship between the dorsal and medial regions of the PFC has been proposed, from which a process of affect regulation is also generated (Taylor and Liberzon, 2007).

Other structures that play a central role in the modulation of subcortical limbic responses - more associated with downward regulation of the negative affect - are the ventrolateral prefrontal cortex (vlPFC) and the ventromedial prefrontal cortex (vmPFC). The vlPFC serves as an association cortex, integrating complex internal and external sensory signals to generate cognitive judgments linked to priority and goal setting (e.g., positive reinterpretation of the negative valence stimulus, and refocusing on planning). This information is then sent to the vmPFC, which acts as a visceromotor cortex that modulates the response of subcortical limbic structures such as the amygdala. Through the interaction of these regulatory systems, the body can influence and attenuate the affective and expressive components of the emotional response (Garland et al., 2010; Park et al., 2019; Peverill et al., 2019).

The dorsomedial prefrontal cortex (dmPFC) is also relevant for the regulation of emotions from top to bottom. Abnormalities in the functioning of this structure are associated with difficulties in maintaining separate emotional state and RNT (Park et al., 2019). Finally, the temporal lobes also allow the modulation of the emotional response as a function of context, assessing emotional significance by integrating complex environmental stimuli (as opposed to the amygdala, which makes a "crude and rapid" assessment of emotional significance). Its function is similar to that of the vlPFC, serving as an association cortex to generate the cognitions that modulate the emotional response of the amygdala (Park et al., 2019).

4. Differential activation of brain networks involved in emotional regulation during recovery

Post-stress recovery periods are key in preventing problems associated with chronic stress. For this reason, an adequate understanding of the stress process implies considering both: exposure to stress and recovery time (Gormally et al., 2019). Recently, we have begun to explore the factors involved in recovery from stress (Gormally et al., 2019). Studies conducted in animal models indicate that increases in the intensity and duration of stress compromised the recovery of the HPA axis and the immune system (García et al., 2000; Sarjan and Yajurvedi, 2018). It has also been observed that chronic stress-induced neuronal atrophy of the hippocampus can be reduced after periods of recovery (Ortiz and Conrad, 2018).

Efficacy in stress recovery will depend largely on the cognitive regulation processes involved. The predominance of top-down processes is related to better recovery from stress. The prefrontal control decreases amygdala activation, which is associated with the experience of negative emotions, via the cortical-subcortical pathway (Wager et al., 2008). Several studies associated the use of reappraisal with prefrontal activation (Dillon and Pizzagalli, 2013; Nelson et al., 2015; Uchida et al., 2015; Urry et al., 2009; Vanderhasselt et al., 2013). For example, it is observed that re-evaluation predicts better recovery from cardiovascular stress, as measured by heart rate variability (Jentsch and Wolf, 2020).

On the contrary, the predominance of bottom-up processes during the recovery phase has a negative influence. It has been observed, for example, that after exposure to negative stimuli, rumination is associated with altered cardiac stress recovery, measured through blood pressure (Glynn et al., 2002; Radstaak et al., 2011), and altered cortical surface stress recovery, measured through fNIRS (Rosenbaum et al., 2018).

Recently, Murray et al. (2021) developed an experimental study to determine whether the use of top-down and bottom-up cognitive strategies correlate with recovery levels. Thus, it was hypothesized that participants who appeal to the use of top-down strategies such as reinterpretation, positive focusing, putting in perspective, and acceptance will have better recovery than participants who use bottom-up strategies such as rumination or self-incrimination. In this work, for the first time, empirical evidence was obtained indicating an increase in activity in both the ventral (e.g., hippocampus) and dorsal regions (e.g., dACC, pgACC). This means that increased neuronal activity in ventral regions signals to the dorsal regions to improve neuronal functioning, thus moderating the excitatory affective response of the respective ventral regions (e.g. Bogdan et al., 2015; Del Río-Casanova et al., 2016; Phillips et al., 2008). The increased dorsal and ventral activity suggests that the use of functional strategies of emotion regulation requires greater volitional executive control (dorsal) rather than a preponderant automatic affective response (ventral).

5. Discussion

The inability to regulate stress is associated with the development of a wide variety of psychological disorders and adverse long-term health problems. In this sense, different psychopathological models have proposed chronic stress as a trigger for various disorders (Taylor et al., 2019). For this reason, clarifying the mechanisms of emotion regulation involved during the process of stress is of great transdiagnostic value (Tobia et al., 2017).

The relationship between stress and emotion regulation is very complex and results from an interaction of biological, psychological and environmental factors (Gotlib et al., 2008; De Raedt and Koster, 2010). The difficulty in studying the relationship between stress and emotion regulation is due to the need to contemplate a dynamic perspective that analyzes the moderating role of stress. In fact, stress involves different phases or stages, and the neurocognitive processes involved in emotion regulation differ significantly between these phases.

The research findings analyzed in the present review allow us to make two statements: a) during cognitive stress regulation, two main and bidirectionally associated brain processes can be differentiated, and b) these brain systems function in a differential way in different phases of stress. In this way, the interaction of both systems during the different phases of stress would be the factor that explains an adequate or inadequate stress regulation.

The processes involved in stress cognitive and emotion regulation could be differentiated into two main systems. System 1 (bottom-up) characterized by being automatic and triggered reactively to a stressful stimulus, and system 2 (top-down) which involves more complex cognitive processes and implies a subsequent deliberative effort. Based on the interaction of both systems, the resulting cognitive regulation can generate a decrease or intensification of the stress response. This differentiation between an automatic-reactive and an elaborating-active system is consistent with several theoretical models, although with different names (e.g. Kahneman, 2011).

Although the main brain structures involved in both systems are known, the interaction between them and the factors that lead to the predominance of one over the other still requires further study. In this line, the concept of Temporal Dynamics of Emotions and Stress and the Dynamic Functional Connectivity approach has been proposed (Dosenbach et al., 2008; Peverill et al., 2019; Tobia et al., 2017; Zald, 2003). Specifically, it is postulated that emotions and components of the stress response give rise to different neuronal dynamics, reconfigured over time as a function of internal regulatory factors of the body, including cognitive states (Cohen et al., 2016). In this way, during an adverse or stressful situation different and neuro-dynamically independent brain networks are activated, linked firstly to negative and positive affect and secondly to the regulatory processes of these primary affective responses (Hofmann et al., 2012; McNaughton, 2019). This last functional system of brain networks does not act as an independent factor (unlike affects), but rather interacts with the systems of negative and positive affect (NA and PA) in the regulation and control of affect and behavior (Whittle et al., 2006). This means that: a) networks linked to affect generation can make a unique and simultaneous contribution in situations of stress (Park et al., 2019); and b) it is the functional connection between these structures, rather than their separate and specialized function, that explains the differences observed in affective response and affect regulation, depending on the content of the stimuli (e.g., stressful vs. benign-safe) (Anand et al., 2019; Lu et al., 2017; Whittle et al., 2006).

A dynamic factor that has not been clearly contemplated is that which refers to stress phases. In general terms the process of stress involves at least three phases: anticipation, confrontation with the stressor (stress) and recovery. During each one of these phases the neurocognitive processes involved in emotional regulation differ notably, depending on the phase and level of stress response the predominance of one system over another can be affected. For this reason it is important to develop dynamic models that contemplate the moderating role of stress on emotion regulation.

In previous studies it was analyzed the role of neurocognitive processes involved during the anticipation period of stressful events (Ottaviani, 2018; Nasso et al., 2019). In this study we focus on the analysis of the cognitive and neuronal mechanisms involved during the stress and recovery situation. In general terms, it can be affirmed that during the stress experience it is observed a primacy of bottom-up processes where ascending signals from limbic subcortical structures (e.g. hypothalamus and amygdala) influence the activity of higher cortex structures through their connections to the medial prefrontal cortex (mPFC; Weis et al., 2019). In contrast, the prefrontal (top-down) control decreases amygdala activation, which is associated with the experience of negative emotions, via the cortical-subcortical pathway (Wager et al., 2008). Several studies associated the use of reappraisal with prefrontal activation (Dillon and Pizzagalli, 2013; Nelson et al., 2015; Uchida et al., 2015; Urry et al., 2009; Vanderhasselt et al., 2013). A better understanding of the mechanisms underlying successful and unsuccessful stress regulation would contribute to the improvement of prevention and treatment interventions for mental disorders (Nasso et al., 2019). This theoretical model is of clinical relevance as it would help identifying which different aspects of stress resilience should be targeted by therapeutic interventions and how to better tailor such interventions.

In this sense, it is well established that cognitive behavior therapy (CBT) is efficacious in the treatment of stress and emotional disorders. Despite this, pooled meta-analytic response rates for CBT varies between 38% and 82% depending on the specific disorder (Hofmann et al., 2012). Thus, there is still a substantial room for improvement. Even when a treatment is found to be effective, it is unlikely to be effective for everybody. Tailoring treatments to individual characteristics is one of the aims of so-called personalized (or precision) medicine, a concept which has received increased attention over the last years in mental health field (Simon and Perlis, 2010).

The present review is a step forward in this sense, since it would allow a clearer and more precise precision when performing interventions on patients with stress regulation difficulties. In fact, two patients may have problems in regulating stress response for different reasons. In one case, difficulties could be observed during the stress phase, and in another case, during the recovery phase. Both patients would therefore present similar symptoms, but the treatment would require different interventions. For example, training in distraction techniques may be useful during the stress recovery phase, but counterproductive during coping with stress situation (Salzmann et al., 2018). Similarly, interventions focused on reinterpretation during the stress phase have been observed to reduce the HPA axis response and inhibit activation of the amygdala (Nasso et al., 2019), but the use of interventions focused on gratitude and compassion are not as effective during this phase (Salzmann et al., 2018), although they would likely work better for recovery strategy.

It is unwarranted to assume that the same strategy will be equally effective at different phases of the stress process. Thus, during the anticipation or coping with a stressful situation, focusing on plans will be a more functional strategy than positive focusing, while during the recovery phase the opposite process would be observed (Medrano et al., 2013). An adequate training in the use of emotion regulation strategies involves analyzing during which phases of the stress process the most difficulties are observed. We consider that from the present review we contribute to achieve a better understanding of the mechanisms underlying successful and unsuccessful stress regulation would contribute to the improvement of prevention and treatment interventions for mental disorders.

Thus, practical principles are derived from the present review. First, the need to consider dynamic factors when examining the role of the cognitive processes involved in emotional regulation is emphasized. Thus, rather than analyzing whether the use of a given emotional regulation strategy is functional or not, the moment in which it is used should be considered. Given that the functionality of a strategy will be largely determined by the "moment" in which it is applied, the second principle derived is that clinical psychologists should train patients not only to learn the strategy (what do I do?), but also to be able to apply it at the right moment (when do I do it?). For example, several studies indicate that mindfulness training is an appropriate strategy to reduce stress (Mak et al., 2018). This may be because it is an appropriate strategy to recover from stress, or to avoid the use of dysfunctional strategies in the anticipation phase. However, it may be an unhelpful strategy during the stress coping phase. Therefore, training a patient in mindfulness without an adequate explanation of when to use such a strategy may be counterproductive. The same happens with the training of other emotional regulation strategies, such as cognitive reinterpretation, positive focus or localization in plans, for example. Thus, the main contribution of the present work consists in highlighting the need to train patients in the "dynamic" use of emotional regulation strategies. Knowing "what" to do to regulate stress and knowing "when" to do it.

Declarations

Author contribution statement

All authors listed have significantly contributed to the development and the writing of this article.

Funding statement

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability statement

No data was used for the research described in the article.

Declaration of interests statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

References

Aldao, A., Sheppes, G., Gross, J.J., 2015. Emotion regulation flexibility. Cognit. Ther. Res. 39 (3), 263–278.

Anand, A., Jones, S.E., Lowe, M., Karne, H., Koirala, P., 2019. Resting state functional connectivity of dorsal raphe nucleus and ventral tegmental area in medication-free young adults with major depression. Front. Psychiatr. 9, 765.

Beauchaine, T.P., Zisner, A., 2017. Motivation, emotion regulation, and the latent structure of psychopathology: an integrative and convergent historical perspective. Int. J. Psychophysiol. 119, 108–118.

Belden, A.C., Luby, J.L., Pagliaccio, D., Barch, D.M., 2014. Neural activation associated with the cognitive emotion regulation of sadness in healthy children. Dev. Cogn. Neurosci. 9, 136–147.

Bogdan, R., Pagiaccio, D., Baranger, D.A.A., Hariri, A.R., 2015. Genetic moderation of stress effects on corticolimbic circuitry. Neuropsychopharmacology 41, 275–296.

Bornas, X., Tortella-Feliu, M., Balle, M., Llabrés, J., 2013. Self-focused cognitive emotion regulation style as associated with widespread diminished EEG fractal dimension. Int. J. Psychol. 48 (4), 695–703.

Chaby, L.E., Cavigelli, S.A., Hirrlinger, A.M., Caruso, M.J., Braithwaite, V.A., 2015. Chronic unpredictable stress during adolescence causes long-term anxiety. Behav. Brain Res. 278, 492–495.

Cohen, S., Gianaros, P.J., Manuck, S.B., 2016. A stage model of stress and disease. Perspect. Psychol. Sci. 11 (4), 456–463.

Coifman, K.G., Kane, M.J., Bishop, M., Matt, L.M., Nylocks, K.M., Aurora, P., 2019, March 18. Predicting negative affect variability and spontaneous emotion regulation: can working memory span tasks estimate emotion regulatory capacity? Emotion. Advance online publication.

Crosswell, A.D., Coccia, M., Epel, E.S., 2019. Mind wandering and stress: when you don`t like the present moment. Emotion.

De Raedt, R., Hooley, J.M., 2016. The role of expectancy and proactive control in stress regulation: a neurocognitive framework for regulation expectation. Clin. Psychol. Rev. 45, 45–55.

De Raedt, R., Koster, E.H., 2010. Understanding vulnerability for depression from a cognitive neuroscience perspective: a reappraisal of attentional factors and a new conceptual framework. Cognit. Affect Behav. Neurosci. 10 (1), 50–70.

Del Río-Casanova, L., González, A., Páramo, M., Van Dijke, A., Brenlla, J., 2016. Emotion regulation strategies in trauma-related disorders: pathways linking neurobiology and clinical manifestations. Rev. Neurosci. 27 (4), 385–395.

Dillon, D.G., Pizzagalli, D.A., 2013. Evidence of successful modulation of brain activation and subjective experience during reappraisal of negative emotion in unmedicated depression. Psychiatr. Res. 212 (2), 99–107.

Disner, S.G., Beevers, C.G., Haigh, E.A., Beck, A.T., 2011. Neural mechanisms of the cognitive model of depression. Nat. Rev. Neurosci. 12 (8), 467–477.

Dosenbach, N.U.F., Fair, D.A., Cohen, A.L., Schlaggar, B.L., Petersen, S.E., 2008. A dual-networks architecture of top-down control. Trends Cognit. Sci. 12 (3), 99–105.

Epel, E.S., Crosswell, A.D., Mayer, S.E., Prather, A.A., Slavich, G.M., Puterman, E., Mendes, W.B., 2018. More than a feeling: a unified view of stress measurement for population science. Front. Neuroendocrinol. 49, 146–169.

Flores-Kanter, P.E., 2020. Commentary: how we know what not to think. Front. Psychol. 11.

Flores-Kanter, P.E., Medrano, L.A., 2020. Commentary: putting 'emotional intelligences' in their place: introducing the integrated model of affect-related individual differences. Front. Psychol. 11.

García, A., Martí, O., Vallès, A., Dal-Zotto, S., Armario, A., 2000. Recovery of the hypothalamic-pituitary-adrenal response to stress. Effect of stress intensity, stress duration and previous stress exposure. Neuroendocrinology 72 (2), 114–125.

Garland, E.L., Fredrickson, B., Kring, A.M., Johnson, D.P., Meyer, P.S., Penn, D.L., 2010. Upward spirals of positive emotions counter downward spirals of negativity: insights from the broaden-and-build theory and affective neuroscience on the treatment of emotion dysfunctions and deficits in psychopathology. Clin. Psychol. Rev. 30 (7), 849–864.

Glynn, L.M., Christenfeld, N., Gerin, W., 2002. The role of rumination in recovery from reactivity: cardiovascular consequences of emotional states. Psychosom. Med. 64 (5), 714–726.

Gomez-Bernal, F., Madva, E.N., Puckett, J., Amonoo, H.L., Millstein, R.A., Huffman, J.C., 2019. Relationships between life stressors, health behaviors, and chronic medical conditions in mid-life adults: a narrative review. Psychosomatics 60 (2), 153–163.

Gormally, B., Ramos, S., Yin, H., Romero, L.M., 2019. Recovery periods during repeated stress impact corticosterone and behavioral responses differently in house sparrows. Horm. Behav. 112, 81–88.

Gotlib, I.H., Joormann, J., Minor, K.L., Hallmayer, J., 2008. HPA axis reactivity: a mechanism underlying the associations among 5-HTTLPR, stress, and depression. Biol. Psychiatr. 63 (9), 847–851.

Hervas, G., Vazquez, C., 2011. What else do you feel when you feel sad? Emotional overproduction, neuroticism and rumination. Emotion 11 (4), 881–895.

Hofmann, S.G., Sawyer, A.T., Fang, A., Asnaani, A., 2012. Emotion dysregulation model of mood and anxiety disorders. Depress. Anxiety 29 (5), 409–416.

Jentsch, V.L., Wolf, O.T., 2020. The impact of emotion regulation on cardiovascular, neuroendocrine and psychological stress responses. Biol. Psychol. 154, 107893.

Jordan, C., Ohlsen, R., Hayee, B., Chalder, T., 2018. A qualitative study exploring the experience of people with IBD and elevated symptoms of anxiety and low mood and the type of psychological help they would like. Psychol. Health 33 (5), 634–651.

Karatsoreos, I.N., McEwen, B.S., 2011. Psychobiological allostasis: resistance, resilience and vulnerability. Trends Cognit. Sci. 15 (12), 576–584.

Kahneman, D., 2011. Thinking, Fast and Slow. Farrar, Straus and Giroux, New York.

Koval, P., Kuppens, P., Allen, N.B., Sheeber, L., 2012. Getting stuck in depression: the roles of rumination and emotional inertia. Cognit. Emot. 26 (8), 1412–1427.

Lu, Y., Shen, Z., Cheng, Y., Yang, H., He, B., Xie, Y., et al., 2017. Alternations of white matter structural networks in first episode untreated major depressive disorder with short duration. Front. Psychiatr. 8.

Luca, M., 2019. Maladaptive rumination as a transdiagnostic mediator of vulnerability and outcome in psychopathology. J. Clin. Med. 8 (3), 314.

Mak, C., Whittingham, K., Cunnington, R., Boyd, R.N., 2018. Efficacy of mindfulness-based interventions for attention and executive function in children and adolescents—a systematic review. Mindfulness 9 (1), 59–78.

Martin, R.C., Dahlen, E.R., 2005. Cognitive emotion regulation in the prediction of depression, anxiety, stress, and anger. Pers. Indiv. Differ. 39 (7), 1249–1260.

McNaughton, N., 2019. Brain maps of fear and anxiety. Nat. Human Beh. 3 (7), 662–663.

Medrano, L.A., Moretti, L.S., Ortiz, A., Pereno, G., 2013. Validación del Cuestionario de Regulación Emocional Cognitiva en Universitarios de Córdoba, Argentina. Psykhe 22 (1).

Miklósi, M., Martos, T., Szabó, M., Kocsis-Bogár, K., Perczel Forintos, D., 2014. Cognitive emotion regulation and stress: a multiple mediation approach. Transl. Neurosci. 5 (1), 64–71.

Murray, R.J., Apazoglou, K., Celen, Z., Dayer, A., Aubry, J.M., Van De Ville, D., et al., 2021. Maladaptive emotion regulation traits predict altered corticolimbic recovery from psychosocial stress. J. Affect. Disord. 280, 54–63.

Nasso, S., Vanderhasselt, M.A., Demeyer, I., De Raedt, R., 2019. Autonomic regulation in response to stress: the influence of anticipatory emotion regulation strategies and trait rumination. Emotion 19 (3), 443.

Nasso, S., Vanderhasselt, M.A., Schettino, A., De Raedt, R., 2020. The role of cognitive reappraisal and expectations in dealing with social feedback. Emotion. Advance online publication.

Nelson, B.D., Fitzgerald, D.A., Klumpp, H., Shankman, S.A., Phan, K.L., 2015. Prefrontal engagement by cognitive reappraisal of negative faces. Behav. Brain Res. 279, 218–225.

Ortiz, J.B., Conrad, C.D., 2018. The impact from the aftermath of chronic stress on hippocampal structure and function: is there a recovery? Front. Neuroendocrinol. 49, 114–123.

Ottaviani, C., 2018. Brain-heart interaction in perseverative cognition. Psychophysiology 55 (7), e13082.

Park, A.T., Leonard, J.A., Saxler, P.K., Cyr, A.B., Gabrieli, J.D.E., Mackey, A.P., 2018. Amygdala–medial prefrontal cortex connectivity relates to stress and mental health in early childhood. Soc. Cognit. Affect Neurosci. 13 (4), 430–439.

Park, C., Rosenblat, J.D., Lee, Y., Pan, Z., Cao, B., Iacobucci, M., McIntyre, R.S., 2019. The neural systems of emotion regulation and abnormalities in major depressive disorder. Behav. Brain Res. 367, 181–188.

Paulus, M.P., 2015. Cognitive control in depression and anxiety: out of control? Curr. Opin. Beh. Sci. 1, 113–120.

Peverill, M., Sheridan, M.A., Busso, D.S., McLaughlin, K.A., 2019. Atypical prefrontal–amygdala circuitry following childhood exposure to abuse: links with adolescent psychopathology. Child. Maltreat.

Phillips, M.L., Ladouceur, C.D., Drevets, W.C., 2008. A neural model of voluntary and automatic emotion regulation: implications for understanding the pathophysiology and neurodevelopment of bipolar disorder. Mol. Psychiatr. 13 (9), 833–857.

Pulopulos, M.M., Baeken, C., de Raedt, R., 2020. Cortisol response to stress: the role of expectancy and anticipatory stress regulation. Horm. Behav. 117.

Radstaak, M., Geurts, S.A., Brosschot, J.F., Cillessen, A.H., Kompier, M.A., 2011. The role of affect and rumination in cardiovascular recovery from stress. Int. J. Psychophysiol. : Offic. J. Int. Org. Psychophysiol. 81 (3), 237–244.

Rosenbaum, D., Hilsendegen, P., Thomas, M., Haeussinger, F.B., Nuerk, H.C., Fallgatter, A.J., Nieratschker, V., Ehlis, A.C., Metzger, F.G., 2018. Disrupted prefrontal functional connectivity during post-stress adaption in high ruminators. Sci. Rep. 8 (1), 15588.

Salzmann, S., Euteneuer, F., Strahler, J., Laferton, J., Nater, U.M., Rief, W., 2018. Optimizing expectations and distraction leads to lower cortisol levels after acute stress. Psychoneuroendocrinology 88, 144–152.

Sarjan, H.N., Yajurvedi, H.N., 2018. Chronic stress induced duration dependent alterations in immune system and their reversibility in rats. Immunol. Lett. 197, 31–43.

Siegle, G.J., Steinhauer, S.R., Carter, C.S., Ramel, W., Thase, M.E., 2003. Do the seconds turn into hours? Relationships between sustained pupil dilation in response to emotional information and self-reported rumination. Cognit. Ther. Res. 27 (3), 365–382.

Simon, G.E., Perlis, R.H., 2010. Personalized medicine for depression: can we match patients with treatments? Am. J. Psychiatr. 167 (12), 1445–1455.

Stout, D.M., Bomyea, J., Risbrough, V.B., Simmons, A.N., 2018. Aversive distractors modulate affective working memory in frontoparietal regions. Emotion.

Taylor, S.F., Grove, T.B., Ellingrod, V.L., Tso, I.F., 2019. The fragile brain: stress vulnerability, negative affect and GABAergic neurocircuits in psychosis. Schizophr. Bull. 45 (6), 1170–1183.

Taylor, S.F., Liberzon, I., 2007. Neural correlates of emotion regulation in psychopathology. Trends Cognit. Sci. 11 (10), 413–418.

Tobia, M.J., Hayashi, K., Ballard, G., Gotlib, I.H., Waugh, C.E., 2017. Dynamic functional connectivity and individual differences in emotions during social stress. Hum. Brain Mapp. 38 (12), 6185–6205.

Uchida, M., Biederman, J., Gabrieli, J.D., Micco, J., de Los Angeles, C., Brown, A., Kenworthy, T., Kagan, E., Whitfield-Gabrieli, S., 2015. Emotion regulation ability varies in relation to intrinsic functional brain architecture. Soc. Cognit. Affect Neurosci. 10 (12), 1738–1748.

Urry, H.L., van Reekum, C.M., Johnstone, T., Davidson, R.J., 2009. Individual differences in some (but not all) medial prefrontal regions reflect cognitive demand while regulating unpleasant emotion. NeuroImage 47 (3), 852–863.

Vanderhasselt, M.A., Baeken, C., Van Schuerbeek, P., Luypaert, R., De Mey, J., De Raedt, R., 2013. How brooding minds inhibit negative material: an event-related fMRI study. Brain Cognit. 81 (3), 352–359.

Wager, T.D., Davidson, M.L., Hughes, B.L., Lindquist, M.A., Ochsner, K.N., 2008. Prefrontal-subcortical pathways mediating successful emotion regulation. Neuron 59 (6), 1037–1050.

Whittle, S., Allen, N.B., Lubman, D.I., Yücel, M., 2006. The neurobiological basis of temperament: towards a better understanding of psychopathology. Neurosci. Biobehav. Rev. 30 (4), 511–525.

Weis, C., Huggins, A.A., Bennett, K.P., Parisi, E.A., Larson, C.L., 2019. High resolution resting state functional connectivity of the extended amygdala. Brain Connect.

Zald, D.H., 2003. The human amygdala and the emotional evaluation of sensory stimuli. Brain Res. Rev. 41 (1), 88–123.