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# Adaptive and maladaptive emotion processing and regulation, and the case of alexithymia

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**ABSTRACT**

In this conceptual review, we discuss models of emotion and its regulation and identify a spectrum of processes that characterise adaptive adjustment to the affective environment. We describe a *dynamic-phasic model* of emotion processing and regulation, focusing on five stages: *anticipation, response, recovery, habituation and rest* as part of a cascade of responses to emotional challenges, as these become progressively expected, proximal, chronic or repeated. We argue for the need to investigate beyond simple reactivity to emotional stimuli, in order to understand mental and physical health conditions where emotional dysregulation plays a role. We propose that a hallmark of an effective and adaptive emotion regulation system is its flexibility, in the service of life goals and values. Consistent with McEwen's model (1998, *Stress, adaptation, and disease: Allostasis and allostatic load*. *Annals of the New York Academy of Sciences*, *840*(1), 33–44), inflexible emotion regulation can lead to increased allostatic load, from frequent stress, inadequate reactivity, failed shutdown and habituation, which may result in physical and mental illness. Alexithymia exemplifies inflexible emotion regulation, with dysfunctions potentially across all stages of emotion processing, both psychologically and physiologically. These maladaptive processes and their consequence on allostatic load potentially explain the association between alexithymia and physical and mental illness.

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Alexithymia involves difficulties in identifying, differentiating and describing emotions, and externally oriented thinking and has been linked to physical and mental health problems. Research examining emotional dysregulation in alexithymia has focused on subjective and physiological *reactivity* during emotional challenges, as the many studies on this topic, reviewed by Panayiotou, Panteli, and Vlemincx (2018b), show. However, emotion processing and regulation entail a wider spectrum of psychological and physiological processes. In this paper, we examine how these processes may explain the link between alexithymia and poor mental and physical health.

We draw from theories of emotion (Lang, 1979; Watson & Tellegen, 1985), emotion processing (Baumann, Kaschel, & Kuhl, 2007; Davidson, Jackson, & Kalin, 2000; Fanselow, 1994; Skinner & Zimmer-Gembeck, 2007) and regulation (Aldao, Sheppes, & Gross, 2015; Kashdan & Rottenberg, 2010; Koole, 2009), to argue that to understand alexithymia (and other conditions involving emotion difficulties) the full range of emotional processes must be considered.[1] We discuss a dynamic-phasic perspective, addressing (dys)regulation during five essential phases: *anticipation* of emotion stimuli, *reactivity* to these, *recovery* after a stimulus is no longer present, *habituation* to repeated stimuli, and *rest* in the absence of emotional events.

Within this framework, we focus on flexibility as a premise for adaptive emotion regulation.[2] Flexible emotion regulation allows these five phases to unfold fluidly, in a manner consistent with goals, needs and values, synchronised with situational

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demands, promoting physical and psychological health. Behaviourally, this constitutes psychological flexibility, i.e. *the capacity to persist with and change behavior in a manner that incorporates conscious and open contact with thoughts and feelings, and that is consistent with one's values and goals* (Scott, McCracken, & Norton, 2015, p. 286). Physiologically, it means adjusting autonomic, endocrine and neuromuscular responses to match situational demands and accomplish these goals.

To the contrary, inflexible regulation disrupts adaptive processes through rigid use of strategies to modulate psychological and physiological responses irrespective of the situation, one's goals and values. For example, strategies that down-regulate negative affect, like avoidance (or poor emotional awareness, as in alexithymia), bring relief from stressors in the short-run (Davydov, 2017; Davydov, Stewart, Ritchie, & Chaudieu, 2010). However, when used inflexibly, in the long-run, they predict psychological and physical dysfunction (Stabbe, Rolffs, & Rogge, 2019; Tavakoli, Broyles, Reid, Sandoval, & Correa-Fernández, 2019), through progressive disengagement from meaningful and reinforcing activities, goals, needs and values (Scott & McCracken, 2015). Individuals high, compared to those low, in psychological flexibility have greater emotional clarity (Salovey, Mayer, Goldman, Turvey, & Palfai, 1995), autonomy, competency (Johnston & Finney, 2010) and vitality (Kubzansky & Thurston, 2007) and fewer health problems. We rely on the well-established model of allostatic load (McEwen, 1998) to discuss how inflexibility in managing affect at any of the five phases, indexed by various markers of stress accumulation, may explain the association between alexithymia (and other emotional disorders) and somatic and mental illness.

## What are emotions?

Emotions are subjective, physiological, expressive, and functional phenomena reflecting motivational tendencies of approach toward appetitive and withdrawal from aversive stimuli (Bradley & Lang, 2000; Davidson et al., 2000). Lang's (1979) Bio-Informational Theory describes emotions as action dispositions, which instigate behaviours, in response to emotional events, even before one is aware of an emotion or consciously selects appropriate action. This early response involves activation of physiological processes that mobilise autonomic, somatomotor, endocrine and metabolic systems (Phillips, Drevets, Rauch, & Lane, 2003).

In studying emotions in psychopathology and conditions like alexithymia, most research has focused on reactivity to emotional stimuli. Going beyond reactivity, however, several theorists (e.g. Baumann et al., 2007; Davidson et al., 2000; Fanselow, 1994; Lang, Bradley, & Cuthbert, 1997; Skinner & Zimmer-Gembeck, 2007) describe how emotional responses vary along a "cascade" of stages, that unfold as the eliciting event becomes more imminent, threatening or proximal in time and space. According to Fanselow (1994), "pre-encounter" takes place when harm is distant or unlikely, eliciting anticipation, vigilance and risk assessment, whereas "post-encounter" occurs when actual or perceived threat is detected, evoking freezing and orienting, to prevent encounter with threat. When actually facing threat ("circa-strike"), active fight or flight are initiated. Specific patterns of the dynamic, fluctuating trajectories of emotions are suggested by Kuppens and Verduyn (e.g. 2017) to reflect emotion dysregulation and illness vulnerability, while Davidson (2015) and Trull, Lane, Koval, and Ebner-Priemer (2015) stress the need to study individual differences in affective dynamics in order to understand psychopathology and well-being.

## Unfolding emotional processes

We propose a dynamic-phasic approach to studying emotion processing, focusing on *anticipation, response, recovery, habituation*, and *rest* (Figure 1). These stages are not delineated by sharp boundaries, but smoothly evolve into one another, not always in sequence (e.g. a predator may surge while the

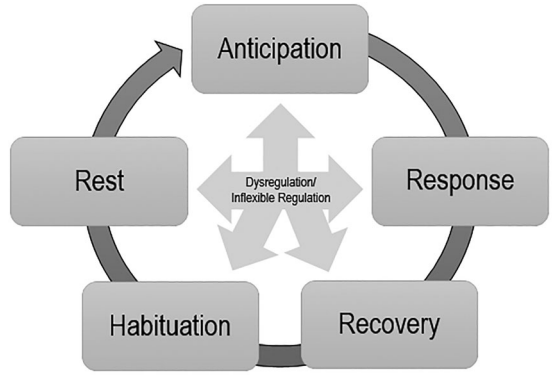


**Figure 1.** A dynamic-phasic model of emotion processing.

organism recovers from a previous attack, without the chance for rest or anticipation). Studying each stage, involving processes serving different survival functions, can inform conclusions regarding when and under what circumstances emotion dysregulation occurs.

### Anticipation

Organisms anticipate challenges that can be foreseen, using contextual cues to predict upcoming motivationally relevant stimuli based on previous learning and memory (Cravo, Rohenkohl, Santos, & Nobre, 2017), in order to minimise bodily disruption and optimise perception and action generation (Mossbridge, Tressoldi, & Utts, 2012). However, one's ability for anticipation is influenced by characteristics of the cueing environment and individual differences. Worry, rumination and anxiety may lead to chronic arousal and hypervigilance (Verkuil, Brosschot, Putman, & Thayer, 2009), unnecessarily depleting resources, whereas attentional avoidance impairs anticipation of potentially relevant stimuli.

### Response

Organisms react to perceived emotional stimuli, based on their identification and appraisal (Phillips et al., 2003). Individual differences, e.g. in anxiety, affect appraisals and may produce attention biases, higher engagement and difficulty in disengagement from threatening stimuli (Cisler & Koster, 2010). Also, stimulus and task characteristics influence perception, evaluation (Grynberg, Chang, et al., 2012) and response, including the presence of competing stimuli (i.e. affective priming; task perceptual load), the subjective meaning of a stimulus, its modality, intensity, salience, familiarity, concreteness and integrity. In this complex context, the individual must attend to significant stimuli, ignore insignificant ones and initiate appropriate coping.

Flexible and adaptive emotional functioning means producing goal-appropriate, timely and fittingly intense responses, rather than reacting invariably, irrespective of the emotional events' proximity and significance (Waugh, Thompson, & Gotlib, 2011). Consistently derailing or interrupting the smooth evolution of emotional responses because of unwillingness or inability to engage or disengage with an event, irrespective of its relevance for survival or other goals, can increase allostatic load. Flexibility should pertain to all aspects of emotional responses. Poor valence granulation (e.g. anxious individuals invariably perceiving facial expressions as disapproving) will result in disproportionate approach/withdrawal behaviours, such as unnecessary social withdrawal. Excessive or deficient arousal (e.g. consistently perceiving situations as threatening) can interfere with attention deployment, performance, and learning, depletion of resources, increased risk exposure or reduced reinforcement opportunities.

Essential for adequate reactivity is appropriate appraisal of one's ability to cope (Carver & Scheier, 1988). Emotional challenges that are appraised to exceed coping resources may elicit low physiological reactivity as in depression, melancholia, generalised anxiety or PTSD (Liu, Sarapas, & Shankman, 2016; McTeague & Lang, 2012). This freezing response is adaptive for short-term escape (Porges, 2001), but can accumulate stress in the long-run.

Finally, adaptive reactivity itself evolves dynamically, dependent on the activity of different physiological and neural systems, in response to contextual requirements. For example, during a novel stimulus, heart rate accelerates for the first 4–6s, followed by deceleration in 17–23s, involving sensory intake and release from parasympathetic control, and then by secondary, sympathetically mediated, acceleration at 31–76s (del Paso, Godoy, & Vila, 1993) reflecting mental processing and action preparation (Bradley & Lang, 2000). Parallel processes occur in the electrodermal, facial and other systems.

Responding inflexibly as if all stimuli at all times are equally significant, threatening, appetitive, or indifferent, irrespective of their proximity and relevance to immediate or long-term goals, characterises conditions like anxiety (extreme threat responses), depression (lack of pleasure even from previously desired activities), psychopathy (lack of fear, even in danger), and alexithymia (low emotional response irrespective of stimulus significance). Such inflexible responding may disrupt psychological and physiological processes (McEwen, 2007), triggering mental and physical illness progression, or disengagement from goals, needs, values and relationships.

### Recovery

Reactivity is adaptive for survival, only while the emotional stimulus is or will be present. Once the stimulus ends, one should recover and return to

resting state, in order to conserve energy and restore homeostasis (Zautra, 2009). Good recovery involves quickly regaining equilibrium physiologically and psychologically, by deactivating the sympathetic and activating the parasympathetic system (Appelhans & Luecken, 2006), and is essential to make resources available for exploration, need satisfaction, life goals and novel challenges. Inability to inhibit reactivity and disengage from stressors can cause exhaustion through tonic hyperarousal (McEwen, 1998). Slowed recovery and "difficulty in processing safety signals" (Pieper, Brosschot, van der Leeden, & Thayer, 2007) as often found in anxiety, can result in exhaustion and desensitisation from responding to later, real threat.

### *Habituation*

Habituation describes the process of becoming less reactive toward repeated or familiar stimuli that no longer carry the same novelty and significance (Grissom & Bhatnagar, 2009; Rankin et al., 2009). Habituation rates depend on the intensity and familiarity of the stimulus; less intense and more familiar stimuli produce stronger habituation (Herman, 2013). Although habituation, strictly speaking, results from non-associative learning processes, it may become dependent on context through associative learning, being thus affected by memory and learning processes. Whereas fast habituation has been linked to health (Bower, Low, Moskowitz, Sepah, & Epel, 2008) habituating too slowly is connected to PTSD, (Shalev et al., 2000), anxiety (Frith, Stevens, Johnstone, & Crow, 1982) and depression in fibromyalgia (de Tommaso et al., 2011); habituating too fast has been linked to suicide propensity in depression (Jandl, Steyer, & Kaschka, 2010; Sarchiapone et al., 2017).

### *Resting state*

When an imminent challenge is not present or anticipated, the adaptive response is to recuperate and conserve resources. Intense emotional and physiological activation during such times is inefficient, deters one from pursuing goals and experiencing life, while remaining mindful of and open to new opportunities and challenges, and characterises conditions like anxiety and depression (Abercrombie et al., 1998).

## Flexible emotion regulation

Humans can adjust their emotional reactions along dimensions of valence, arousal, and approach/avoidance, before, during or after an emotional event (Koole, 2009). Persevering with a strategy to modulate affect, irrespective of the context and one's goals, can incur mental and physical costs. There is evidence that reappraisal strategies predict positive health outcomes and well-being, whereas avoidance, suppression and rumination are less adaptive, predicting depression, eating disorders, substance use and anxiety (Aldao, Nolen-Hoeksema, & Schweizer, 2010). However, even "adaptive" strategies can be inappropriate for certain situations (Sheppes, Scheibe, Suri, & Gross, 2011), while strategies considered "maladaptive" are sometimes conducive to health. We, and others, suggest that adaptive emotion regulation depends on synchrony with the context and one's needs, goals and values (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004; Kashdan & Rottenberg, 2010), and thus no strategy is inherently good or bad.

Adaptive emotion regulation requires psychological, behavioural and physiological processes and strategies to change fluidly as the situation evolves and new demands arise (Aldao et al., 2015; Leonidou, Panayiotou, Bati, & Karekla, 2019). Physiologically, homeostatic feedback and feedforward processes must be flexibly enlisted (Friedman, 2007). Allostasis defines such dynamic regulatory processes that allow bodily systems to meet situational demands (McEwen, 1998). Although allostasis is essential to optimal bodily functioning, it comes with a cost, called allostatic load, defined as the cumulative wear and tear on the body as a result of adaptation (McEwen, 2007). Under normal circumstances and for most individuals, adaptive allostasis involves neuroendocrine (e.g. cortisol, (nor)epinephrine), cardiovascular (e.g. blood pressure, heart rate), metabolic (e.g. glucose, insulin) and immune (e.g. cytokines; Kano, Grabe, & Terock, 2018; McEwen, 1998) response fluctuations. However, when one's affective response does not fit the situation, the organism may incur allostatic load and illness vulnerability, via malfunctions in neuroendocrine (e.g. emotional and cognitive deficits), cardiovascular (e.g. atherosclerosis, myocardial infarction), metabolic (e.g. diabetes, obesity) and immune systems (immune suppression; Juster, McEwen, & Lupien, 2010). In the long run, allostatic load can exhaust mediating stress systems, reduce functionality of effector systems (cardiovascular,

respiratory, gastrointestinal) and predispose to disease (McEwen, 1998, 2007).

In sum, emotion processing and regulation must be flexible, producing psychological and physiological responses that are timely and commensurate to the situation. Flexibility requires a wide repertoire of strategies and the ability to select and implement those contextually appropriate. The next section describes how alexithymia may involve low flexibility and dysregulation, potentially along all five stages of processing, leading to accumulation of allostatic load that may explain its link to negative physical and psychological outcomes. The section also notes areas where current evidence is inconclusive, necessitating additional research.

## Emotional dysregulation in alexithymia

The focus of research on the response stage of processing has influenced theorising about alexithymia. Based on early observations of high autonomic reactivity but low emotional awareness in alexithymia, the "blindfeel" hypothesis was proposed (Lane, Ahern, Schwartz, & Kaszniak, 1997). Later evidence proved to be quite mixed, and in fact seems to weigh in the opposite direction, of high or normal subjective responses paired with low physiological arousal (Panayiotou et al., 2018b). Hence, emotion-processing deficits in alexithymia remain elusive making theorising difficult and hindering development of interventions. Adopting a dynamic-phasic approach, may broaden the scope of research, clarifying the forms of emotion dysregulation associated with this trait. As an example, this approach can be used to assess hypotheses that alexithymia involves a defensive way of dealing with emotions or experiential avoidance (Krystal, 2015; Panayiotou, Leonidou, Constantinou, & Michaelides, 2018a; Panayiotou et al., 2015). Such avoidance might take the form of low physiological and/or subjective reactivity during the response stage. Null findings when testing this hypothesis can be further explored through examination of all five phases. They might be due to reactivity being too high during baseline or anticipation, masking the expected effects. Findings of inappropriate reactivity during the response stage may in turn, generate further predictions about delayed recovery, poor habituation to repeated exposure, heightened anticipation of future exposures, and high tonic reactivity during rest. Even within the response stage, a dynamic perspective would prompt micro-analysis of responses to identify moment-to-moment dynamic response changes within very short time windows, to observe the engagement, maintenance and disengagement of processes such as orienting, vigilance, selective attention, sustained attention and arousal. It is possible, that, as predicted by vigilance-avoidance models of anxiety, early hypervigilance and hyper-reactivity is followed by attentional withdrawal and blunted responses in alexithymia as well, but only a dynamic approach would unveil this.

Indeed, the extant literature suggests that alexithymia may entail emotion dysregulation across all or most of the five phases, resulting in allostatic load and illness (Honkalampi, De Berardis, Vellante, & Viinamäki, 2018; Porcelli & Taylor, 2018). Allostatic load may accumulate along four main markers of maladaptation: *frequent stress exposure, inadequate response, failed shutdown*, and *failure to habituate* (McEwen, 1998; McEwen & Gianaros, 2010). We next review evidence for these markers in alexithymia, discuss how these fit within our dynamic-phasic framework and highlight areas where additional research is clearly needed.

### *Anticipation*

Hypervigilance to and over-perception of emotional stimuli or failure to adequately anticipate them using prior learning, can result in unnecessary stress exposure, inefficient coping and stress accumulation (Grupe & Nitschke, 2013), contributing to *frequent stress* (Loewenstein & Lerner, 2003). Inadequate anticipation may also disrupt social relationships, creating new interpersonal stressors. Some of the few studies examining anticipatory processes in alexithymia support hypervigilance to and enhanced the anticipation of emotional stimuli (Delle-Vigne, Kornreich, Verbanck, & Campanella, 2014), with increased physiological arousal (Fukunishi, Sei, Morita, & Rahe, 1999; Panayiotou & Constantinou, 2017). de Timary, Roy, Luminet, Fillée, and Mikolajczak (2008) found that difficulty in describing feelings was associated with increased cortisol levels during stress anticipation, but not during stress exposure. Two studies showed increased skin conductance to a first exposure to an emotional stimulus, but no alexithymia effects during later exposures, (Grynberg, Davydov, Vermeulen, & Luminet, 2012; Rabavilas, 1987), suggesting hyper-reactivity to novelty; this perhaps could reflect increased vigilance. Byrne and Ditto (2005) found increased blood pressure in alexithymia during stress anticipation.

Some contradictory results also exist: Pollatos et al. (2011) and Starita, Làdavas, and Di Pellegrino (2016) found decreased skin conductance in alexithymic participants before a public speaking stressor and aversive electrical stimulation respectively. Bokeriia, Golukhova, Polunina, Davydov, and Kruglova (2008) reported reduced baseline heart rate in alexithymic patients awaiting cardiac surgery.

Anticipation deficits may relate to difficulties in learning and memory (Luminet, Vermeulen, Demaret, Taylor, & Bagby, 2006; Vermeulen, Domachowska, & Nielson, 2018) leading to failed recognition of predictors of repeated emotional events (Rankin et al., 2009). However, further research is needed to document anticipation deficits and link them to these observed difficulties, as dysregulation during anticipation remains a rather under-studied domain. Future, well-designed studies should control the amount and type of information conveyed by cues predicting repeated emotional stimuli, while differentiating clearly between inappropriate reactivity during anticipation, baseline, and exposure phases.

### Response

Emotional hyper- and hypo-reactivity, i.e. *inadequate response*, is an important source of allostatic load (McEwen, 1998), possibly due to poor evaluation of the stimulus as potentially threatening (valence) or its proximity/intensity (arousal), and deficits in learning and memory, as found in alexithymia (Luminet et al., 2006; Vermeulen, Toussaint, & Luminet, 2010). They may also relate to deficiencies in deploying attention appropriately during perception, to initiate reflexes and behaviour. Limited available electrophysiological and neuroimaging evidence indeed suggests poor early encoding, differentiation, recognition and granulation of emotional stimuli in alexithymia, (see systematic review, Grynberg et al., 2012): Pollatos and Gramann (2011) showed reduced P1 amplitudes to emotional pictures, suggesting early perceptual difficulties that may deter later processing and awareness. No emotional modulation of the N190, indicating poor encoding of fearful postures was also found (Borhani, Borgomaneri, Làdavas, & Bertini, 2016), as well as delayed N2b component, showing delayed attentional disengagement and shifting toward new information (Vermeulen, Luminet, De Sousa, & Campanella, 2008). Contradictory evidence of enhanced evoked potentials during early processing in alexithymia (Delle-Vigne et al., 2014; Schäfer, Schneider, Tress, & Franz, 2007) has been interpreted as a need to deploy greater cognitive resources for emotion perception. fMRI studies concur with poor attention allocation and encoding, showing lower activation of emotional brain areas in alexithymia during brief presentations of facial expressions (Grynberg, Chang, et al., 2012).

Ample research now supports hypo-reactivity in alexithymia across physiological systems. Although some findings show equivalent autonomic responses to controls, and some show hyper-reactivity, a substantial corpus of studies (reviewed in Panayiotou et al., 2018b) shows reduced cardiovascular and electrodermal responses to emotional challenges, indexing limited sympathetic activation, energy mobilisation and action preparation that potentially impairs coping and awareness, leading to inadequate emotional responding. Specifically, the sympatho-adreno-medullary (SAM) system responds to acute stress by releasing catecholamines, allowing energy mobilisation, through glucose and fat release, increased respiration, heart rate and blood flow (see Kano et al., 2018). Hypo-reactivity may indicate inadequate fight-flight responses, increasing risk to the organism. Stress also activates the hypothalamic pituitary adrenal (HPA)-axis and glucocorticoid production, which acutely inhibits pro-inflammatory cytokine production, preventing inflammation (Juster et al., 2010). A deficient immune response elevates risk for physical and psychiatric disease, as immune responses fight infections and maintain health (McEwen, 2007). However, findings on endocrine responses in alexithymia remain inconclusive, with some studies suggesting HPA axis hyper-reactivity during stress, and others supporting no relationship between alexithymia and neuroendocrine activity (Kano et al., 2018). Somewhat more consistently, findings suggest HPA hyporeactivity during baselines, which may result from prolonged stress and repetitive HPA axis overstimulation (Kano et al., 2018). Thus, the impact of observed hypo-reactivity in alexithymia on primary mediators of allostatic load needs to be further examined in order to delineate illness pathways.

### Recovery

Difficulties in returning to baseline when an emotional stimulus ends may result in *failed shutdown* (McEwen, 1998). For fast and adequate recovery, flexible attention shifting and response inhibition are essential.

Considering attention allocation deficits in alexithymia, compromised inhibitory and switching capacities could contribute to slow or inadequate recovery from stressors (Gotlib & Joormann, 2010), tonic hyperarousal and *failed shutdown*, leading to greater allostatic load. Further, given that emotion modulation is facilitated by the ability to represent affect symbolically and connect it to past experiences, memory and learning deficits in alexithymia (Darrow & Follette, 2014; Luminet et al., 2006) may compromise the ability to update one's evaluations of and responses to emotional information (e.g. that a feared stimulus has moved to a safe distance).

In effect, evidence for recovery difficulties in alexithymia is inconclusive, with few studies specifically addressing this issue. Many studies report similarities in physiological responses between high and low alexithymic individuals following music (Grynberg et al., 2012), public speech (Eastabrook, Lanteigne, & Hollenstein, 2013; Pollatos et al., 2011), Stroop tasks (Connelly & Denney, 2007), and other emotional, cognitive and stress tasks (e.g. Martínez Sánchez & García, 2011; Martínez Sánchez, Ortiz Soria, & Ato García, 2001). Nevertheless, a few studies support no or slow recovery in alexithymia. Davydov, Luminet, and Zech (2013) found that participants high in externally oriented thinking had no return to baseline after a sad-avoidance film; Gündel et al. (2004) showed that alexithymic spasmodic torticollis patients had increased skin conductance during recovery from affective stimuli. However, these findings must be seen cautiously, as alexithymic participants showed poor change from baseline during emotional stimulation.

Altogether, some evidence of aberrant recovery in alexithymia exists, but most findings support normal physiological recovery (Panayiotou et al., 2018b). In the future, the methodological issues of differences in baselines preceding, and reactivity during emotional tasks need to be better accounted to draw firm conclusions regarding recovery dysregulation in alexithymia, a phase of processing for which data are currently inconclusive.

### Habituation

Memory deficits in alexithymia (Luminet et al., 2006; Vermeulen et al., 2010), and difficulties in learning from experience (Darrow & Follette, 2014) may lead to continued reactivity in response to repeated emotional exposures, and contexts that have become associated with these, as if the stimulus continued to carry the same novelty and significance. Evidence for *failure to habituate* (McEwen & Gianaros, 2010) in alexithymia is limited, with some results suggesting reduced habituation compared to controls, while other studies showing stronger habituation (see Panayiotou et al., 2018b). Luminet, Rimé, Bagby, and Taylor (2004), showed that difficulty in describing feelings was positively correlated with heart rate both at exposure and re-exposure suggesting lack of habituation. Bogdanov et al. (2013) found delayed skin conductance habituation in alexithymia during a computer game.

In contrast, Starita et al. (2016) provide evidence for faster extinction of electrodermal responses when cues were no longer followed by aversive electrical stimulation in high compared to moderate or low alexithymic participants, indicating stronger habituation. Grynberg et al. (2012) and Rabavilas (1987) showed that alexithymic participants exhibited high autonomic reactivity during a first exposure to an affective stimulus, but findings became nonsignificant during subsequent exposures, suggesting the presence of fast habituation. Habituating too fast may represent a form of dysregulation as one may not fully process a significant stimulus or override necessary coping too quickly, placing oneself at risk (Sarchiapone et al., 2017). Thus, although dysregulation during habituation is another potential route to continued stress exposure in alexithymia, current evidence is mixed, and it is unclear if it converges towards habituation being too fast or too slow, necessitating further research.

### Rest

*Failed shutdown* can also result from increased activity during resting or baseline states. Increased tonic arousal may result from hypervigilance to threat and inability to process safety signals (Panayiotou & Constantinou, 2017), along with reduced inhibitory capacities (Delle-Vigne et al., 2014), resulting in chronic activation of SAM and HPA systems. Although acute release of glucocorticoids inhibits the production of pro-inflammatory cytokines, chronic release of glucocorticoids by the HPA-axis enhances pro-inflammatory cytokines production, resulting in immune response suppression. Prolonged glucocorticoid release has been implicated in Cushing's disease, dementia, sleep deprivation, depression and anxiety, and seems to cause atrophy in the prefrontal

cortex, which may instigate mental disorders (McEwen, 2007). Chronic SAM activation contributes to strokes, hypertension and heart disease (Fredrikson & Matthews, 1990) and susceptibility to infections (Cohen & Rodriguez, 1995) via chronic sympathetic arousal and low parasympathetic control. Currently, a few studies provide evidence for baseline hypofunction of the HPA axis in alexithymia (Kano et al., 2018); this however, may be a result of chronic hyper-activation which desensitises the endocrine system. Chronic arousal may also affect illness perceptions, causing bodily sensations, which may be misinterpreted as illness symptoms (Leonidou & Panayiotou, 2018), explaining in part the link between alexithymia and somatisation.

With regards to physiological arousal during rest in alexithymia, several studies suggest increased cardiovascular or electrodermal activity (Davydov et al., 2013; Eastabrook et al., 2013; Gündel et al., 2004; Martínez Sánchez & García, 2011; Panayiotou & Constantinou, 2017), although many studies find equivalent, and a few reduced tonic arousal compared to control participants (Panayiotou et al., 2018b). Depending on the instructions given to participants, baselines may represent either rest or anticipation, necessitating more studies with experimental designs more clearly differentiating between rest and other phases of processing.

### *Inflexible emotion regulation strategies*

Research on self-reported emotion regulation strategies suggests inflexible regulation in alexithymia, leading to *frequent stress*. Alexithymia reliably correlates positively with strategies known to generally increase the severity and frequency of unwanted thoughts and feelings including non-acceptance, experiential avoidance and suppression, and negatively with active coping and reappraisal (Ghorbani, Khosravani, Bastan, & Ardakani, 2017; Panayiotou et al., 2018a). Further, alexithymic individuals may have a limited repertoire of emotion regulation skills (Darrow & Follette, 2014). Reduced corrective learning due to inflexible use of ineffective coping can compromise decision making, solidification of coping repertoires, resilience during future challenges and social effectiveness (Aldao et al., 2010; Craske, 2015; Kashdan & Rottenberg, 2010) incurring increased allostatic load and compromised physical and mental health. In line with this hypothesis, research suggests that reliance on avoidance, mediates the association between alexithymia and depression, psychosomatic symptoms and social anxiety (Panayiotou, 2018; Panayiotou et al., 2015).

## Summary and conclusions

In this conceptual review, we first argue for a dynamic-phasic perspective to the study of emotion processing and regulation in order to understand the complex etiologies of conditions and disorders involving emotional difficulties. The case of alexithymia nicely demonstrates this point, as much research has focused primarily on the response stage of emotion processing, whereas limited evidence regarding anticipation, recovery, habituation and rest, hints to the possibility that dysregulation may occur during these phases as well. The proposed dynamic phasic approach contributes to a future research agenda, aiming to examine emotional processes throughout the cascade of responses to emotion challenges, setting *a priori* hypotheses and launching appropriate designs to address them, in order to understand emotion dysregulation in alexithymia, and other disorders. In future designs, phases must be clearly delineated, manipulated and compared within-subject, so that, (1) rest and anticipation are clearly distinguishable, (2) emotional reactivity is assessed as difference from rest and anticipation (using stimuli and challenges varying in valence and arousal), (3) recovery is evaluated as return from an active response to a stimulus, (4) habituation is examined to repeated stimulation. Comparing activity elicited by each of these phases between participants with different levels of alexithymia (while controlling for confounding traits such as anxiety and depression) will enhance understanding of which aspects of emotional stimuli (valence, arousal, social/non-social) and which processes underlying emotion regulation (e.g. attention, learning, memory) are deficient in alexithymia. A future agenda can also utilise additional markers of anticipation (e.g. eye fixations), habituation (e.g. skin conductance responses to acoustic startle; Walker et al., 2019) or startle reflex habituation; (Panayiotou & Constantinou, 2017) or recovery (e.g. face-locked P3 component; Morriss, Taylor, Roesch, & van Reekum, 2013). Because psychological and physiological mechanisms within each stage are dynamic, analysis of moment to moment responses is also necessary, including ecological momentary assessment of emotion regulation, subjective experience, cognitions, behaviours and physiological

responses in real-life settings (Shiffman, Stone, & Hufford, 2008).

The second argument made in this paper concerns the need for a flexible emotion regulation system that helps one adjust to the changing demands of the environment, while serving one's values, goals and needs. In a future research agenda for alexithymia, questions relevant to flexibility involve the appropriateness of valence evaluations and levels of arousal for the time and stage of processing, considering the significance and proximity of the stimulus, and the ability to modulate responses accordingly. Inflexibility at the psychological and physiological level and in the use of dispositional coping should also be examined in relation to its effect on immune, neuro-endocrine, cardiovascular and muscular systems, to identify specific disease progression mechanisms. For example, alexithymic hypo-reactivity during the response stage may result in system insensitivity; when chronically unresponsive to emotional stimuli, the HPA and SAM systems don't 'practice' and desensitise, which further impairs phasic responding to emotional stimuli. This pattern of 'desensitization' is commonly seen, in the form of blunted physiological reactivity to stressors, in depression, PTSD and trauma (e.g. McTeague & Lang, 2012). On the other hand, excessive stress exposure, due to mechanisms described above, could lead to disease through acute surges in autonomic parameters, such as heart rate or blood pressure leading to atherosclerosis and/or myocardial infarction (McEwen, 1998). Chronic activation of glucocorticoids reduces immune responding to acute challenges (Irwin & Miller, 2007), documented in alexithymia (see review Uher, 2010), which can also in part explain the high prevalence of alexithymia in mental (Honkalampi et al., 2018; Morie & Ridout, 2018) and physical illness (see reviews Porcelli & Taylor, 2018; Uher, 2010) and medically unexplained symptoms (De Gucht, Fischler, & Heiser, 2004). In the long term, inappropriate levels of activation during various stages of processing may become the new body norm, via alteration of allostatic set points, further increasing inflexibility, physiological dysregulation and illness risk (Herman, 2013).

Progress in the treatment and prevention of alexithymia and associated disorders can be more forthcoming when central pathogenic mechanisms are identified and targeted. Psychological and physiological inflexibility may present such a target. Training in diverse emotion regulation skills may address difficulties at different stages of processing (e.g. exposure for improved habituation, mindfulness and relaxation training for difficulties at rest, thought restructuring skills for recovery), permitting alexithymic individuals to more fully process emotional challenges through improved awareness, tolerance, acceptance and flexibility, in ways consistent with the situation and one's life goals, needs and values.

## Notes

1. We use the term emotion regulation to broadly encompass efforts to change emotions, and refer to emotional processing as the unfolding of responses from the time before an emotional event occurs until it is no longer present.
2. In addition to inflexibility, there are potentially other characteristics of suboptimal emotion regulation, e.g. decoupled responses among response systems (as found in alexithymia; see Panayiotou et al., 2018b) but these, and their relation to the model presented here are beyond the scope of this discussion.

## Disclosure statement

No potential conflict of interest was reported by the authors.

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