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Reflecting on Rumination:

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We review research showing that rumination has multiple negative consequences: (a) exacerbating psychopathology by magnifying and prolonging negative mood states, interfering with problem-solving and instrumental behaviour and reducing sensitivity to changing contingencies; (b) acting as a transdiagnostic mental health vulnerability impacting anxiety, depression, psychosis, insomnia, and impulsive behaviours; (c) interfering with therapy and limiting the efficacy of psychological interventions; (d) exacerbating and maintaining physiological stress responses. The mechanisms underlying rumination are examined, and a model (H-EX-A-GO-N – Habit development, EXecutive control, Abstract processing, GOal discrepancies, Negative bias) is proposed to account for the onset and maintenance of rumination. H-EX-A-GO-N outlines how rumination results from dwelling on problematic goals developing into a learnt habit that involves the tendency to process negative information in an abstract way, particularly in the context of poor executive control and negative information-processing biases. These proximal factors integrate experimental evidence to provide a partial answer to the critical question of what maintains rumination. They constitute a pathway by which more distal biological and environmental factors increase the likelihood of rumination developing. Treatments for rumination are reviewed, with preliminary trials suggesting that psychological interventions designed to specifically target these mechanisms may be effective at reducing rumination.

Highlights

- We review and synthesise evidence of the multiple negative consequences of rumination. .
- Past models and empirical evidence are integrated to form a new theoretical account.
- The H-EX-A-GO-N model outlines proximal mechanisms driving pathological rumination.
- These mechanisms map well onto distal biological and environmental vulnerabilities..
- Treatments specifically targeting rumination are found to be especially effective.

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Declarations of interest: One author (EW) has developed a variant of cognitive-behavioural therapy designed to target rumination (rumination-focused CBT), and receives royalties from Guilford Press for a published treatment manual for this intervention and receives remuneration for providing training workshops in this intervention. There are no other conflicts of interest.

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Rumination is repetitive, prolonged, and recurrent negative thinking about one's self, feelings, personal concerns and upsetting experiences (Watkins, 2008). Depressive rumination has been conceptualized as repetitive thinking about the symptoms, causes, circumstances, meanings, implications, and consequences of depressed mood and distress, as outlined in Response Styles Theory (Nolen-Hoeksema, 1991). Rumination has been robustly implicated in the onset and maintenance of depression (Nolen-Hoeksema, 2000; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008) and multiple other disorders, leading to the hypothesis that it is a transdiagnostic pathological process (Ehring & Watkins, 2008; Nolen-Hoeksema & Watkins, 2011). As a consequence and indicating its perceived importance, there has been significant growth in research into rumination (e.g., in Web of Science 7197 papers involving rumination in total to April 2019; 87 papers published in 2000; 246 papers published in 2008; 826 papers published in 2018) and multiple review papers examining distinct aspects of rumination (Grierson, Hickie, Naismith, & Scott, 2016; Nolen-Hoeksema & Watkins, 2011; Nolen-Hoeksema et al., 2008; Smith & Alloy, 2009; Watkins, 2008; Watkins & Nolen-Hoeksema, 2014).

These reviews have tended to focus on specific elements of rumination in isolation, without providing an overall integration. For example, prior reviews have focused on the measurement (Smith & Alloy, 2009) or consequences (Watkins, 2008; Nolen-Hoeksema et al., 2008; Nolen-Hoeksema & Watkins, 2011) of rumination, without reviewing developments in treatment or understanding its underlying mechanisms. Earlier reviews highlighted critical and outstanding questions to resolve including: "What are the developmental antecedents of individual differences in rumination? How can depressed people avoid falling into rumination when they are trying to understand their very real problems? What makes it so difficult to break free of rumination once it has begun?" (Nolen-Hoeksema et al., 2008, p. 418). A key gap is a review of the underlying mechanisms that lead to the onset and maintenance of rumination. Moreover, the rapid growth in the rumination literature means that even relatively recent reviews are out-of-date.

This review therefore has four aims. First, to review the up-to-date literature on the consequences of rumination and why it is such an important construct in psychopathology. Second, to attempt to answer the unresolved questions noted above, and in particular to consider what mechanisms may underlie the development and maintenance of rumination, building on earlier models (Martin & Tesser, 1996; Nolen-Hoeksema, 1991; Watkins, 2008; Watkins & Nolen-Hoeksema, 2014). Third, to seek to integrate this understanding of rumination with implications for its treatment. Fourth, to describe an elaboration of the existing Response Styles Theory (Nolen-Hoeksema, 1991) and Control Theory (Martin & Tesser, 1996) accounts of rumination that focuses on five interacting mechanisms (Habit development, EXecutive Control, Abstract processing, GOal discrepancies and Negative bias – the H-EX-A-GO-N model) in order to account for the existing literature and to point towards future research. We propose an integrated theoretical framework for understanding rumination that synthesizes multiple prior models that each explain particular aspects of rumination (Control Theory, Martin & Tesser, 1996; processing mode, Watkins, 2008; goal-habit account, Watkins & Nolen-Hoeksema, 2014; transdiagnostic account, Nolen-Hoeksema & Watkins, 2011; impaired disengagement hypothesis, Koster, De Lissnyder, Derakshan, & De Raedt, 2011).

Prior reviews have identified rumination as one example of repetitive thinking about negative content, along with worry, perseverative cognition, and obsessions (Ehring & Watkins, 2008; Watkins, 2008). Although there is now extensive evidence that these different forms of repetitive negative thought are highly correlated, produce similar consequences and have overlapping processes (for evidence and further discussion of similarities and differences see Ehring & Watkins, 2008; Watkins, 2008), this review focuses specifically on rumination rather than other forms of repetitive negative thought.

We operationalized rumination as repetitive thinking about the symptoms, causes, circumstances, meanings, and consequences of negative mood. This is consistent with earlier definitions of depressive rumination as “behaviors and thoughts that focus one's attention on one's depressive symptoms and on the implications of these symptoms” (Nolen-Hoeksema, 1991, p. 569)

and as “passively and repetitively focusing on one’s symptoms of distress and the circumstances surrounding these symptoms” (Nolen-Hoeksema, McBride, & Larson, 1997). We distinguish rumination from worry, which has been defined as “a chain of thoughts and images, negatively affect-laden and relatively uncontrollable”, and as “an attempt to engage in mental problem-solving on an issue whose outcome is uncertain but contains the possibility of one or more negative outcomes” (Borkovec, Robinson, Pruzinsky, & Depree, 1983) (p. 9).

The majority of the evidence reviewed in this manuscript is based on paradigms and measures specific to the depressive rumination construct, and all is consistent with the operationalization above. For example, depressive rumination is typically assessed on the Response Styles Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991), which asks participants to endorse how much they ruminate in response to sad or depressed mood (e.g., When you feel sad, down or depressed how often do you: "Think 'Why do I always react this way?'"), and many of the studies reported here used this measure. Within depressive rumination, based on factor analyses of the RSQ, specific subtypes have been identified, most particularly, brooding, which is characterized by “moody pondering”, negative self-evaluative thinking (e.g., “Why can’t I handle things better?”) and comparative thinking about the self (e.g. “Why do I have problems other people don’t have?”) and which has been found to be the most pathological form of rumination (Treynor et al., 2003).

This review will first consider up-to-date evidence on the consequences of rumination, before outlining the developmental antecedents and mechanisms that underlie the onset and maintenance of rumination. Finally, we review treatments for rumination, and future research directions.

Consequences of Rumination

Rumination Exacerbates Psychopathology

A key finding across experimental and prospective studies is that rumination can exacerbate psychopathology in at least four ways: (a) it magnifies and prolongs existing negative mood states and associated negative thinking; (b) it interferes with effective problem-solving; (c) it interferes

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with active instrumental behaviour; (d) it reduces sensitivity to changing contingencies and context.

Each of these effects is briefly reviewed in turn.

Rumination as an emotional magnifier.

Experimental studies manipulating rumination have indicated that rumination has negative causal effects on mood and mood-related cognition in the short-term, which if occurring chronically or repeatedly, would necessarily lead to emotional disorders (Nolen-Hoeksema et al., 2008). These experimental manipulations have typically compared a standardized rumination induction, in which participants are instructed to think about prompts focusing their feelings, symptoms, and their causes and consequences, versus a distraction induction, in which participants are instructed to imagine visual scenarios unrelated to the self or to feelings (Lyubomirsky & Nolen-Hoeksema, 1995).

The first negative effect of rumination found in experimental studies is that it exacerbates and prolongs existing emotional states such as sadness, anger, anxiety and depression (Blagden & Craske, 1996; Bushman, 2002; Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005; Bushman & Gibson, 2011; Nolen-Hoeksema et al., 2008; Offredi et al., 2016; Rusting & Nolen-Hoeksema, 1998; Vasquez et al., 2013) and, in parallel, elaborates and further polarizes any thought content focused on during the rumination (Watkins, 2008). These differential effects of rumination versus distraction are only found when participants are already in a negative rather than an euthymic mood (e.g., in depressed or anxious patients; after a sad, anxious or angry mood induction, or when exposed to emotionally challenging stimuli), with rumination exacerbating whatever negative mood is currently active in the individual. For people already in a depressed or dysphoric mood, rumination results in more negative thoughts about the past, present, and future (Lavender & Watkins, 2004; Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; Lyubomirsky & Nolen-Hoeksema, 1995; Lyubomirsky, Tucker, Caldwell, & Berg, 1999; Rimes & Watkins, 2005). Experimental studies have similarly found that induced rumination slows emotional recovery to a prior failure event (Watkins, 2004) and increases negative emotional reactivity to a subsequent

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stressful event (Watkins, Moberly, & Moulds, 2008). When using traumatic films to induce intrusive thoughts as an analogue to Post-Traumatic Stress Disorder (PTSD), rumination exacerbates analogue PTSD symptoms and slows emotional recovery from the film (Zetsche, Ehring, & Ehlers, 2009).

These magnifying effects seem to occur because rumination increases self-focus and amplifies the vicious repetitive cycle between negative mood and negative cognition, wherein each increases the likelihood of the other (Ciesla & Roberts, 2007; Nolen-Hoeksema, 1991; Teasdale, 1988), and focuses attention on the discrepancy between one's desired state and the actual situation, making this discrepancy more salient. In vulnerable individuals, this pattern of mutual amplification between negative thinking (including rumination itself) and negative mood produces an emotional cascade, in which rumination can lead to very intense levels of negative affect. Vicious cycles of rumination and intense negative affect may thus in turn trigger further dysregulated responses in an attempt to escape these aversive states, such as distracting or compensatory impulsive behaviours, including non-suicidal self-injury (NSSI), reassurance-seeking, or bingeing on food, alcohol or drugs (Selby, Anestis, & Joiner, 2008).

Rumination interferes with problem-solving.

The second negative consequence of rumination is that it impairs problem-solving (Lyubomirsky et al., 1999), even though ruminators report that it increases perceived insight into problems (Lyubomirsky & Nolen-Hoeksema, 1993). Experimental studies demonstrate that rumination interferes with effective problem-solving both by making individuals more pessimistic, and also more abstract and less able to access specific details of how to resolve a difficulty (Donaldson & Lam, 2004; Lyubomirsky & Nolen-Hoeksema, 1995; Lyubomirsky et al., 1999; Watkins & Baracaia, 2002; Watkins & Moulds, 2005) including in dysphoric mothers with an infant under 12 months (O'Mahen, Boyd, & Gashe, 2015).

Rumination interferes with active approach behaviour.

The third negative effect of rumination revealed in experimental studies is that it interferes with the facilitation of active instrumental behaviour, such as reducing willingness to engage in pleasant activities (Lyubomirsky & Nolen-Hoeksema, 1993). Rumination is associated with increased uncertainty and reduced confidence in plans (Ward, Lyubomirsky, Sousa, & Nolen-Hoeksema, 2003), as well as increased avoidance both cross-sectionally (Bishop, Ameal, & Reed, 2018; Giorgio et al., 2010; Moulds, Kandris, Starr, & Wong, 2007) and prospectively over 7 days in undergraduates (Dickson, Ciesla, & Reilly, 2012). In bereaved adults, baseline rumination predicts avoidance at 6 months follow-up, which in turn mediates the effects of rumination on symptoms at 12 months (Eisma et al., 2013).

Rumination impairs concentration and sensitivity to context.

Experimental studies have shown that rumination impairs concentration and central executive functioning (Lyubomirsky, Kasri, & Zehm, 2003; Watkins & Brown, 2002). As a form of internal and often abstract preoccupation, rumination has been hypothesized to make ruminators less sensitive and responsive to contextual cues and events in the world around them (Watkins, 2008), including signals of potential reward, changing contingencies or interpersonal reactions. Such abstract and internal preoccupation could prevent ruminators from adaptively responding to changes in the environment or from benefiting from corrective learning that disconfirms negative beliefs (Reilly et al., 2019).

Consistent with this hypothesis, a cross-sectional study of 203 mothers found that maternal rumination mediated the relationship between postnatal maternal depressive mood and self-reported maternal responsiveness (attunement) to their infant when infants were low in negative temperament (Tester-Jones, O'Mahen, Watkins, & Karl, 2015). Multiple experimental studies have found that relative to distraction or control conditions, induced rumination interferes with contextual sensitivity, whether indexed by poor recall of details of a video in female undergraduates selected for high preoccupation for eating, shape and weight (Lehtonen et al., 2009), reduced responsiveness to infant vocalisations in mothers with Generalized Anxiety Disorder or Major

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Depressive Disorder (Stein et al., 2012; Stein, Lehtonen, Harvey, Nicol-Harper, & Craske, 2009), reduced sensitivity to infants in mother-child interactions for both dysphoric and non-dysphoric mothers (Tester-Jones, Karl, Watkins, & O'Mahen, 2017), and impaired learning that a particular stimulus would be associated with punishment in depressed individuals (Whitmer, Frank, & Gotlib, 2012). Using a reversal learning paradigm in which negative cognition was initially reinforced with an economic reward and this was subsequently reversed to reinforce positive cognition, depressive rumination was associated with slower updating of action-outcome contingencies in order to shift from retrieving negative memories to retrieving positive memories (Takano, Van Grieken, & Raes, 2019).

These four negative effects can directly exacerbate negative emotions, but also leave unresolved real-world personal difficulties that could contribute to chronic stress and the development or maintenance of emotional disorders. Consistent with this, prospective studies find that rumination mediates the association between stressful life events and later anxiety and depression (McLaughlin & Hatzenbuehler, 2009; McLaughlin & Nolen-Hoeksema, 2012; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013). In adolescents, rumination predicted increased emotional abuse and victimization from peers at 7 (McLaughlin & Nolen-Hoeksema, 2012) and 8.4 months follow-up (Shapiro, Hamilton, Liu, Abramson, & Alloy, 2013). In patients with depression, rumination predicted relationship difficulties and increased submissive interpersonal styles (Pearson, Watkins, Kuyken, & Mullan, 2010; Pearson, Watkins, & Mullan, 2010).

Importantly, there is evidence to corroborate the ecological validity of these findings that the consequences of rumination observed in the lab lead to increased distress and psychopathology in the medium-term. Studies utilizing Ecological Momentary Assessment (EMA) designs, in which the occurrence of negative affect, rumination and stress are measured multiple times per day, find that rumination predicts, mediates, and moderates subsequent distress over days and weeks. Momentary rumination has been found to predict subsequent negative affect (and vice versa) (Moberly & Watkins, 2008a; Selby, Kranzler, Panza, & Fehling, 2016; Takano, Sakamoto, &

Tanno, 2013, 2014) and to mediate the role of life stress and negative events in prospectively predicting negative affect and depressive symptoms over several weeks in both nonclinical (Genet & Siemer, 2012; Moberly & Watkins, 2008b) and clinical samples (Ruscio et al., 2015) and to moderate the role of life stress and current depressive symptoms in predicting future depressive symptoms in undergraduates (Connolly & Alloy, 2017).

Rumination as a Transdiagnostic Mental Health Vulnerability

It is unsurprising, given the extensively documented negative consequences of depressive rumination that it contributes to the onset, maintenance and recurrence of multiple disorders. This was first demonstrated in large-scale longitudinal studies in which rumination prospectively predicted the onset of major depressive episodes and depressive symptoms in non-depressed and currently depressed individuals across follow-up periods ranging from 6 weeks to 5 years (Nolen-Hoeksema, 2000; Nolen-Hoeksema et al., 2008; Watkins, 2008). Considerable subsequent evidence further implicated rumination in anxiety disorders including PTSD, substance and alcohol abuse, and eating disorders (Nolen-Hoeksema & Watkins, 2011). For example, a meta-analysis of studies revealed that rumination was significantly related to four distinct symptom types (depression, anxiety, eating, alcohol abuse) (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Large-scale prospective longitudinal studies found that rumination predicted subsequent substance abuse, eating disorders (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007), alcohol abuse (Caselli et al., 2010), and PTSD symptoms following trauma (Ehring, Ehlers, & Glucksman, 2008; Ehring, Frank, & Ehlers, 2008; Kleim, Ehlers, & Glucksman, 2007; Michael, Halligan, Clark, & Ehlers, 2007) even after controlling for initial symptoms. Further, rumination explained the concurrent and prospective associations between symptoms of anxiety and depression (McLaughlin & Nolen-Hoeksema, 2011), indicating that it is not simply an epiphenomenon of increased disorder. These results led to the proposal that rumination is a transdiagnostic process, that is, a process present across multiple

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psychiatric disorders and that causally contributes to the onset and maintenance of these disorders (Ehring & Watkins, 2008; Nolen-Hoeksema & Watkins, 2011).

In the 15 years since this transdiagnostic hypothesis was first proposed, the evidence supporting it has continued to grow. There is extensive evidence from cross-sectional studies that rumination is elevated across multiple disorders, that rumination is highest in individuals with multiple disorders relative to those with a single disorder (McEvoy, Watson, Watkins, & Nathan, 2013), including in EMA studies (Kircanski, Thompson, Sorenson, Sherdell, & Gotlib, 2015), and that comorbid anxiety and mood disorders are associated with greater rumination (Olatunji, Naragon-Gainey, & Wolitzky-Taylor, 2013). A study of adult twins confirmed that rumination was associated with increased likelihood of major depression, generalized anxiety disorder, eating disorders and substance abuse (Johnson et al., 2016).

Several new longitudinal prospective studies have found that rumination predicts multiple disorders subsequently. A longitudinal prospective study in over 1000 adolescents across 7 months found that rumination was a transdiagnostic process mediating between internalizing and externalizing symptoms in males: rumination predicted increases over time in aggressive behaviour and mediated the association with subsequent anxiety symptoms, and of both depression and anxiety with subsequent aggression in boys but not girls (McLaughlin, Aldao, Wisco, & Hilt, 2014). A 4 year prospective study in 2981 adults using the Netherlands Study of Anxiety and Depression cohort (NESDA; Penninx et al., 2008), including healthy individuals and those with a current or past affective disorder, found that rumination accounted for the co-morbidity between emotional disorders: rumination mediated the associations between both baseline fear disorders (social anxiety disorder, panic disorder, agoraphobia) and subsequent changes in distress disorders (major depression, dysthymia, generalized anxiety disorder), and vice versa (Drost, van der Does, van Hemert, Penninx, & Spinhoven, 2014). In 1859 adults also from the NESDA cohort, both healthy and with a prior history of affective disorder, baseline depressive disorders and symptom severity predicted subsequent anxiety disorders five years later and vice versa, with these effects

mediated by repetitive negative thought at 2 years follow-up (Spinhoven, van Hemert, & Penninx, 2019). A recent prospective study in newly recruited paramedics undergoing training found that rumination about memories of stressful events at the start of training uniquely predicted the onset of an episode of PTSD over the subsequent two years (Wild et al., 2016). Recent reviews have implicated rumination in post-natal depression (DeJong, Fox, & Stein, 2016), with antenatal rumination in pregnant women predicting increased depression postpartum (Barnum, Woody, & Gibb, 2013; O'Mahen, Flynn, & Nolen-Hoeksema, 2010). Rumination contributed to the maintenance of social anxiety (Modini & Abbott, 2016), including pre-event rumination before a social situation, rumination during a social situation, and post-event processing after leaving an anxiogenic social situation.

As well as this substantial evidence of the transdiagnostic effects of rumination in mood and anxiety disorders, there is increasing evidence for a role for rumination in other forms of psychopathology. We briefly summarise the emerging evidence implicating rumination in impulsive and dysregulated behaviours, insomnia, and psychosis.

Rumination and impulsive and dysregulated behaviours: alcohol and substance abuse, binge eating, non-suicidal self-injury and suicidal behaviour.

Many impulsive behaviours, including alcohol and substance abuse, bingeing, and self-harm have been hypothesized to be used functionally as a means to temporarily escape or block out strong negative emotions. Due to the emotional cascade effects of rumination in exacerbating and exaggerating negative affect, it has been hypothesized to act as a precursor to such compensatory actions (Selby et al., 2008). There is emerging evidence consistent with this view.

Rumination increases subsequent harmful alcohol and/or substance abuse, especially in female adolescents (Caselli, Bortolai, Leoni, Rovetto, & Spada, 2008; Caselli et al., 2010; Nolen-Hoeksema et al., 2007). Recent studies have found that, relative to distraction, induced rumination increased craving for alcohol in alcohol-dependent patients, but not in problem drinkers or controls

(Caselli et al., 2013), and found an association between brooding (the most maladaptive subtype of depressive rumination, characterised as "moody pondering", Treynor, Gonzalez, & Nolen-Hoeksema, 2003, p. 251) and marijuana use (Adrian, McCarty, King, McCauley, & Vander Stoep, 2014).

A recent meta-analysis found that rumination was significantly positively associated with eating disorder psychopathology including binge eating, body dissatisfaction, and eating concerns, both concurrently and prospectively, especially for rumination on eating disorder themes (Smith, Mason, & Lavender, 2018). Experimental studies that induced sadness or eating or body-related concerns and then manipulated state rumination found that rumination increased negative mood, reduced body satisfaction, and increased analogue symptoms (e.g., desire to binge or abstain) relative to acceptance or distraction conditions in women with eating disorders (Naumann, Tuschen-Caffier, Voderholzer, Caffier, & Svaldi, 2015; Naumann, Tuschen-Caffier, Voderholzer, Schaefer, & Svaldi, 2016), obese individuals (Svaldi, Naumann, Trentowska, Lackner, & Tuschen-Caffier, 2013) and undergraduate females (Wade, George, & Atkinson, 2009).

The trait tendency to ruminate, and its interaction with negative emotions, is associated with NSSI in correlational studies (Selby, Anestis, Bender, & Joiner, 2009; Selby, Connell, & Joiner, 2010), predicted subsequent episodes of NSSI over 8 weeks in a diary study in undergraduates (Nicolai, Wielgus, & Mezulis, 2016), and predicted number of NSSI episodes in a 2 week EMA study of individuals with elevated levels of dysregulated impulsive behaviour (Selby, Franklin, Carson-Wong, & Rizvi, 2013). Momentary rumination and negative emotion predicted independently and interactively the number of NSSI (Selby et al., 2016), especially in those with Borderline Personality Disorder symptoms (Selby & Joiner, 2013). A recent meta-analysis of the relationship between rumination, suicidal ideation, and suicidal attempts across 17 cross-sectional studies and 8 longitudinal studies, found that trait depressive rumination and brooding were significantly positively associated with suicidal ideation (large effect size, Hedges $g = .82$ for cross-sectional studies, $g = .62$ for longitudinal studies) (Rogers & Joiner, 2017). Depressive rumination

($g = .26$) and brooding ($g = .47$) were also significantly associated with suicide attempts, although this was only examined in cross-sectional studies, leaving the direction of causality unresolved.

Insomnia.

Rumination has been robustly implicated in poor sleep and insomnia. Increased rumination is associated with longer night-time sleep onset latency and poorer sleep quality and efficiency, whether sleep is assessed by self-report and with actigraphy after a stressful experience (a 5-minute speech) (Zoccola, Dickerson, & Lam, 2009), in young adults with elevated depressive symptoms (Pillai, Steenburg, Ciesla, Roth, & Drake, 2014) or via objective polysomnography in patients with insomnia disorder compared to healthy controls (Galbiati, Giora, Sarasso, Zucconi, & Ferini-Strambi, 2018). In undergraduates, elevated rumination predicted impaired sleep quality three months later (Takano, Iijima, & Tanno, 2012), and two EMA studies each lasting 1 week found that rumination before going to sleep in the evening predicted longer sleep onset latency on both diaries and wrist actigraphy (Pillai et al., 2014; Takano et al., 2014). In PhD students, daily variation in perseverative thinking in a daily diary predicted subjective sleep quality and objective sleep efficiency assessed by actigraphy over 2 months incorporating their viva (Van Laethem, Beckers, van Hooff, Dijksterhuis, & Geurts, 2016). In an experimental study, high trait ruminators randomized to the rumination condition just prior to sleep following an exam self-reported worse sleep relative to those randomized to distraction or low ruminators in either rumination or distraction conditions (Guastella & Moulds, 2007).

Psychosis.

It has been proposed that rumination about stressful events may provide the source material to generate auditory verbal hallucinations (Jones & Fernyhough, 2009). Consistent with this, a recent systematic review of 51 studies examining meta-cognitive processes in psychosis (rumination $n = 10$) found both experimental and cross-sectional evidence supporting an association

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between rumination and symptoms of psychosis (Sellers, Wells, & Morrison, 2018). Cross-sectional evidence consistent with this hypothesis includes rumination being correlated with self-reported hallucination-proneness in students, with this relationship mediated by intrusive thoughts (Jones & Fernyhough, 2009), and individuals experiencing psychosis reporting elevated rumination relative to controls (Vorontsova, Garety, & Freeman, 2013). In prospective studies, rumination predicts psychotic symptoms: an EMA study over 6 days in patients with psychotic disorders found that worry and rumination at one sampling point predicted self-reported delusions and hallucinations and distress at the next and subsequent sampling points (Hartley, Haddock, Sa, Emsley, & Barrowclough, 2014); rumination about a physical assault assessed 4 weeks after the trauma in participants recruited in an emergency department predicted hallucinations six months later (Geddes, Ehlers, & Freeman, 2016).

A recent study using an auditory task where participants listened to anomalous stimuli and recorded what words/phrases were heard as an analogue of voice-hearing failed to find relationship between rumination and hallucination-like experiences in undergraduates (Hartley, Bucci, & Morrison, 2017). However, this may reflect limitations in the paradigm or the relative health of the sample. Experimental studies in non-clinical samples have demonstrated that compared to a distraction condition (Martinelli, Cavanagh, & Dudley, 2013), and a mindfulness task (McKie, Askew, & Dudley, 2017), induced rumination maintained elevated levels of paranoia following a paranoia induction. Consistent with a causal role of rumination in psychotic symptoms, a recent controlled trial found that an intervention targeting worry in patients with a diagnosis of psychosis and persistent persecutory delusions significantly reduced persecutory delusions and paranoia relative to standard care over 8 weeks (Freeman et al., 2015).

Rumination Interferes with Psychological Therapy

The negative consequences of rumination could also make it harder for patients to recover from psychological disorders and respond to treatment. Impaired concentration and problem-

solving, reduced responsiveness to external contingencies and feedback, and reduced instrumental action could limit the ability of a patient to process ideas or evidence reviewed in psychological therapy, or to implement and benefit from behavioural plans, thereby interfering with therapy. Indeed, there is growing evidence that elevated rumination at the start of treatment predicts poorer outcomes to cognitive-behavioural therapy (CBT) for depression, such as increased time to remission and reduced likelihood of achieving remission (Ciesla & Roberts, 2002; Jones, Siegle, & Thase, 2008; Kertz, Koran, Stevens, & Björgvinsson, 2015; Schmaling, Dimidjian, Katon, & Sullivan, 2002). Rumination at the end of mindfulness-based CBT treatment predicts depressive relapse (Michalak, Holz, & Teismann, 2011). Changes in rumination are associated with treatment outcome for anxiety and depression (Newby, Williams, & Andrews, 2014; van Aalderen et al., 2012). For patients receiving brief CBT in a partial hospitalization program, baseline rumination predicted subsequent anxiety and depression, and examination of symptom trajectories indicated that when rumination did not improve, symptoms of depression and anxiety did not improve and were likely to worsen over time (Kertz, Koran, Stevens, & Bjorgvinsson, 2015).

Similar patterns have been found for the treatment of anxiety disorders: elevated pre-treatment worry and rumination predicts poorer treatment response in social anxiety symptoms at post-treatment (Wong et al., 2017) and to individual and group CBT at post-treatment and one year follow-up (Mortberg & Andersson, 2014). For patients receiving trauma-focused CBT for PTSD, patient rumination observed in early sessions (e.g., repeatedly returning to the same themes, asking “what if” “why” questions) was significantly elevated in poor responders to therapy (symptoms reducing $<1/3$) relative to good responders (symptom reduction $>2/3$) (Brady, Warnock-Parkes, Barker, & Ehlers, 2015).

Physical and Physiological Consequences of Rumination

It has previously been suggested that rumination may have negative consequences for physical health as well as for mental health (Brosschot, Gerin, & Thayer, 2006; Brosschot, Verkuil,

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& Thayer, 2010; Watkins, 2008), with studies finding that rumination and worry prospectively predict increased heart disease over a 20-year follow-up (Kubzansky et al., 1997). The perseverative cognition hypothesis (Brosschot et al., 2006) proposed that rumination and other repetitive thought processes repeatedly and chronically reactivate cognitive representations of stressful events and thus prolong the psychological, emotional and physiological responses produced to stressors beyond the actual occurrence of the initial event. Thus, rather than the body systems associated with stress (including increased sympathetic nervous arousal and concomitant changes in cardiovascular, hypothalamic pituitary adrenal, and immune systems), only showing an acute, time-limited and often adaptive response, these responses become chronically activated and re-occur long after the initial event, with associated greater risk for poor physical health and the development of disease (Glynn, Christenfeld, & Gerin, 2002).

Research over the last decade broadly supports the perseverative cognition hypothesis. Rumination is elevated in individuals with chronic health conditions (e.g., cardiovascular disease, obesity, chronic pain) and is associated with dysregulated physiological function, such as reduced heart rate variability, increased heart rate, and increased blood pressure (Busch, Possel, & Valentine, 2017; Ottaviani et al., 2016). There is evidence that rumination may causally contribute to (a) impaired parasympathetic flexibility; (b) exaggerated cardiovascular stress responses; and (c) disruptions in hypothalamic-pituitary-adrenal (HPA) axis stress responding.

Rumination and impaired parasympathetic flexibility.

The ability to shift the relative activation of the sympathetic and parasympathetic autonomic nervous systems in a contextually appropriate way to different environments and contexts (“parasympathetic flexibility”) is hypothesized to underpin and indicate self-regulation and emotional regulatory abilities (Kashdan & Rottenberg, 2010; Porges, 2007; Stange, Hamilton, Fresco, & Alloy, 2017). The sympathetic autonomic nervous system is responsible for stimulating “fight-or-flight” responses to perceived stress that prime the body for action via increasing the

release of adrenaline, primarily by acting on the cardiovascular system, such as accelerating heart rate, constricting blood vessels and raising blood pressure. In contrast, the parasympathetic autonomic nervous system is responsible for controlling homeostasis and the body at rest, and it tends to calm the body, decreasing heart rate and increasing digestive processes. Two key indices of parasympathetic flexibility are heart rate variability (HRV) and respiratory sinus arrhythmia (RSA), a measure of variability in heart rate that occurs over the breathing cycle (Beauchaine & Thayer, 2015; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Both indicate vagal tone, which increases with increasing activation of the parasympathetic nervous system, but reduces during stress and the activation of the sympathetic nervous system. Depression is characterized by reduced RSA reactivity to sadness and reduced HRV (Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014; Rottenberg, Clift, Bolden, & Salomon, 2007). Lowered HRV is also a widely recognized prognostic risk factor for somatic disease, including cardiovascular disorders (Thayer et al., 2012; Thayer, Yamamoto, & Brosschot, 2010).

A recent meta-analysis suggested that reduced HRV is a consequence of rumination (Ottaviani et al., 2016). When individuals are experimentally induced to ruminate about an upsetting or angry event in the lab, blood pressure and heart rate increase and there is a reduction in HRV that lasts over the next day (Ottaviani, Shapiro, Davydov, Goldstein, & Mills, 2009; Ottaviani, Shapiro, & Fitzgerald, 2011). High trait ruminators show greater reduction in HRV to an interpersonal stressor (Woody, Burkhouse, Birk, & Gibb, 2015), and trait rumination is associated with lower HRV (Carnevali, Thayer, Brosschot, & Ottaviani, 2018; Woody, McGeary, & Gibb, 2014). When individuals spontaneously ruminate in daily life their HRV is reduced as if they were facing an actual stressor (Cropley et al., 2017; Ottaviani, Medea, Lonigro, Tarvainen, & Couyoumdjian, 2015; Ottaviani, Shahabi, et al., 2015; Ottaviani et al., 2011). In prospective studies, a smaller reduction in RSA in response to a sad film predicted future symptoms of depression over 12 weeks, with this effect moderated by trait rumination (Stange, Hamilton, et al., 2017). In another study, baseline levels of rumination predicted predicted subsequent HRV at 13 months follow-up,

which in turn mediated the relationship between rumination at baseline and depression at 34 months follow-up (Carnevali et al., 2018). This convergence of experimental and prospective results suggests that rumination may causally reduce parasympathetic flexibility, which in turn predicts depression.

Rumination and exaggerated cardiovascular stress responses.

Individuals with elevated trait rumination take longer to return to cardiovascular baseline following a stressor (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Glynn, Christenfeld, & Gerin, 2007; Johnson, Key, Routledge, Gerin, & Campbell, 2014; Radstaak, Geurts, Brosschot, Cillessen, & Kompier, 2011), including in adolescents (Aldao, McLaughlin, Hatzenbuehler, & Sheridan, 2014). A recent meta-analysis of 43 experimental studies found statistically significant and large reactivity effect sizes of inducing both anger and sadness rumination on increasing cardiovascular reactivity, as indexed by heart rate, diastolic blood pressure, and systolic blood pressure, with standardized mean effect sizes (d) ranging from 0.75 to 1.39 (Busch et al., 2017). Effects were stronger on blood pressure than heart rate and for angry rumination than sad rumination.

Rumination and disruptions in hypothalamic-pituitary-adrenal (HPA) axis stress responding.

Disruptions in HPA axis responses are indexed by abnormal cortisol levels. A review of studies examining cortisol and rumination (Zoccola & Dickerson, 2012) found that higher levels of state rumination were associated with increased cortisol concentrations, and that elevated state rumination to a stressor predicted greater cortisol reactivity to or delayed recovery after a social evaluative stressor task such as the Trier Social Stress Task (TSST; giving a presentation to a committee followed by an arithmetic test, e.g., Zoccola, Dickerson, & Yim, 2011; Zoccola, Dickerson, & Zaldivar, 2008; Zoccola, Quas, & Yim, 2010). These findings were replicated in later studies including an increased cortisol response to a second TSST the next day (Gianferante et al.,

2014; Puterman et al., 2011). In depressed adolescents, elevated trait rumination was associated with slower recovery of cortisol levels following the TSST (Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013). A meta-analysis of laboratory studies that involved mood or appraisal inductions and assessed change in rumination found that rumination was associated with increases in cortisol and suppression of T lymphocytes, which is an index of immune function activity (Denson, Spanovic, & Miller, 2009).

Relative to distraction, self-focused rumination inductions elevated cortisol and prolonged duration of cortisol activation in young women (Zoccola, Figueroa, Rabideau, Woody, & Benencia, 2014), female high trait ruminators (Shull et al., 2016), undergraduates completing a difficult anagram task (Denson, Fabiansson, Creswell, & Pedersen, 2009) and patients with depression (LeMoult & Joormann, 2014). There is thus convergent evidence that rumination may play a causal role in slowing cortisol recovery following a stressor.

These physiological effects suggest that rumination may be a mechanism to partially account for the high co-morbidity observed between poor mental health and poor physical health, for example, the strong associations between depression and cardiovascular disease (Rutledge, Reis, Linke, Greenberg, & Mills, 2006; Sgoifo, Carnevali, Alfonso, & Amore, 2015; Van der Kooy et al., 2007). Indeed, rumination prospectively predicts increased depression in patients with acute coronary syndrome (Denton, Rieckmann, Davidson, & Chaplin, 2012). In a prospective community sample of over 1000 participants, individuals who experienced higher levels of negative affect the day after a stressor had greater frequency of chronic conditions and worse functional limitations 10 years later (Leger, Charles, & Almeida, 2018). Since rumination is demonstrated to lead to prolonged negative affect (as reviewed earlier), it is a strong candidate for contributing to poor chronic health, although this was not directly tested.

The next step is to explicitly test the relationship between rumination, these psychophysiological indices and long-term poor health, for example, determining whether increased rumination leads to worse physical health over time and whether this is mediated by reduced

parasympathetic flexibility, exaggerated cardiovascular responses or dysregulated HPA axis. This will require prospective studies and research in clinical populations.

Beneficial Consequences of Rumination

It is important to emphasise that rumination does not always have negative and maladaptive consequences. There is extensive evidence that under some circumstances, repetitive thought about negative mood and difficulties can be constructive and adaptive, as reviewed in detail in Watkins (2008). As summarized there, repetitive thought on negative topics can result in: (a) successful cognitive processing and recovery from upsetting and traumatic events; (b) adaptive preparation and planning for the future; (c) recovery from depression; and (d) uptake of health-promoting behaviours (see Watkins, 2008). This is consistent with the proposal that rumination is an evolved and adaptive response that involves extensive analysis to solve problems – the analytical rumination hypothesis (Andrews & Thomson, 2009; Bartoskova et al., 2018; Watson & Andrews, 2002). This hypothesis proposes that depression is an evolved response to complex problems, and that a core function of depression is to engender a sustained focus on analytic rumination in order to resolve these problems. The evidence (e.g., reviewed in Watkins, 2008) partially supports this: repeated focus on problems can sometimes be adaptive. However, the analytical rumination hypothesis does not directly address the extensive evidence reviewed earlier of the negative consequences of rumination, and in particular, that depressive rumination can impair problem-solving. Nonetheless, the existence of both positive and negative consequences of repetitive negative thought means that any comprehensive model of rumination needs to be able to explain why repetitive self-focus on negative feelings and mood can sometimes be adaptive: as explained later, one relevant mechanism is the processing style adopted during repetitive thinking – the abstract and evaluative style characteristic of depressive rumination is shown to be maladaptive.

Developmental Antecedents and Mechanisms Underlying Rumination

In considering the potential developmental antecedents and mechanisms of rumination, we first review what environmental and biological factors are associated with increased risk for depression. We then briefly review the most detailed accounts of rumination to date - Response Styles Theory (Nolen-Hoeksema, 1991) and Control Theory (Martin & Tesser, 1996), and their implications for mechanisms underlying rumination. We then describe a new model (H-EX-A-GO-N) that emerges from and elaborates on features of these models and more recent models (Raes et al., 2010; Koster et al., 2011; Watkins, 2008; Watkins & Nolen-Hoeksema, 2014), and the more recent literature to suggest five mechanisms: (a) Habit development, (b) EXecutive control, (c) Abstract processing, (d) GOal discrepancies, and (e) Negative information-processing biases, that, in combination, underlie pathological rumination (Figure 1).

<INSERT FIGURE 1 ABOUT HERE>

When considering antecedents and mechanisms for rumination, we adopt the distal-proximal factor framework used to explain the development of symptomatology (Nolen-Hoeksema & Watkins, 2011) and adapt it to rumination. In this context, distal factors are usually distant from the expression of a behaviour, in probability, mechanism, and time, independent of the actions of the individual, and only influence rumination via mediating proximal factors. In contrast, proximal factors are the within-person variables that directly precede and influence a behaviour via specific mechanisms, similar to the concept of intermediate phenotypes or endophenotypes (Cannon & Keller, 2006; Insel & Cuthbert, 2009). Distal factors are typically either prior environmental context factors such as stressful and traumatic events or biological variability, such as in brain structure or in genes, and, as such, correspond to the developmental antecedents of rumination. Proximal factors correspond to the mechanisms directly underlying the expression of rumination. A full explanatory model of rumination needs to account for both the mechanisms directly influencing rumination (i.e., proximal factors) and its developmental and biological antecedents (distal factors). We propose that considering how the proximal factors (mechanisms) and distal factors (vulnerabilities) inter-relate has explanatory utility: identifying distal risk factors for rumination will help to answer the question

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of “What are the developmental antecedents of individual differences in rumination?” (Nolen-Hoeksema et al., 2008, p. 418), and delineating proximal risk factors will examine the mechanisms directly underpinning rumination.

Distal Factors Conferring Vulnerability to Rumination

Both environmental and biological factors are associated with trait rumination. Twin studies utilising respectively 674 pairs of same-gender adolescent twins (Chen & Li, 2013), 663 adult twins (Johnson, Whisman, Corley, Hewitt, & Friedman, 2014) and 756 adolescent twins (Moore et al., 2013) consistently found that both environmental and heritable features influence rumination, with rumination estimated to be moderately heritable (20-41% of variance), but with still significant shared environmental and non-shared environmental influences. Furthermore, rumination is found to have moderate to large shared genetic variability with depression, suggesting that rumination may be an endophenotype reflecting genetic risk for depression (Chen & Li, 2013).

Environmental context associated with vulnerability to rumination.

The major environmental context factors associated with increased trait tendency to ruminate can be broadly categorized into (a) early adversity including childhood sexual and emotional abuse; (b) interpersonal stress and difficult circumstances; (c) unhelpful parenting styles and (d) socio-cultural expectancies and socialization.

Early adversity and rumination.

Across diverse populations including college students (Conway, Mendelson, Giannopoulos, Csank, & Holm, 2004; Spasojevic & Alloy, 2001; Spasojevic & Alloy, 2002), community participants (Sarin & Nolen-Hoeksema, 2008, 2010), both depressed and non-depressed pregnant women (O'Mahen, Karl, Moberly, & Fedock, 2015) and depressed patients (Watkins, 2009),

individuals who retrospectively self-report a childhood history of sexual and emotional abuse show higher trait rumination than those without such a history. Moving beyond cross-sectional data, a 3-wave longitudinal study with repeated assessment of nearly 1000 adolescents found that parental emotional abuse or peer bullying predicted increased depressive rumination, with brooding mediating the association between abuse and future depressive symptoms (Paredes & Calvete, 2014).

Interpersonal stress and adversity.

Rumination is elevated in those with family histories of mental health difficulties, and those experiencing interpersonal stress, socioeconomic disadvantage, stressful life-transitions, bullying or abuse, and acts as a common mediator between these risk factors and later psychopathology (Fritz, de Graaff, Caisley, van Harmelen, & Wilkinson, 2018; Kinderman, Schwannauer, Pontin, & Tai, 2013; Michl et al., 2013; Nolen-Hoeksema, 2000; Spasojevic & Alloy, 2001; Spasojevic & Alloy, 2002). In prospective studies, rumination fully mediated the relationship between stressful life events and later anxiety and depression among adolescents, but only partially mediated the association in adults (McLaughlin & Hatzenbuehler, 2009; McLaughlin & Nolen-Hoeksema, 2012). Rumination thus appears to act as a final common pathway for multiple distal risk factors for emotional disorders, further indicating the significance of rumination as an important target for research and intervention. Such abusive and stressful experiences could lead to the development of proximal risk factors for rumination, such as hypervigilance for threat, feeling helpless (Spasojevic & Alloy, 2002), and turning attention inwards to analyze problems abstractly and passively (Sarin & Nolen-Hoeksema, 2010).

Parenting style.

Spasojevic and Alloy (2002) found that college students who retrospectively reported that their parents were overcontrolling had higher levels of rumination. A prospective study following 337 children from ages 3-4 years to ages 13-15 found that over-controlling parenting and a family style involving higher levels of expression of submissive negative emotions such as sadness, guilt,

and embarrassment, predicted increased rumination in adolescence, with this effect moderated by children's temperamental negative affectivity and effortful control (Hilt, Armstrong, & Essex, 2012). The lack of positive support and feedback from mothers has also been implicated in the development of rumination in a multi-wave prospective study (Gate et al., 2013). During an interaction between mother and child at age 12 within the laboratory, observation of fewer positive behaviours from the mother prospectively predicted later trait rumination in girls (but not boys) at age 15. Such parenting styles may engender more passive ways of responding relative to active problem-solving.

Socialisation and socio-cultural expectancies.

Socialisation and socio-cultural expectancies also increase vulnerability for rumination. Most notably, socialization into the stereotypical feminine gender role identity, characterized by a greater focus on emotions and a more passive style, is a risk factor for rumination. Two prospective longitudinal studies in school children (Broderick & Korteland, 2002; Cox, Mezulis, & Hyde, 2010) found that greater identification with the stereotypically feminine role identity at baseline predicted greater trait rumination at follow-up, with the feminine role identity at age 11 mediating the relationship between child sex and increases in trait rumination by age 15 (Cox, Mezulis, & Hyde, 2010). During observations on a difficult computerized math task, the mother encouraging the child to focus on his or her feelings mediated between child sex and subsequent increase in depressive rumination.

Socio-cultural expectancies that place an extreme value on happiness and that emphasize the importance of not experiencing negative emotional states may also increase rumination (McGuirk, Kuppens, Kingston, & Bastian, 2018). Following failure induced via an impossible anagram task, participants primed as to the importance of happiness (test room decorated with motivational posters, well-being books and the experimenter emphasizing the importance of staying positive) reported more state rumination post-failure than participants in a neutral room without these primes (McGuirk et al., 2018). Participants who reported stronger beliefs about the value of not having

negative emotion cross-sectionally reported greater trait rumination and depression. These socio-cultural values may set up expectancies for what should be normal emotional experience, such that any deviation from this would engender negative comparisons and lead to rumination. However, this requires further testing to indicate a causal effect on trait rumination.

Biological characteristics associated with rumination.

There is emergent evidence that particular patterns of brain activation and genetic polymorphisms are associated with rumination, raising the possibility that these biological substrates contribute to the vulnerability for rumination. However, caution is needed here, as the direction of causality is not yet established: biological changes could be cause or consequence of rumination, or associated with a common third factor.

Default mode network activation

Convergent evidence from multiple neuroimaging studies has associated increased rumination with greater activation and connectivity within the amygdala, medial prefrontal cortex (MPFC) and posterior cingulate cortex (PCC), whether assessed in clinically depressed populations at rest (Berman et al., 2011; Cooney, Joormann, Eugene, Dennis, & Gotlib, 2010; Nejad, Fossati, & Lemogne, 2013; Philippi et al., 2018; Siegle, Steinhauer, Thase, Stenger, & Carter, 2002), in response to induced self-referential processing, reappraisal or emotion processing tasks (Jones, Fournier, & Stone, 2017; Mandell, Siegle, Shutt, Feldmiller, & Thase, 2014; Murphy, Barch, Pagliaccio, Luby, & Belden, 2016; Ray et al., 2005), in remitted depressed groups (Nejad et al., 2019) or in non-clinical populations (Fox et al., 2018; Nejad et al., 2019). These brain regions include areas (e.g., MPFC, PCC, inferior parietal lobe, medial temporal gyrus) that make up the Default Mode Network (DMN), which is typically activated during resting states, when an individual is focused internally rather than on external tasks or environmental stimuli, and which reduces after and during goal-directed non-self-referential activities. Consistent with the content of rumination, the DMN brain regions have been argued to reflect increased self-referential and

emotional processing. Individuals high in trait rumination show elevated activity within the DMN, especially relative to the task-positive network (TPN), which consists of prefrontal and parietal regions utilized in attention-demanding tasks (Hamilton, Farmer, Fogelman, & Gotlib, 2015; Hamilton et al., 2011; Whitfield-Gabrieli & Ford, 2012). Trait ruminators also show high connectivity within DMN regions during rest, reflecting an inability to suppress self-referential thinking (Sheline et al., 2009), especially for the more pathological brooding form of rumination (Berman et al., 2011; Bessette et al., 2018; Burkhouse et al., 2017; Hamilton et al., 2015; Jacobs et al., 2016; Ordaz et al., 2017; Zhu, Zhu, Shen, Liao, & Yuan, 2017). Rumination is also associated with elevated connectivity between the DMN and salience networks (PCC, subgenual anterior cingulate, and amygdala) and regions of the Cognitive Control Network (CCN), in adolescents with a history of depression (Burkhouse et al., 2017; Stange, Bessette, et al., 2017).

BDNF Val66Met and 5-HTTLPR polymorphisms

In addition to the evidence that rumination is moderately heritable, there is also some preliminary evidence linking specific genotypes with elevated rumination, although this needs to be treated with caution because of recent critiques of the candidate gene approach (Culverhouse et al., 2018; Duncan, Ostacher, & Ballon, 2019, although see also Vrshek-Schallhorn, Corneau, & Starr, 2019), the relatively small samples of the studies and the mixed pattern of findings. A recent review of candidate gene analyses predicting rumination aligns with this controversy in finding a somewhat inconsistent pattern of findings regarding genetic variants linked to rumination (Shaw, Hilt, & Starr, 2019). Several studies have found the Val66Met polymorphism in the brain-derived neurotrophic factor (BDNF) gene to be associated with rumination. BDNF is a neurotrophin involved in synaptic plasticity and neurogenesis, and a methionine (Met) substitution for valine (Val) at codon 66 (Val66Met) has been implicated in neurocognitive alterations and the pathophysiology of mental health conditions such as depression. It has been suggested that Met carriers may be genetically sensitive to adverse experiences such as stressful life events and childhood abuse or neglect, and this is a hypothesised mechanism underlying the observed interaction between the BDNF Val66Met

polymorphism and life stress in predicting susceptibility to depression (see Hosang, Shiles, Tansey, McGuffin, & Uher, 2014 for a systematic review and meta-analysis). The Val66Met polymorphism has also been associated with a number of domains of executive functioning, although the pattern of findings to-date is complex and conflicting (Mandelman & Grigorenko, 2012). A meta-analysis of 32 independent samples (5922 subjects) found that carriers of the Met allele exhibited poorer declarative memory ($d = 0.16$) and reduced hippocampal volume ($d = 0.12$) and activation ($d = 0.59$) (Kambeitz et al., 2012).

The serotonin transporter gene (5-HTT) encodes a protein that is important to the regulation of serotonin neurotransmission and associated emotional sensitivity. The 5-HTTLPR polymorphism includes variable repeats (a base pair insertion/deletion) in the promoter, and the short allele is associated with reduced 5-HTT availability and concomitant reductions in serotonin transporter expression, as compared to the long allele. This may modify vulnerability to the effects of stress, and there is evidence that carriers of the short allele are more susceptible to stress, both neurally (e.g., augmented amygdala response to aversive stimuli, Munafò, Brown, & Hariri, 2008), and through increased risk of negative affect (e.g., elevated levels of neuroticism, Munafò et al., 2009). A number of high profile longitudinal studies have reported a gene x environment interaction whereby carriers of the short allele exhibited increased risk for depression in response to stressful life events (e.g., Caspi et al., 2003), although recent meta-analyses call into question the extent to which these findings generalise (e.g., Culverhouse et al., 2018).

In healthy non-depressed adults individuals who carried the met allele variants of the polymorphism in the BDNF gene reported greater levels of trait rumination than those who did not (Beevers, Wells, & McGeary, 2009). Consistent with the gene x stress interaction hypothesis, in healthy never depressed individuals, those with two short alleles of the 5-HTTLPR polymorphism or two Met alleles of the BDNF Val66Met polymorphism ruminate more under conditions of life stress, compared to the other genotypes (Clasen, Wells, Knopik, McGeary, & Beevers, 2011). Similarly, individuals with two short alleles of the 5-HTTLPR polymorphism who experienced

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higher levels of emotional abuse in childhood report higher levels of rumination in adulthood than individuals carrying at least one copy of the high-expressing long allele (Antypa & Van der Does, 2010).

In another pattern of findings, in young adolescent girls at increased risk of depression (due to having a mother with depression, or being low socioeconomic status) and their mothers, differing associations between the Val66Met polymorphism, depressive symptoms, and rumination were observed in the two groups: in adolescents, the Val/Val genotype was associated with greater depression and rumination than the Val/Met genotype, with rumination mediating this relationship; in women with adult-onset depression, the Val/Met genotype was associated with greater depression and rumination again mediated this relationship (Hilt et al., 2007) . A subsequent study of 8-14 year olds, some of whom had mothers with a history of depression, replicated Hilt et al.'s finding regarding adolescence: in adolescents aged 10-14, individuals who were homozygous for the Val allele reported higher rumination scores than carriers of the Met allele (Stone, McGeary, Palmer, & Gibb, 2013). In a sample of adolescent twins ($N = 441$) aged 12-18 years, carriers of the Met allele reported higher brooding scores than individuals with the Val/Val genotype, and post-hoc analyses indicated there was an interaction with pubertal status such that this association was reliable in individuals who were more advanced in puberty, but not in those who were less mature (Van Hulle, Clifford, Moore, Lemery-Chalfant, & Goldsmith, 2017).

Hormonal variations

It has also been suggested that sex hormone concentration may be linked to the selection and effectiveness of different emotional regulation strategies including rumination in women (Graham, Denson, Barnett, Calderwood, & Grisham, 2018). Increased levels of estradiol in women were associated with greater use of rumination, whilst rumination was positively associated with negative affect, but only in women low in estradiol. These findings are only preliminary and correlational, and need to be replicated and directions of causality tested – nonetheless, they raise the possibility that hormones may also influence tendency towards rumination.

Proximal Mechanisms Underlying Rumination

In considering the mechanisms underpinning rumination, we start by considering the two predominant theories of rumination – Response Styles Theory (Nolen-Hoeksema, 1991) and Control Theory (Martin & Tesser, 1996), which respectively contribute two key mechanisms: the formation of a pathological habit through repeated learning and activation of rumination via unsatisfactory goal progress.

Response Styles Theory (Nolen-Hoeksema, 1991)

Response Styles Theory hypothesizes that depressive rumination is a stable, enduring, and habitual trait-like tendency to engage in repetitive self-focus in response to depressed mood. It further proposes that rumination is principally dysfunctional and that it contributes to depression by enhancing negative mood-congruent thinking, impairing problem-solving and interfering with instrumental behaviour (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008).

The ruminative response style is hypothesized to be learnt, typically in childhood, either as a result of parents modelling a passive coping style to their children (e.g., via rehearsal and copying of parental response style), or shaped as a consequence of overcritical, intrusive or overcontrolling parenting. The evidence reviewed above concerning early adversity and parenting style and rumination is consistent with this hypothesis. The evidence that socialization into the stereotypical feminine role increases risk for rumination is also consistent with rumination as a learnt style of responding.

Response Styles Theory notes that depressive rumination is more prevalent amongst women than men, and that this individual difference in rumination partially mediates the gender difference in depression. Evidence in support of Response Styles Theory comes from the large body of research indicating that depressive rumination is indeed unhelpful and predictive of psychopathology.

Central to Response Styles Theory is the assumption that rumination is an automatic response conditioned to triggering stimuli such as low mood (Watkins & Nolen-Hoeksema, 2014). This is entirely consistent with conceptualizations of rumination as a habit of thought that often starts automatically and involuntarily (Hertel, 2004). Because rumination occurs frequently, unintentionally, and repetitively in the same emotional context of low or depressed mood, it fulfills the usual definitions of a habit (Verplanken, Friborg, Wang, Trafimow, & Woolf, 2007; Wood & Neal, 2007). Indeed, the leading instrument of depressive rumination (the Response Styles Questionnaire), the measure used in the majority of research reported earlier on the negative consequences of rumination, asks respondents to indicate how frequently they ruminate when “feeling sad, down, or depressed”. Consistent with having a habitual quality, depressive ruminators report that rumination occurs without conscious intent, and that they are unable to control it (Watkins & Baracaia, 2001). Response Styles Theory therefore explains individual differences in rumination as different learnt trait tendencies to engage in rumination. It does not explain why an individual would necessarily ruminate more or less in one situation than another, other than that the rumination is triggered by the occurrence of sad or low mood.

Control Theory (Martin & Tesser, 1996)

The other prominent long-standing model of rumination is based on Control Theory, which conceptualizes rumination as recurrent instrumental thinking about unsatisfactory goal progress (Martin & Tesser, 1996). It proposes that rumination is triggered by the perception of slower than anticipated progress in pursuing a goal, and that rumination will continue until either satisfactory progress is made in reducing the goal discrepancy or the individual disengages from the goal (Martin & Tesser, 1996). As such, in contrast to Response Styles Theory, Control Theory is predominantly an explanation of a proximal cause of episodes of state rumination, rather than of trait rumination.

Within this model, rumination is not necessarily pathological: under some circumstances, it can be adaptive and functional as an instrumental means to highlight and then to address unresolved difficulties and problems, if progress is made in reducing the discrepancy. Control Theory therefore has some overlap with theories that propose that rumination is an adaptive response to difficulties, focused on analytical problem-solving to address the difficulties that cause low mood (Andrews & Thomson, 2009; Bartoskova et al., 2018; Watson & Andrews, 2002). However, to the extent that rumination does not aid in either resolving the goal discrepancy or disengaging from the goal, it is an unsuccessful attempt at problem-solving that is unhelpful insofar as it causes perseveration on the discrepancy in goal progress and intensifies negative affect (Carver, 1996; Watkins, 2008).

Consistent with this account, thoughts relating to interrupted goals persist longer than those associated with resolved goals (Zeigarnik, 1938). Moreover, naturalistic diary and EMA studies have found that unsatisfactory progress on personally important goals is associated with increased rumination on a momentary basis (Gebhardt, Van Der Doef, Massey, Verhoeven, & Verkuil, 2010; Moberly & Watkins, 2010; Verkuil, Brosschot, Gebhardt, & Korrelboom, 2015). In an experimental study, participants asked to focus on an unresolved goal ruminated significantly more and longer over a 30-60 minute period, as indexed by thought probes during a cognitive task, than participants focusing on a resolved goal (Roberts, Watkins, & Wills, 2013). Problematic goal progress can thus be a proximal factor in the onset of an acute episode of rumination.

The nature and motives of the goals held by individuals may also determine both the likelihood of rumination starting and the ability of the individual to make progress on the goal, and, hence, the degree of rumination. The type of goals individuals hold may influence their trait propensity to rumination. An individual can pursue a goal because it feels inherently enjoyable to do so or because they wholeheartedly value it (autonomous reasons) or because they would feel guilty or ashamed if they did not or because pursuing the goal facilitates an extrinsic outcome (controlled reasons). Pursuing goals for controlled reasons is hypothesized to cause internal conflict and problematic rumination (Thomsen, Tønnesvang, Schnieber, & Olesen, 2011). Consistent with

both Control Theory and this hypothesis, relative to autonomous motives, pursuing goals with controlled motives is associated with less goal progress (Moberly & Dickson, 2016), greater trait and goal focused rumination (Thomsen et al., 2011) and prospectively predicts greater rumination over time in adolescents and young adults (Luyckx, Duriez, Green, & Negru-Subtirica, 2017). Similarly, goal pursuit that is focused on avoiding an undesirable state rather than approaching a desired state may make it hard to make sufficient progress towards the goal as it provides little concrete guidance regarding necessary steps and may be hard to definitively resolve: high trait ruminators are more likely to have avoidance goals than low trait ruminators (Thomsen et al., 2011).

Relatedly, having extremely high standards for goals, such as found in perfectionism, may contribute to increased frequency and duration of rumination. Individuals who are perfectionistic have both harder-to-achieve goals and also are less likely to discard their goals, and thus according to Control Theory, they would be hypothesized to experience more frequent and prolonged rumination. Consistent with this, a strong association is found between rumination and perfectionism on self-report measures (Randles, Flett, Nash, McGregor, & Hewitt, 2010; Xie, Kong, Yang, & Chen, 2019), increased perfectionism predicts more state rumination following failure feedback or a stressful social task (Brown & Kocovski, 2014; van der Kaap-Deeder et al., 2016), and rumination is found to mediate the effect of perfectionism on distress concurrently and prospectively (Harris, Pepper, & Maack, 2008; Macedo et al., 2015; Olson & Kwon, 2008; Riviere & Douilliez, 2017; Senra, Merino, & Ferreiro, 2018). The observation that holding socio-cultural beliefs that place an extreme value on happiness and emphasize the importance of avoiding negative emotional states increases rumination (McGuirk et al., 2018) is consistent with Control Theory: goals such as to always be happy / never be unhappy – are unlikely to be easily resolvable, leading to rumination.

In sum, Control Theory offers a highly elaborated account of goal discrepancies as a key proximal mechanism underpinning state rumination, i.e., the incidence of acute episodes of

rumination. It explains why most people experience episodes of rumination at some point in their lives, typically following a loss or set-back (e.g., a bereavement, loss of employment, a relationship break-up). However, it does not fully explain individual differences in the trait tendency towards rumination, other than through differences in goal types and standards. Many people who are not perfectionists become ruminators. Many people experience goal discrepancies without becoming frequent ruminators. It is critical therefore to identify which other mechanisms might interact with goal discrepancies to underpin the trait tendency to unhelpful rumination, and to understand how rumination can become habitual in some individuals.

Habit Development and Goals Discrepancies

A first step to explain both state and trait rumination is to combine the two key mechanisms of habit development (H-) and goal discrepancies (-GO-), as proposed by Watkins and Nolen-Hoeksema (2014) in an integration of the habit and goal-discrepancy theories. This goal-habit account hypothesizes that rumination initially occurs as a goal-directed action in response to goal discrepancies but over time can become a habit automatically triggered by low mood. This shift from deliberate to automatic behaviour is based on theory and evidence that any response repeated frequently that is contingent on the same context can result in the development of a habitual response to that context, consistent with a process of automatic association and classic stimulus–response theories of learning (Wood & Neal, 2007), see Dickinson (1985).

For rumination, this means that individuals experiencing repeated and extended periods of problematic or difficult to resolve goals, associated with low mood, such as those experiencing chronic stress or emotional, physical or sexual abuse and neglect, are likely to learn an association between feeling sad and the initiation of repetitive thinking, such that the individual develops the mental habit of rumination. Thus, insufficient progress on personally important goals can lead to state rumination, but if this occurs repeatedly with rumination contingent on the same stimulus such as low mood, the individual develops rumination-as-a-mental-habit, i.e., the trait tendency to

ruminate. The association of early adverse events, and interpersonal stress and adversity, with increased rumination is consistent with this account: such difficult life circumstances would increase the likelihood of problematic goal discrepancies triggering rumination and being paired with low mood.

This interaction between multiple and prolonged goal discrepancies and associative learning that supports habit formation is proposed as a key process in the development of pathological rumination and a central element within the H-EX-A-GO-N model. This goal-habit approach can explain how state rumination can both be triggered by specific circumstances in all people and how trait rumination can develop in some individuals who have experienced the relevant setting and learning conditions. However, even combining the mechanisms of habit development and goal discrepancies fails to account for the evidence implicating other factors in the onset and maintenance of rumination.

Executive Functioning

A further mechanism that may causally contribute to the initiation and perpetuation of depressive rumination is a deficit in the application of executive functions in the context of negative information (Koster, De Lissnyder, Derakshan, & De Raedt, 2011; EX in the H-EX-A-GO-N model). The efficient application of executive functions is necessary to flexibly process goal-relevant information and suppress or discard task irrelevant information. Executive functions can be fractioned into three major components: shifting, updating and monitoring of representations within working memory (updating), and inhibition (Miyake et al., 2000). Impairments in executive control have been hypothesized to contribute to the difficulties experienced by high trait ruminators in disengaging from repetitive thinking about personal concerns and upsetting events (Joormann & Tanovic, 2015).

Executive functioning deficits could contribute to rumination in several ways. First, difficulties in monitoring, shifting and updating the content of working memory may cause negative

irrelevant material to proliferate in working memory and become disproportionately accessible, leading to a pattern of dwelling on negative content (Joormann, 2010; Joormann & Vanderlind, 2014; Koster et al., 2011).

Second, executive functioning deficits can impair the ability to override habitual ruminative response tendencies. Executive functions are required in order to support goal-directed behaviour and overcome habitual, prepotent and automatic processes that might interfere with this. Reduced executive functioning capacity would make it harder to successfully overcome the tendency to ruminate. In this way, the mechanisms of habit formation and executive functioning are hypothesized to interact in influencing rumination.

There is now a substantial literature evidencing the role of executive functioning deficits in rumination. Impairments in executive functioning are widely documented in individuals with high levels of depressive rumination (De Lissnyder, Derakshan, De Raedt, & Koster, 2011; De Lissnyder, Koster, & De Raedt, 2012; De Lissnyder, Koster, Everaert, et al., 2012; De Lissnyder, Koster, Goubert, et al., 2012; Demeyer, De Lissnyder, Koster, & De Raedt, 2012; Koster et al., 2011; Whitmer & Banich, 2010). There is correlational evidence consistent with rumination-related impairments in inhibitory control (Whitmer & Banich, 2007), working memory updating (Joormann & Gotlib, 2008), and task-switching (De Lissnyder, Koster, & De Raedt, 2012).

Critically, there is also experimental evidence that manipulating executive functioning can influence rumination. One approach to improve executive functioning is cognitive control training (CCT), which is based on the assumption that cognitive control capabilities such as working memory can be improved with repeated practice (Jaeggi, Buschkuhl, Jonides, & Shah, 2011), although there is some debate about its effectiveness (see Shipstead, Redick, & Engle, 2012 for a critical review).

Different tasks have been used to try to train working memory and cognitive control, including the dual n-back task and the adaptive Paced Auditory Serial Addition Task (PASAT; e.g., Siegle, Ghinassi, & Thase, 2007). During the dual n-back task, participants are presented with trials

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consisting of both visual stimuli (e.g., a square in different locations) and auditory stimuli (e.g., spoken letters) and on each trial have to indicate whether each stimuli matches stimuli that appeared n trials previously. During the adaptive PASAT, participants are presented with a stream of audially presented digits and are instructed to indicate the sum of the last two digits, which relies on continuously updating working memory.

An important issue is whether the repeated practice of these cognitive operations only has reliable effects on the specific training task (i.e., rehearsal effects), or leads to improvements in performance on related tasks (“near transfer”) or can lead to improvements on tasks that are of a different nature and paradigm than the training task (“far transfer”). An influence on rumination would be evidence of far transfer. A review of the literature indicates that single-session training typically is not adequate to produce near transfer on cognitive functioning or impact on mood or rumination but that more intensive multiple sessions are needed, and that training is more likely to be impactful if it involves emotional material or occurs in the context of emotional challenge (Koster, Hoorelbeke, Onraedt, Owens, & Derakshan, 2017). It may also be that the effectiveness of cognitive control training depends on the particular paradigm used.

Four studies failed to find evidence that dual n-back training is effective in either improving cognitive control or reducing rumination in healthy adults (De Putter, Vanderhasselt, Baeken, De Raedt, & Koster, 2015), patients with depression or anxiety (Wanmaker, Geraerts, & Franken, 2015), or individuals with high levels of repetitive negative thinking (Course-Choi, Saville, & Derakshan, 2017; Onraedt & Koster, 2014). However, as the CCT did not improve executive functioning in these studies, it is not clear that the studies were able to test the hypothesis that improving executive functioning reduces rumination.

In contrast, several studies have found evidence that CCT influences rumination using an adaptive version of the PASAT, which is experienced as frustrating and hypothesized to be a potential trigger for rumination, such that training involves practice in the application of cognitive control to stay on the task rather than ruminate (Siegle et al., 2014). CCT using the PASAT reduced

trait rumination or maladaptive brooding in depressed patients (Siegle, Ghinassi, & Thase, 2007; Siegle et al., 2014; Vanderhasselt et al., 2015), stress reactivity and brooding to a naturalistic stressor in high ruminating undergraduates (Hoorelbeke, Koster, Vanderhasselt, Callewaert, & Demeyer, 2015), and patients with remitted depression with the CCT delivered by the internet (Hoorelbeke & Koster, 2017). In an unselected student sample examined via EMA in daily life, the effects of the PASAT were limited to reducing spontaneous rumination in low positive affective states (Hoorelbeke, Koster, Demeyer, Loeys, & Vanderhasselt, 2016). There is thus accumulating evidence that CCT using the PASAT reduces rumination in at risk and clinical samples, consistent with the hypothesis that executive functioning plays a causal role in rumination.

In addition to CCT studies, a different paradigm has examined the causal effect of training individuals to recruit executive control resources when processing negative information (Cohen, Mor, & Henik, 2015; Daches & Mor, 2014; Daches, Mor, & Hertel, 2015). Daches and Mor (2014) used an adapted version of the negative affective priming task to train high trait brooders to inhibit negative distractors whilst processing neutral stimuli. Relative to a sham condition, they found a trend towards improved inhibition of negative irrelevant material and reduced brooding. However, a subsequent study (Daches, Mor & Hertel, 2015) failed to replicate these effects. Cohen, Mor and Henik (2015) used an adapted flanker task in which incongruent, but not congruent, trials required participants to recruit executive control resources in order to be able to accurately indicate the orientation of a central stimulus. In the experimental condition, 90% of trials that recruited executive control (incongruent trials) were followed by a negative image, thereby training the recruitment of executive control to be paired with the processing of negative stimuli. Relative to the neutral condition, the training condition reported significantly lower state rumination in response to a subsequent negative recall task. These findings provide preliminary evidence consistent with the hypothesis that difficulties in using executive control to manage negative information partially underpins state rumination. However, further research needs to clarify how robust these effects are

and to test whether manipulating individual differences in executive control causally influences trait rumination.

The experimental evidence to date is broadly consistent with the hypothesis that impaired executive functioning causally contributes to depressive rumination, especially in the context of stress. However, the alternative direction of causality, namely that ruminative preoccupation on negative thoughts will lead to impaired executive control is also supported. There is evidence that induced rumination impairs performance on tasks assessing executive functioning (Philippot & Brutoux, 2008; Watkins & Brown, 2002; Whitmer & Gotlib, 2012), although other studies have failed to find such effects (Roberts, Watkins, & Wills, 2013; Wong & Moulds, 2008).

An as-yet-unresolved issue is whether a general impairment in executive functioning or a more specific difficulty in manipulating negative information underpins rumination. There is some evidence that this association is most clear when the stimuli used are negative, emotional and personally relevant (Beckwe, Deroost, Koster, De Lissnyder, & De Raedt, 2014; Koster, De Lissnyder, & De Raedt, 2013). However, a recent systematic review found that the association between repetitive negative thinking and deficits in executive functioning did not differ between neutral and emotional stimuli (Zetsche, Burkner, & Schulze, 2018), although this study combined negative and positive stimuli, so it could not resolve if there are selective impairments in processing negative content.

Abstract Processing Style

A fourth mechanism identified in the H-EX-A-GO-N model is the processing style adopted (Abstract style) when repetitively dwelling on negative information (Watkins, 2008; A in the H-EX-A-G-ON model). As noted earlier, a key issue any theory of rumination needs to address is delineating what determines whether repetitive thinking on negative content is adaptive or maladaptive and how to explain that sometimes repeated thinking about difficulties can be constructive (Watkins, 2008). This is a particular limitation of both Response Styles Theory and

Control Theory. Response Styles Theory only focuses on detrimental rumination and has no account of how rumination can be helpful, although it views rumination as a failed attempt at problem-solving. Control Theory proposes that rumination is unhelpful when it is not successful at addressing or resolving a discrepancy in goal progress, but does not operationalize in detail what influences this.

There is now extensive evidence that there are different processing styles during rumination, each with distinct helpful versus unhelpful consequences. The processing style characteristic of the phenomenology of depressive rumination (and especially the most pathological “brooding” style) is abstract and analytical. This processing mode involves general, superordinate, and decontextualized mental representations that convey the essential meaning, causes, and implications of goals and events (the “*why*” aspects of an action). In contrast, there is a more adaptive processing style which is more concrete and that involves a focus on the direct, specific, and contextualized experience of an event. This processing mode addresses the details of goals, events, and actions that denote the feasibility, mechanics, and means of “*how*” to do the action (Watkins, 2008).

Experimental manipulations of these thinking styles have robustly found that abstract rumination causes negative consequences relative to concrete rumination. Prompting abstract rumination (with questions like “Why did this problem happen?”) impaired social problem-solving in a recovered depressed group, who performed as well as never-depressed participants in a no-prompt control condition, whereas prompting concrete thinking (“How are you deciding what to do next?”) ameliorated the problem-solving deficit found in currently depressed patients (Watkins & Baracaia, 2002). In depressed patients, compared to abstract rumination, concrete rumination reduced negative global self-judgments (Rimes & Watkins, 2005), improved social problem solving (Watkins & Moulds, 2005), and increased specificity of autobiographical memory recall (Watkins & Teasdale, 2001). Repeated training to think in a concrete mode reduced subsequent emotional reactivity to analogue loss events, relative to training in an abstract mode (Watkins, Moberly, & Moulds, 2008). Abstract thinking is also particularly detrimental during repetitive thought both in

prospective (Ehring, Frank, et al., 2008) and experimental analogue studies for PTSD examining intrusive thoughts (Schaich, Watkins, & Ehring, 2013). Thus, this distinction between different processing styles explains how ruminative thinking about negative events can sometimes function as effective analysis of difficulties that supports problem-solving and emotional recovery (Andrews & Thomson, 2009; Bartoskova et al., 2018; Watson & Andrews, 2002), but also indicates how rumination can go wrong as an attempt at problem-solving, and rather than resolving negative affect, instead prolong and exacerbate it.

Since processing mode was first hypothesized as a key mechanism in the outcomes of rumination (Watkins, 2008), this patterns of findings has been further confirmed and extended across a number of independent research groups. Experiments manipulating abstract versus concrete rumination have found that the abstract rumination has more negative consequences with respect to: (a) Eating disorder risk: abstract rumination increased body dissatisfaction in women after comparing themselves to images of female models (Riviere, Rousseau, & Douilliez, 2018; Spinhoven et al., 2018) and elevated estimates of weight and greater efforts to neutralize stress in participants high in eating disorder psychopathology after they imagined eating fatty food (Rawal, Williams, & Park, 2011). (b) PTSD: abstract rumination increased negative intrusions after watching upsetting videos of accidents as an analogue to PTSD symptoms (Schaich et al., 2013). (c) Negative cognitions: abstract rumination resulted in slower decision-making in high dysphoric participants (Di Schiena, Luminet, Chang, & Philippot, 2013); increased regret after writing about a prior decision (Dey, Joormann, Moulds, & Newell, 2018); and more negative generalization in dysphoric students following a learning phase and more generalization of angry faces to the self (Van Lier, Vervliet, Boddez, & Raes, 2015; Van Lier, Vervliet, Vanbrabant, Lenaert, & Raes, 2014). (d) Psychotic experiences: abstract rumination elevated experience of schizotypic symptoms in the form of self-reported anomalous perceptions of reality (e.g., feeling someone is touching you but nobody there when you look) in university students (Ricarte, Del Rey, Ros, Latorre, & Berna, 2018; Ricarte, Ros, Fernandez, Nieto, & Latorre, 2018). Abstract thinking is also found to be

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particularly detrimental in prospective studies concerning intrusive thoughts (Ehring, Frank, et al., 2008), and relative to healthy controls, alcohol-dependent individuals report similar levels of concrete adaptive repetitive thinking but significantly higher levels of abstract repetitive thinking (Grynberg et al., 2016).

It is hypothesized that the effects of processing mode are due to their differences in sensitivity to contextual and situational detail (Watkins, 2011). Relative to a concrete mode, an abstract mode insulates an individual from the specific context, affording generalizations and inferences across different situations. In some circumstances (following success, positive mood) this can be adaptive, making an individual less distractible and impulsive, supporting consistency and stability of goal pursuit across time, and producing positive generalizations. However, in other contexts, especially when faced with difficulties and low mood, the abstract mode will be maladaptive, making the individual less responsive to the environment, providing fewer specific contextual guides to action and problem-solving, and generating negative overgeneralisations.

The addition of the mechanism of abstract processing within the H-EX-A-GO-N model adds explanatory power. Because the style adopted is important in determining whether repetitive thought is helpful or unhelpful and whether it supports effective problem-solving or not, it provides a more detailed operationalisation of when ruminating about a goal discrepancy would be effective or not. The H-EX-A-GO-N model hypothesizes that the adoption of an abstract processing style interacts with goal discrepancies to make it less likely that repeated thinking facilitates sufficient goal progress, resulting in more prolonged and more frequent bouts of state rumination and negative affect. Furthermore, the tendency to process information in an abstract way could itself be part of the habitual mental style that is learnt in early life. The mechanism of processing style is hypothesized to influence the process of repeated problematic goal discrepancies leading to habit formation: focusing on goal discrepancies with a concrete processing style would promote active problem-solving and thus foster the habit of problem-solving in response to low mood. In contrast, focusing on poor goal progress by analyzing the meaning and implications of events and feelings

will result in the repeated pairing of negative affect about problematic goals with persistent maladaptive rumination, and so the individual will develop an unhelpful mental habit of abstract rumination.

Negative Information Processing Biases

The fifth mechanism hypothesized to be causally involved in the development and maintenance of rumination is a bias in negative information-processing including the tendencies to preferentially attend to negative information, show delayed disengagement from negative content, interpret ambiguous information negatively, and recall more negative information (Everaert, Koster, & Derakshan, 2012; N in the H-EX-A-GO-N model). Such biases would increase the frequency and accessibility of negative thinking, and, thus drive more recurrent and prolonged rumination.

Attentional Biases.

Biases in attention can involve an engagement bias where attention orients towards negative information or a disengagement bias that features difficulties in shifting attention away from negative information. Increased engagement with negative information may increase the likelihood of rumination starting, whilst difficulties in disengaging attention from negative stimuli may make it hard to stop rumination. Many studies find that trait rumination is associated with an attentional bias towards negative stimuli (Beckwe & Deroost, 2016; Sanchez-Lopez, Koster, Van Put & De Raedt, 2019; Donaldson, Lam, & Mathews, 2007; Duque, Sanchez, & Vazquez, 2014; Grafton, Southworth, Watkins, & MacLeod, 2016; Holas, Krejtz, Rusanowska, Rohnka, & Nezelek, 2018; Joormann, Dkane, & Gotlib, 2006; Kaiser et al., 2018; Owens & Gibb, 2017; Pe, Vandekerckhove, & Kuppens, 2013; Southworth, Grafton, MacLeod, & Watkins, 2017; Yaroslavsky, Allard, & Sanchez-Lopez, 2019) and towards symptom-relevant information (Dondzilo, Rieger, Palermo, Byrne, & Bell, 2017), and reduced attention to positive information (Owens & Gibb, 2017; Pe et al., 2013; Yaroslavsky et al., 2019), although see Hilt, Leitzke, and Pollak (2017). Rumination has additionally been shown to be associated with emotion-induced blindness, another index of

attentional bias, capturing interference between spatiotemporal targets and emotional distractors (Onie & Most, 2017).

However, most of these paradigms focus on external visuo-spatial attention, rather than the internal focus of naturally occurring rumination. Moreover, the majority of these paradigms cannot distinguish between biases in engagement or disengagement of attention. Recently, using an adapted version of the dot probe attention paradigm designed to disentangle engagement from disengagement bias, rumination has specifically been associated with deficits in disengaging spatial attention from negative stimuli (Grafton et al., 2016; Southworth et al., 2017).

Prospective studies also suggest that delayed disengagement from negative and positive stimuli may be involved in rumination: the longer that depressed individuals dwelled on negative words during a visual gaze task the greater they reported ruminating in daily life over the subsequent week (Holas et al., 2018), and slower disengagement from positive faces predicted reductions in ruminative brooding over five months, whilst ruminative brooding prospectively predicted delayed disengagement from negative faces in individuals who experienced stressful life events (Sanchez-Lopez, Koster, Van Put, & De Raedt, 2019).

To test the hypothesized causal role of information-processing biases on rumination, these biases need to be experimentally manipulated. Cognitive Bias Modification (CBM) uses systematic practice that introduces training contingencies to modify automatic patterns of processing selectivity, for example, by selectively reinforcing attention towards positive relative to negative words (Cognitive Bias Modification-Attention, CBM-A) (Hertel & Mathews, 2011). Studies of the effects of CBM-A on rumination have had mixed findings. A spatial cueing task used to train depressed and dysphoric participants to direct their attention away from negative words and towards positive words was not effective at changing attentional bias, and, thus did not provide a suitable test of whether shifting attentional bias influences rumination (Baert, De Raedt, Schacht, & Koster, 2010). Yang, Ding, Dai, Peng, and Zhang (2015) found that CBM-A reduced negative attentional bias, depression and rumination in a dysphoric sample, and the change in attentional bias mediated

the change in rumination. However, in a second study with depressed adolescents, CBM-A reduced negative attentional bias and depressive symptoms, but did not reduce rumination (Yang, Zhang, Ding, & Xiao, 2016). In female undergraduates, Dondzilo, Rieger, Palermo, and Bell (2018) found a single session of dot-probe training to attend towards or away from thin images was effective in modifying attentional biases, but did not reduce state rumination to a body-image stressor. In contrast, in a novel paradigm, eye gaze-contingent feedback was used to direct participants' attention towards positive words when generating interpretations. Relative to a control task of freely generating interpretations without feedback, the eye gaze-contingent feedback training increased sustained attention to positive information and reduced state rumination in response to negative images (Sanchez-Lopez, Everaert, Van Put, De Raedt, & Koster, 2019).

Thus, there is mixed evidence with respect to attentional bias playing a causal role in rumination, but there is not robust and reliable evidence for attentional bias causally contributing to rumination. One issue may be that most CBM-A paradigms are focused on external visuo-spatial attention, when rumination is characterized by internal preoccupation and attention focused on mental themes and semantic information. We need to determine whether current positive findings are false positives or whether the failures to influence rumination can be understood with respect to potential moderators of training efficacy such as the dose of training or focus and nature of CBM-A.

Interpretative and Memory Biases.

A bias to interpret ambiguous information negatively and to recall negative memories has also been hypothesized as a maintaining process in rumination (Hertel, 2004). Trait rumination is robustly associated with biases in interpretation (Connolly & Alloy, 2018; Krahe, Whyte, Bridge, Loizou, & Hirsch, in press; Suslow, Wildenauer, & Gunther, 2019; Wisco, Gilbert, & Marroquin, 2014) and memory (Kuo et al., 2012; Mellings & Alden, 2000; Morgan & Banerjee, 2008; Raes, Hermans, & Williams, 2006; Wisco et al., 2014).

There is extensive evidence from prospective (Connolly & Alloy, 2017) and experimental studies that rumination itself causally contributes to negative interpretation biases (Hertel, Mor,

Ferrari, Hunt, & Agrawal, 2014; Hertel & El-Messidi, 2006; Wisco & Nolen-Hoeksema, 2010) and the recall of more negative (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998) and overgeneral autobiographical memories (Watkins & Teasdale, 2001; Watkins, Teasdale, & Williams, 2000). However, experimental studies have also used Cognitive Bias Modification of Interpretation (CBM-I) to test the reverse direction of causality.

CBM-I has tested the causal effect of biases on rumination through repeated practice in reading or listening to ambiguous descriptions of scenarios, which are then resolved in a benign manner, followed by a 'comprehension' test requiring confirmation of the positive resolution. There is emergent evidence that training individuals to make negative resolutions of ambiguous situations increased state rumination following recall of a negative personal experience, relative to training to make benign resolutions (Hertel, Mor, et al., 2014), providing proof-of-principle that negative cognitive bias can influence state rumination.

There is also extensive evidence that worry, another form of repetitive negative thought, is influenced by manipulations of attentional bias and interpretative bias, further suggesting that such negative cognitive biases may play a causal role in the development and maintenance of repetitive negative thought (Hayes, Hirsch, Krebs, & Mathews, 2010; Hayes, Hirsch, & Mathews, 2010; Hirsch et al., 2018; Hirsch et al., 2011; Hirsch, Meeten, Krahe, & Reeder, 2016). Consistent with this, a recent trial found that repeated CBM-I successfully manipulated interpretation biases and reduced trait rumination and worry in individuals with depression or anxiety, with the change in interpretation bias partially mediating the effects of training on rumination (Hirsch et al., 2018).

In sum, there is reasonable evidence to suggest that manipulating interpretative bias, can influence state and trait rumination, consistent with the hypothesis that such biases have a causal effect on rumination. It may be that CBM-A is effective at influencing rumination when it also influences interpretations, as is afforded by the paradigm used by Sanchez-Lopez et al., (2019).

With respect to the H-EX-A-GO-N model, these negative biases can be conceptualized as a learnt habit: as for the abstract processing style, the formation of negative information-processing

biases as a habit increases the likelihood of developing a trait tendency towards rumination.

Negative information-processing biases have been conceptualized as developing through the same associative and instrumental learning processes as habit formation and CBM-I has been shown to change automatic habitual processes (Hertel, Holmes, & Benbow, 2014; Hertel & Mathews, 2011).

The Integrative H-EX-A-GO-N Model

The H-EX-A-GO-N model hypothesizes that each of the five mechanisms outlined contributes to the onset and maintenance of rumination in an additive and synergistic way: with each additional mechanism present, the greater the likelihood that an individual develops a propensity towards trait rumination. It proposes that a combination of multiple elements is necessary for pathological rumination to develop, as each factor on its own is not necessarily sufficient to produce an increase in trait rumination, except perhaps for the mechanism of habit formation when supported by strong distal vulnerability factors. For example, goal discrepancies do not necessarily produce maladaptive rumination, and poor executive control does not always result in elevated trait rumination.

The H-EX-A-GO-N model hypothesizes that the core mechanism for the development of pathological rumination is the formation of unhelpful repetitive thinking as a mental habit (H), reflecting this central characteristic of depressive rumination. This development of mental habits across repeated multiple occasions through rehearsal and conditioning is a central route for the development of depressive rumination. However, the model hypothesizes that this route is facilitated and made much more (or less) likely through the effects of the other mechanisms. Critically, the H-EX-A-GO-N model considers that the five key factors identified are not independent and are highly likely to interact both in habit formation and in the expression and maintenance of rumination. Thus, habit-formation could occur through rehearsal and repetition of a passive self-focused style, as shaped by parenting style (H alone), or from repeated occurrences of problematic goals, resulting from difficult life circumstances (H-GO interaction), especially in the

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context of an abstract processing style (e.g., H-A or H-A-GO interactions) or negative information-processing bias (H-N or H-GO-N interactions), each of which separately or together (e.g., H-A-N) would facilitate unhelpful rumination (Figure 2 panel 1).

Examples of how the mechanisms interact and are not independent of each other include: impaired executive control reduces an individual's ability to inhibit a habitual response (H-EX interaction); shifting processing style is likely to involve executive control and thus deficits in executive control may make it harder to shift to a more adaptive concrete style (EX-A interaction); adopting the abstract processing style makes it harder to successfully problem-solve and end an episode of rumination (A-GO interaction); negative information processing biases are likely to increase the perception of poor goal progress, resulting in more frequent ruminative episodes (GO-N interaction); such biases may also increase the likelihood of the formation of ruminative habits as a consequence of increased attending to, and processing of negative content (N-H interaction). Examples of these interactions are illustrated in Figure 2 (see panel 2). Further, the onset of rumination, whether as a learnt habit or in response to poor goal progress, itself occupies working memory and can impact on executive functioning (Philippot & Brutoux, 2008; Watkins & Brown, 2002; Whitmer & Gotlib, 2012), and exacerbate biases to make negative interpretations and dwell on negative memories (e.g., Hertel & El Messidi, 2006).

As well as learning to dwell on problems as a habit through early experience, individuals can also learn to think in an abstract way or to process information negatively as a mental habit, resulting in habitual abstract, negative thinking in response to negative moods (H-A-N interaction). In turn, this process of habit formation would be facilitated by the presence of poor executive control, making it hard for an individual to shift away from negative thinking, and/or more abstract processing, which would perpetuate the tendency to depressive rumination.

The H-EX-A-GO-N model explicitly links these proximal mechanisms to the identified distal vulnerability factors for rumination (Kingston, Watkins, & O'Mahen, 2013; see Figure 2). The distal vulnerabilities of early adversity and interpersonal stress can drive multiple proximal

factors: (a) such circumstances will necessarily involve periods of prolonged or repeated unresolved goals and provide the setting conditions for rumination to develop as a learnt habit to low mood or stress (Figure 2, panel 1); (b) exposure to multiple or impactful negative experiences has also been hypothesized to develop negative information-processing biases through exposure to environmental contingencies such as extended stressful life events, in which context such biases are at least temporarily adaptive (Clarke, MacLeod, & Shirazee, 2008); (c) the accompanying experiences of loss of mastery and hopelessness can drive the development of more passive and abstract processing (Dweck, Chiu, & Hong, 1995; Dweck & Leggett, 1988). Unhelpful parenting styles and socialisation may directly teach individuals to (a) repeatedly dwell on emotions, increasing the likelihood of developing a ruminative habit, (b) to think in an abstract and analytical manner, (c) to interpret ambiguous information in a more negative way (Figure 2, panel 2). Hence, each environmental vulnerability factor can potentially drive multiple proximal mechanisms. In addition, these environmental vulnerabilities are unlikely to be independent: for example, increased experience of early adverse events is associated with greater likelihood of experiencing later interpersonal stress.

<INSERT FIGURE 2 ABOUT HERE>

Explaining What Makes it so Hard to Break Free of Rumination Once it Starts

The elaborated H-EX-A-GO-N model provides a detailed account of why it is difficult to break free of rumination. First, conceptualizing pathological rumination-as-a-habit partially explains why people find it so hard to stop ruminating despite finding it unhelpful. Once developed, habits are resistant to changes in goals, beliefs, outcomes or intention and difficult to stop because control of the habit is outsourced directly to the contextual cues paired with the past enactment of the behaviour rather than mediated by goals or outcomes (Wood & Neal, 2007). Old habits also tend to reoccur when an individual returns to an old context, or when the individual is fatigued, under stress, or experiencing cognitive load (Bouton, 2000; Schwabe & Wolf, 2011). These are

exactly the situations where more rumination is experienced, as well as the circumstances where rumination is most deleterious and where people most desire to stop ruminating but find it hardest to do so.

In addition, the habit (H) of rumination is likely to be strengthened by negative biases (N) due to increased opportunities for rehearsal, thereby, making it more difficult to override. Prolonged goal discrepancies (GO) can act as a stressor and deplete cognitive resources, making it harder to resist the habit.

This also has implications for treatment. Interventions focused on changing individual's beliefs, attitudes and intentions and providing new information are not typically effective at changing habitual behaviours (Verplanken & Wood, 2006; Webb & Sheeran, 2006) because they do not directly address the pattern of context-response learning. This suggests that many common elements of psychological interventions such as psychoeducation (providing new information), changing goals, and changing beliefs, in isolation, would not be successful at changing depressive rumination, because they do not tackle its habitual quality and alter the context-response learning.

Instead, habit change research indicates two effective strategies: first, to disrupt or remove the environmental factors (e.g., time, place, internal state such as low mood, behavioural routine) that automatically cue the habit (Verplanken & Wood, 2006) and, second, to counter-condition an alternative incompatible response to the cues that trigger the habit, i.e., learning a new more helpful habit to the same cues as the original habit (Hertel, 2004; Marteau, Hollands, & Fletcher, 2012; Wood & Neal, 2007). Therefore, to effectively and permanently reduce rumination, instead of the unhelpful abstract response style to the cueing context (e.g., sad mood), the patient needs to repeatedly practice using an alternative incompatible coping strategy to the same triggering cue, in order to learn a new, more adaptive habit. For example, this alternative strategy could be thought challenging or reappraisal but critically the habit change literature suggests that such strategies need to be frequently repeated in the context of the triggers for rumination to be effective: occasional

cognitive restructuring would not be expected to reduce rumination-as-a-habit. These ideas are returned to in the section on treatments for rumination.

Second, because abstract processing is shown to impair problem-solving, increase overgeneralization and exacerbate emotional responses (Watkins, 2008), it directly interferes with attempts to make satisfactory goal progress. Abstract processing is additionally likely to emphasize the higher-order significance of the goal, thereby enhancing its perceived importance and making it more difficult to abandon (Watkins, 2011). Trying to ruminate out of a problem in an abstract manner is therefore likely to fail, and will only serve to make the problem more salient and prominent, trapping the individual in a cycle of rumination.

Third, poor executive control will impair abilities to shift processing away from ruminative content in working memory. This has implications both for the ability to break out of an episode of habitual rumination once it has started, but may also increase the likelihood of developing rumination as a habit in the first place. Individual differences in executive functioning (EX) help to explain why some individuals may find the ruminative habit especially difficult to break free from once established, even in the absence of a significant stressor. In addition, during a period of chronic stress and difficult-to-resolve goals, poor executive control and reduced cognitive flexibility may limit an individual's repertoire of possible responses to a given scenario, impair reversal learning if that response is no longer effective, and increase the likelihood that unhelpful responses (such as abstract thinking or negative biases) are instigated and repeated as a default response to the same or similar contexts.

Unresolved Issues and Further Tests for the H-EX-A-GO-N Model

The evidence reviewed is supportive of the hypothesis that each of the mechanisms identified within the H-EX-A-GO-N model can influence state and trait rumination as individual factors. Nonetheless, a key element of the model that requires further testing is the hypothesized interaction of the mechanisms in driving rumination: whilst some interactions are well-established

(e.g., poor executive control limiting inhibition of habits; rumination impairing executive functioning), others require further testing, especially regarding how the mechanisms interact to influence the onset and maintenance of rumination. For example, one key hypothesis to-be-tested is that the combination of prolonged goal discrepancies and the adoption of an abstract processing style will result in increased individual differences in pathological habitual rumination, and to a greater extent, than the presence of each factor alone. Similarly, the H-EX-A-GO-N model predicts that the addition of each factor would increase the likelihood of pathological rumination developing as an individual difference. In practice, this could be tested by examining the ability of the combined factors to predict the concurrent and subsequent manifestation of trait rumination as a repetitive and perseverative response style: the addition of each factor and respective interaction terms would be expected to account for a greater variance of trait rumination explained in a prospective longitudinal design.

A particularly important area within the model that requires further testing concerns the habit formation element of the model. Whilst the conceptualisation of pathological rumination as a habit is consistent with the aetiological distal factors predicting rumination, and directly maps onto the phenomenology of depressive rumination as automatic, hard-to-control, occurring outside of awareness and without effort and to consistent cues such as low mood, the habitual nature of rumination has not been experimentally unpacked. In particular, experimental and learning paradigms that have been developed to determine whether a process is habitual (automatic) or goal-based (controlled) could perhaps be usefully applied to the study of rumination. One approach involves process-dissociation procedures in which prior task learning and task instructions can either act together or in opposition to distinguish automatic from controlled processes (Hertel, Holmes, et al., 2014; Hertel, Maydon, Ogilvie, & Mor, 2018).

Another approach involves the outcome devaluation paradigm consisting of (a) a learning phase used to train that a particular response (e.g., a particular choice or lever press) produces an outcome (e.g., receipt of food); (b) devaluation of the outcome (e.g., satiation for a food outcome,

or aversion conditioned to the outcome) and (c) an extinction phase, in which the outcome is no longer presented following the response. After only brief training, the trained response is reduced following the devaluation indicating that it was goal directed, but after extensive training, the behaviour continues even after the devaluation, indicating that it is now habitual (de Wit & Dickinson, 2009; Neal, Wood, Wu, & Kurlander, 2011; Tricomi, Balleine, & O'Doherty, 2009), although see also de Wit et al. (2018). For a goal-based behaviour, performance of the behaviour reduces, but this reduction does not occur so much for habitual behaviours, which are insulated from goal change. Habitual accounts of rumination hypothesize that rumination initially started because it resulted in desired outcomes (i.e., goal-driven), but that because of over-learning, it has become a habit and therefore even when the desired outcome is devalued, the ruminative response will persevere.

However, to date, these paradigms have not been used to study rumination-as-a-habit and establish what can facilitate or impair the development of habitual rumination. This in part reflects methodological difficulties in adapting these paradigms to naturally occurring rumination: typically these paradigms often train an arbitrary response in the lab to examine habitual versus goal-based learning, but this is more difficult for spontaneous rumination. In addition, a meaningful way to devalue rumination or its consequences as an outcome is required. Nonetheless, this is likely to be an important area for methodological developments, for example, by examining sensible analogues to ecologically valid rumination, such as training increased recall of negative words/themes in a linked chain and then extinguishing them in the lab.

Further, there is a growing literature indicating that there are individual differences in the propensity to form habits (habit propensity), with increased tendency to form habits implicated in the development of certain disorders such as Obsessive-Compulsive Disorder (Gillan et al., 2014; Gillan et al., 2011; Robbins, Gillan, Smith, de Wit, & Ersche, 2012). This propensity is assessed by a computerized outcome-devaluation task in which points or money can be collected, and in which participants learn different stimulus-response-outcome-contingencies. After the learning phase,

some outcomes are devalued such that they lead to the subtraction of points. Individual's ability to selectively respond to stimuli that are still associated with valuable outcomes and to suppress responses to now devalued outcomes is an index of the balance between goal-directed and habitual control (de Wit et al., 2012). Such a habit propensity could provide a distal and presumably biologically underpinned vulnerability factor for pathological rumination: A specific hypothesis arising from the Habit formation element of the H-EX-A-GO-N model is that this propensity to develop habits will predict increased trait rumination, especially in the context of the other distal and proximal mechanisms.

The H-EX-A-GO-N model also has implications for treatment. First, it predicts that interventions that target each of the five identified mechanisms should be effective in reducing rumination. Second, the model predicts that treatments that target multiple mechanisms simultaneously will be more efficacious. Finally, it predicts that it may be possible to tailor interventions for rumination, such that targeting the particular H-EX-A-GO-N factors present in an individual may produce more efficient reduction in rumination.

Treatment of Rumination

All of the consequences of rumination reviewed earlier highlight the potential value of successfully targeting rumination. Successfully targeting rumination could enhance the efficacy and potency of psychological treatments and improve patient outcomes, especially for patients with co-morbid anxiety and mood disorders, and for those who are more chronic, complex, and less responsive to treatments. Indeed, patients with co-morbid anxiety and depression often have the poorest response to treatment, and rumination has been proposed as an endophenotype of treatment-resistant patients (Mennin & Fresco, 2013).

What treatments may be most effective in treating rumination?

Because rumination involves repetitive negative thought and CBT challenges negative thoughts and increases rewarding behaviours, CBT is indicated to reduce rumination. However,

until recently, many major trials had not included rumination as an outcome measure, which made it difficult to assess the potential efficacy of standard treatments such as CBT in reducing rumination. As noted earlier, there is evidence indicating that CBT may not be effective at reducing rumination in depression (Jones et al., 2008; Schmaling et al., 2002), although other studies have found a positive effect. For example, in adolescents, the addition of CBT to antidepressant treatment as usual was found to produce a greater reduction in rumination, albeit in a small sample (Wilkinson & Goodyer, 2008). A number of treatments have been developed to specifically target rumination, including within a CBT framework (rumination-focused CBT Watkins, 2015; Watkins, 2016), and approaches building on the research linking cognitive biases and executive function deficits with rumination (CBM and CCT approaches).

Rumination-focused CBT (RFCBT).

RFCBT is theoretically informed by the evidence reviewed earlier both that pathological rumination can be conceptualized as learned habitual behaviour that develops through rehearsal and negative reinforcement, and that there are helpful and unhelpful styles of processing within rumination (Watkins, 2008; Watkins, 2015; Watkins & Nolen-Hoeksema, 2014). Whilst still grounded within the core principles and techniques of CBT for depression, RFCBT combines a number of important novel elements. First, RFCBT incorporates the functional–analytic and contextual approach developed in Behavioural Activation (Dimidjian, Barrera, Martell, Munoz, & Lewinsohn, 2011; Jacobson, Martell, & Dimidjian, 2001). Functional analysis is used to examine how, when and where rumination does and does not occur, and its antecedents and consequences, to formulate its possible functions and to make plans that systematically reduce or replace it. This approach explicitly targets rumination-as-a-habit by identifying antecedent cues to rumination (i.e., the triggers to the habit), controlling exposure to these cues, and by repeatedly practising alternative helpful responses to these cues, in order to counter-condition an alternative, more helpful habit.

Second, RFCBT uses functional analysis, imagery, behavioural experiments, and experiential approaches to shift a patient from the unconstructive abstract processing style to the

constructive concrete style. Functional analysis is used to discriminate between helpful versus unhelpful thinking about difficulties and to coach patients towards more helpful thinking. Patients use directed imagery to recreate previous mental states when a thinking style directly counter to rumination was active, including concrete thinking, memories of being completely absorbed in an activity (e.g., 'flow' experiences), and experiences of increased compassion to self or others. In the H-EX-A-GO-N model, RFCBT therefore tackles habit change (H), abstract processing (A), and negative information-processing (N) directly, and goal discrepancies (GO) indirectly by seeking to improve problem-solving.

A randomized controlled trial (RCT) allocated forty-two patients with medication-refractory residual depression to treatment-as-usual (TAU) alone or to TAU plus 12 sessions of individualised RFCBT (Watkins et al., 2011). TAU consisted of ongoing antidepressant medication and out-patient clinical management. TAU plus RFCBT significantly reduced rumination and depression relative to TAU alone (remission rates: TAU 21%; TAU+RFCBT 62%), comparing favourably to adding standard CBT to TAU for the same patient group (25%) (Paykel et al., 1999). Change in rumination mediated the effect of treatment condition on depression, although this was only measured concurrently, preventing conclusions about causal direction. An independent trial confirmed that group-delivered RFCBT improved depressed mood and reduced rumination relative to a waiting list condition in patients with residual depression, with treatment gains maintained over one year follow-up (Teismann et al., 2014).

A further trial comparing RFCBT versus assessment only for 33 adolescents with a history of Major Depressive Disorder found that RFCBT significantly reduced rumination and depressive symptoms relative to control (Jacobs et al., 2016). fMRI scans pre- and post-intervention examining resting state functional connectivity of the DMN found that adolescents who received RFCBT demonstrated significant decreases in connectivity between the left PCC and the right inferior frontal gyrus (IFG, a node of the CCN) and bilateral inferior temporal gyri (ITG), indicating decreased functional connectivity between the DMN and CCN. Degree of change in connectivity

was correlated with changes in self-reported depression and rumination (Jacobs et al., 2016). These results suggest that the DMN and CCN may begin to function more independently as skills for reducing rumination are learned.

Because rumination has been implicated as a risk factor for depression onset, RFCBT has also been tested as a preventative intervention for depression and anxiety (Topper, Emmelkamp, Watkins, & Ehring, 2017). Group and internet RFCBT were compared to a waiting list control group in 251 Dutch adolescents and young adults with elevated rumination but without current major depression or anxiety disorder in a high-risk prevention design. Relative to the waiting list control, both RFCBT interventions significantly reduced worry, rumination, anxiety and depression at post-intervention and one year follow-up, and halved one-year incidence rates of major depression and generalized anxiety disorder, as indexed by standard cut-offs on self-report measures.

A partial replication tested the efficacy of internet RFCBT versus usual practice control in UK undergraduates at risk for depression because of elevated rumination and found that relative to control, guided online RFCBT significantly reduced the subsequent onset of major depression, as assessed by structured diagnostic interview, especially for those young people reporting higher levels of stress at baseline (Cook, Mostazir, & Watkins, 2019). Taken together, these results provide proof-of-principle that rumination increases risk for the onset of major depression and generalized anxiety disorder, and that targeting rumination has transdiagnostic preventive benefit.

Cognitive bias modification (CBM).

One specific element of RFCBT, namely encouraging a more concrete processing style to shift away from maladaptive rumination, has also been tested as an independent treatment within a repeated CBM approach. CBM training is a good candidate to change rumination-as-a-mental habit, because it involves the same associative and instrumental learning processes as habit formation and has been shown to change automatic habitual processes (Hertel, Holmes, et al., 2014; Hertel & Mathews, 2011). In this study, concreteness training CBM was derived from the processing mode

research, and involved repeated practice at focusing on the specific details, context, and sequence (e.g., asking “How?”) of difficult events using audio-recorded mental exercises in response to identified warning signs for rumination. Providing proof-of-principle, an RCT found that training dysphoric individuals to be more concrete when faced with difficulties for one week reduced depression, anxiety, and rumination relative to a no-treatment control and a credible attention placebo control (Watkins, Baeyens, & Read, 2009). A further RCT of 121 patients with major depression recruited in primary care compared TAU provided by general practitioners (half received antidepressants), TAU plus guided self-help concreteness training, or TAU plus guided self-help relaxation training. Both training conditions were matched for rationale, therapist contact, identification of warning signs and daily practice using an audio-recording over 6 weeks, and delivered via one face-to-face session and three 30-min telephone sessions. Both concreteness training and relaxation training were superior to TAU in reducing depression, concreteness training was superior to relaxation training in reducing rumination, and outperformed relaxation training at reducing depression when training was self-reported to be habitual (Watkins et al., 2012).

Other CBM approaches have focused on training information processing away from a negative cognitive bias to a more positive bias, whether in attention or interpretation of ambiguous situations. Emerging evidence indicates that such CBM training, when delivered over repeated sessions, can reduce rumination in clinical populations, consistent with hypothesis that information-processing biases influence rumination. A double-blind study of CBM-A extended to 8 sessions over 2 weeks successfully reduced depression and rumination relative to placebo training and no-training in undergraduate students with elevated depressive symptoms, with rumination mediating the effect of attention bias change on subsequent depressive symptoms (Yang et al., 2015). A recent trial found that training 157 volunteers with either major depression and generalized anxiety disorder to make positive interpretations of ambiguous situations across 10 internet-delivered sessions reduced worry and rumination and depressive symptoms, relative to a control condition (Hirsch et al., 2018). Interestingly, there was no difference in treatment effects for CBM-I whether

or not there was an attempt to prime rumination before the training by instructing participants to think about rumination topics. These findings suggest that there may be value in further considering CBM approaches for rumination, although some caution may be warranted given that recent meta-analyses have not found significant treatment effects for CBM approaches on anxiety and depression (Cristea, Kok, & Cuijpers, 2015) (although this did not include the Yang et al., 2015 or Hirsch et al., 2018 studies) and we need robust replication in patients recruited and treated in clinical settings before we can consider CBM as an effective treatment.

Paralleling some of the principles of RFBCT and concreteness training, such as learning an incompatible response to rumination and repeated practice at experiential exercises, competitive memory training (COMET) involves repeatedly reliving the experience of memories that act counter to rumination such as memories of times in which the individual was able to step back and distance themselves from emotional situations or to adopt an attitude of acceptance to a difficult situation. In a clinical trial, COMET plus TAU, mainly pharmacotherapy, significantly improved rumination and depression relative to TAU alone in 93 older adults with depression (Ekkers et al., 2011).

Metacognitive therapy and Mindfulness-based CBT.

Metacognitive therapy is based on the hypothesis that rumination is initiated by positive metacognitive beliefs about the usefulness of rumination and then exacerbated by negative metacognitive beliefs about the negative consequences of rumination (Wells, 2009). Metacognitive therapy focuses on challenging these metacognitive beliefs and trains patients to disengage their attention from self-focus to external stimuli. To date, metacognitive therapy has only been examined in two case series for patients with treatment-resistant depression and persistent depressive disorder, with positive within-subject change but without any randomisation or control condition (Wells et al., 2012; Winter et al., 2019) and a small RCT against waiting list control, where it significantly reduced depression and rumination and maintained improvements over one

year (Hjemdal et al., 2019). Metacognitive therapy has yet to be compared to an active control or active treatment.

Another treatment hypothesized to reduce rumination is Mindfulness-based CBT (MBCT). MBCT is a psychosocial group-based relapse prevention program that incorporates meditational practice within the framework of CBT principles as a means to increase resilience against depression (Segal, Williams, & Teasdale, 2002). A key element is mindfulness practice in which participants learn experientially to maintain their attention to their breath, thoughts, and feelings, and to hold such experiences in awareness, in a non-judgmental and accepting way. These mindfulness skills are hypothesized to enable individuals to develop alternative responses to negative thoughts and feelings, and, thereby, to step out of habitual patterns of rumination. MBCT has been demonstrated to be an effective relapse prevention treatment for individuals with three or more episodes of depression (Piet & Hougaard, 2011).

Mindfulness approaches reduce rumination in experimental analogue studies (Feldman, Greeson, & Senville, 2010) and in trials of MBCT for patients with a history of recurrent major depression, relative to waiting list control (Geschwind, Peeters, Drukker, van Os, & Wichers, 2011) and treatment-as-usual (van Aalderen et al., 2012), with the reduction of depressive symptoms mediated by decreased levels of rumination (van Aalderen et al., 2012). However, in other trials, MBCT did not reduce rumination more than continuation antidepressants (Kuyken et al., 2008; Kuyken et al., 2015).

Cognitive control training (CCT).

Based on the evidence that impaired executive functioning may be implicated in rumination, CCT has been examined as an intervention. One version uses two tasks delivered by computer: an Attention Control Training intervention (Wells, 2000) in which participants practice directing their attention to multiple distinct sounds, requiring the use of selective attention processes, and an adaptive variant of the PASAT. CCT was first tested as an adjunctive therapy for 2 weeks in addition to TAU versus TAU alone with severely depressed individuals in a day hospitalization

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program (Siegle et al., 2007). Participants in the CCT group demonstrated a significant reduction in depression and rumination symptoms relative to the participants who received TAU. A further trial found that 3 sessions of CCT over 2 weeks reduced depression significantly more than a matched control involving peripheral vision training in community volunteers with elevated depression symptoms, although rumination was not assessed (Calkins, McMorran, Siegle, & Otto, 2015; Siegle et al., 2007). Similarly, CCT based on a more extensive adaptive PASAT training procedure (10 sessions over two weeks), relative to active controls, reduced trait rumination, depression and stress reactivity in high ruminating undergraduates (Hoorelbeke et al., 2015) and in patients with remitted depression (Hoorelbeke & Koster, 2017).

Neurostimulation approaches such as transcranial Direct Current Stimulation (tDCS), which can directly modulate prefrontal excitability, have been used to increase activation in brain areas implicated in working memory, such as the dorsolateral prefrontal cortex (DLPFC). A recent study compared the PASAT CCT plus active tDCS of the DLPFC versus the CCT with sham tDCS in patients with major depression, and found that in both groups, depressive brooding was reduced, and extent of reduction in brooding was associated with improvement in working memory (Vanderhasselt et al., 2015). However, there was no effect of tDCS and in the absence of a control training group, it is not possible to infer the causal effect of the PASAT training.

Self-Systems Therapy (Strauman & Eddington (2017)).

Consistent with the role of problematic goals in rumination and depression, there is preliminary evidence from two small RCTs that for individuals with specific self-regulatory deficits, a structured therapy that specifically targets goal pursuit (Self-Systems Therapy, Eddington, Silvia, Foxworth, Hoet, & Kwapil, 2015; Strauman & Eddington, 2017; Strauman et al., 2006) is superior to cognitive therapy in treating depression, and that rumination may be an important mechanism of action in these effects (Jones, Papadakis, Orr, & Strauman, 2013). However, future research and larger RCTs are needed to establish how robust these findings are.

Are treatments that specifically target rumination necessary to reduce rumination?

A recent systematic review examined whether those treatments for depression that specifically targeted rumination or repetitive negative thought (RNT) including those described above, have a specific effect on rumination and produce better outcomes than treatments that do not specifically target rumination (Spinhoven et al., 2018). This meta-analysis of 36 randomized controlled trials involving 3307 patients found that treatments intended to target rumination noted above (in particular rumination-focused CBT $g = 0.76, p < .01$; CCT, $g = 0.62, p < .01$; MBCT, $g = 0.44, p < .01$), as well as CBT ($g = 0.57, p < .01$), had medium-sized effects on reducing rumination relative to control arms, and had significantly larger effect sizes than other treatments including antidepressant medication. The effects on rumination post-treatment were only significantly associated with reductions in depression severity in rumination-focused CBT. It was concluded that “in particular RNT-focused CBT may have a more pronounced effect on RNT than other types of interventions” (Spinhoven et al., 2018, p. 71). This may be an under-estimate of the potential effects of RNT-focused CBT. Several of the trials included in the meta-analysis tested variants of interventions (e.g., concreteness training; (Mogoase, Brailean, & David, 2013)) that diverged from the original treatment protocol in major ways (e.g., not using autobiographical events; no face-to-face training; not using audio-recorded exercises; different prompt questions; written exercises rather than imagery), potentially weakening overall effect sizes. Critically since this review, a further trial found that group-delivered RFCBT significantly outperformed group-delivered CBT in reducing acute depression in 121 adults with major depression (Hvenegaard et al., 2019), strengthening the evidence for rumination-focused interventions.

When considering the effect of interventions on rumination, it is also important to note that the habit account proposes that any interventions that improve mood state will temporarily disrupt depressive rumination by removing the context (i.e., low mood) that triggers the ruminative habit and, thereby, limit its expression. However, only interventions that modify the underlying habit will lead to long-standing reductions in rumination (Watkins & Nolen-Hoeksema, 2014). Without changing the underlying habit, once the triggering context returns during another period of

depressed mood or stress, the tendency to ruminate would be reactivated, increasing vulnerability to another episode of depression. Many psychological interventions improve symptoms in the short term through positive expectancy, remoralization, increased activation, and therapist support, without necessarily directly targeting the ruminative habit: as such, they may appear to reduce self-reported rumination post-intervention without changing its habitual quality. The most commonly used self-reported measure of rumination (Response Styles Questionnaire; Nolen-Hoeksema, 1991) only assesses the frequency of rumination in response to low mood (i.e., to a common trigger) but does not assess other key dimensions of a habit such as its automaticity, involuntariness, and goal insensitivity. Future studies therefore need to evaluate the longer-term impact of treatment on rumination and relapse/recurrence, as well as include measures that capture all dimensions of the habitual quality of pathological rumination.

In sum, there is emerging evidence of effective interventions for rumination, particularly those that explicitly target rumination itself (Spinhoven et al., 2018). The evidence from treatment trials is broadly consistent with the predictions arising from the H-EX-A-GO-N model: interventions targeting rumination-as-a-Habit, EXecutive functioning, Abstract processing, and Negative biases are all efficacious at reducing rumination. The key next step in enhancing interventions and in testing predictions arising from the H-EX-A-GO-N model is to test the potential positive interactive effects of combining rumination-focused interventions. For example, the model predicts that on average, a combination of RFCBT and CCT would outperform either individually in reducing rumination by targeting multiple relevant mechanisms.

Further Research and New Directions

In addition to the specific hypotheses arising from the H-EX-A-GO-N model that require testing, there are several further areas of merit for further exploration in rumination research. First, whilst self-report will necessarily always be critical to the study of rumination because it is essentially an experience of internal subjective consciousness, the development of well-validated

implicit behavioural and psychophysiological incidences of rumination would be advantageous to the field. Such indices, once established as analogue proxies highly correlated with self-reported rumination, can reduce some of the limitations of current measures, most notably the potential for demand and expectancy effects, the reliance on accurate awareness and recall, and the reflexive nature of studying rumination – i.e., attempting to be aware and report on rumination may increase or decrease rumination. In addition, such indices could enable more fine-tuned investigation of underlying cognitive mechanisms, and potentially support enhanced interventions, for example, “just-in-time” interventions, in which useful strategies are suggested to an individual at risk for rumination based on these indices before a bout of rumination begins. One potential index is based on eyetracking, with evidence that pupillary dilation reflecting increased cognitive-emotional processing may be associated with ruminative processing (Siegle et al., 2015; Siegle, Steinhauer, Carter, Ramel, & Thase, 2003; Siegle, Steinhauer, Friedman, Thompson, & Thase, 2011; Stone et al., 2016) and increased dwell time on emotional content such as faces (Hilt et al., 2017).

Second, there may be merit in further investigation of the potentially bidirectional relationship between rumination and impaired parasympathetic flexibility. As well as evidence that rumination impacts on HRV, interventions designed to directly impact HRV, such as transcutaneous vagal nerve stimulation can influence spontaneous repetitive negative thinking (Burger, Van der Does, Thayer, Brosschot, & Verkuil, 2019). Interventions that influence parasympathetic activity including biofeedback and breathing exercises may thus have potential to influence rumination.

Third, we note that the commonalities and overlap between different forms of repetitive negative thought has led to the hypothesis that the different forms of repetitive negative thought share the same underlying mechanisms but may differ in their specific content and concerns, e.g., rumination being more past-and loss-focused and worry being more future-and threat-focused (Ehring & Watkins, 2008; Watkins, 2008). Because of this review’s focus on evidence with respect to (depressive) rumination, we can only justify our conclusions and H-Ex-A-Go-N model with

respect to rumination. However, this hypothesis suggests that many of these conclusions and the model may be relevant to the understanding and treatment of other repetitive negative thought such as worry, and this is worthy of further investigation.

Conclusion

The current review confirmed that depressive rumination has multiple negative consequences: (a) it exacerbates psychopathology across a range of mental health diagnoses including anxiety and depression, psychosis, insomnia and impulsive behaviours by magnifying and prolonging existing negative mood states and associated negative thinking, interfering with problem-solving and instrumental behaviour, and reducing sensitivity to changing contingencies and context; (b) it acts as a therapy-interfering behaviour, limiting the efficacy of psychological interventions; (c) it exacerbates and maintains physiological stress responses including reduced HRV and exaggerated cardiovascular stress responses. An elaborated theoretical model (H-EX-A-GO-N) builds on existing theories and outlines how five underlying proximal mechanisms (Habit development, EXecutive control, Abstract processing, GOal discrepancies, Negative bias) interact to explain the onset and maintenance of rumination, and especially why it is so hard to stop. These proximal mechanisms provide a pathway by which known biological and environmental risk factors for rumination increase the likelihood of habitual depressive rumination developing. There is emerging evidence for effective psychological interventions to reduce rumination, particularly those interventions that explicitly target rumination and tackle these identified proximal mechanisms.

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AUTHOR DECLARATION TEMPLATE

One author (EW) has developed a variant of cognitive-behavioural therapy designed to target rumination (rumination-focused CBT), and receives royalties from Guilford Press for a published treatment manual for this intervention and receives remuneration for providing training workshops in this intervention.


We wish to confirm that there are no other known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.


We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

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