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Rumination in migraine: Mediating effects of brooding and reflection between migraine and psychological distress

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Objective: The relationship between migraine and psychological distress has been consistently reported in cross-sectional and longitudinal studies. We hypothesised that a stable tendency to perseverative thoughts such as rumination would mediate the relationship between migraine and psychological distress.
Design and Main Outcomes Measures: Self-report questionnaires measuring depressive rumination, current psychological distress and migraine symptoms in two independent European population cohorts, recruited from Budapest ($N = 1139$) and Manchester ($N = 2004$), were used. Structural regression analysis within structural equation modelling was applied to test the mediational role of brooding and reflection, the components of rumination, between migraine and psychological distress. Sex, age and lifetime depression were controlled for in the analysis.
Results: Migraine predicted higher brooding and reflection scores, and brooding proved to be a mediator between migraine and psychological distress in both samples, while reflection mediated the relationship significantly only in the Budapest sample.
Conclusions: Elevated psychological distress in migraine is partially attributed to ruminative response style. Further studies are needed to expand our findings to clinical samples and to examine how rumination links to the adjustment to migraine.

Keywords: migraine; rumination; brooding; depression; cross-cultural; psychological distress

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Introduction

Migraine, as a primary headache disorder with a prevalence of approximately 14% in Western societies (Burch, Loder, Loder, & Smitherman, 2015), is associated with significant disability (Lipton, 2011). Migraine attacks comprise headaches, with a combination of nausea, vomiting and photophobia or phonophobia (Silberstein, 1995), and some individuals may also experience aura symptoms (transient focal neurological symptoms) just before or together with their headaches (Hansen et al., 2012). During the attacks, physical activity usually worsens the symptoms, and cutaneous allodynia (Lipton et al., 2008) and/or olfactory hypersensitivity or osmophobia (Kelman & Tanis, 2006) may occur accompanying the pain. As the headache progresses, other affective and cognitive symptoms (such as depressive mood, irritability and problems with concentration or attention) and autonomic symptoms (such as frequent urination and diarrhoea) are experienced (Silberstein, 1995). Given these symptoms of migraine attacks, it is not surprising that migraine has a substantial impact on quality of life. Indeed, reduced quality of life has been reported by people with either episodic (Park, Shin, Kim, & Lee, 2008) or chronic forms of migraine (Lanteri-Minet, Duru, Mudge, & Cottrell, 2011).

Prevalence of psychological distress – defined as depressive mood (Radat et al., 2008), mood disorders (Breslau & Andreski, 1995; Breslau, Davis, Schultz, & Paterson, 1994; Breslau, Lipton, Stewart, Schultz, & Welch, 2003; Breslau et al., 2000; Modgill, Jette, Wang, Becker, & Patten, 2012), anxiety (Radat et al., 2008) or anxiety disorders (Black, Fulwiler, & Smitherman, 2015; Ligthart, Gerrits, Boomsma, & Penninx, 2013; McWilliams, Goodwin, & Cox, 2004) – is elevated in migraine. Psychological distress negatively affects quality of life (Pompili et al., 2009) and increases the disability reported by patients. In addition, psychiatric comorbidity may affect treatment of migraine (Guidetti et al., 1998). Therefore, identifying factors that contribute to psychological distress in migraine is an important issue. Previous studies addressing psychological factors have focused mainly on pain-related variables such as pain coping (Biagianti, Grazzi, Usai, & Gambini, 2014), pain catastrophising (Goli, Asghari, & Moradi, 2014) and pain acceptance (Chiros & O'Brien, 2011). Since stress is named most frequently as a potential trigger of attacks (Hashizume et al., 2008; Houle et al., 2012), and migraine is considered to be characterised by a maladaptive stress response (Borsook, Maleki, Becerra, & McEwen, 2012), general coping strategies have been also addressed by studies (Chan & Consedine, 2014; Radat et al., 2008). However, these studies have not assessed individual differences in psychological functioning that are reliably associated with psychopathologies and can worsen stress response.

Based on a meta-analytic review, Aldao and colleagues (Aldao, Nolen-Hoeksema, & Schweizer, 2010) concluded that trait depressive rumination – a stable tendency to focus passively and repetitively on feelings related to distress and low mood, and its causes and consequences (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008) – is strongly associated with overall psychopathology, and more specifically with depression and anxiety. Furthermore, rumination has been documented to affect psychological and physiological reactivity to stress (Huffziger et al., 2013; Zoccola & Dickerson, 2012) and recovery from stress (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Johnson, Key, Routledge, Gerin, & Campbell, 2014; Key, Campbell, Bacon, & Gerin, 2008). Based on these findings, we hypothesised that inter-individual differences in rumination would explain the occurrence of psychological distress in migraine.

Moreover, there are findings indirectly suggesting that rumination may be elevated in migraine. People with migraine try to avoid potential triggers and change their work commitment or social plans (Hu et al., 2010), restricting social and work life in a very similar way to when having an acute migraine attack (Ruiz de Velasco, Gonzalez, Etxeberria, & Garcia-Monco, 2003). Therefore, migraine increases the occurrence of situations that impair goal pursuit, and increases the discrepancy between one's current state (e.g. not being able to meet friends) and desired state (e.g. celebrating a friend's birthday in a pub). According to one of the prominent models of rumination (Martin & Tesser, 1996), the discrepancy between one's current state and desired state can induce rumination (e.g. Why can't I get better? Why do I have migraine attacks?) and increase negative emotions (Roelofs et al., 2007). This model would predict that migraineurs will report higher ruminative tendencies than control subjects. It is worth noting that Soo and colleagues (Soo, Burney, & Basten, 2009) explain the role of rumination in chronic illnesses in the framework of the Stress-Reactive Rumination model (Robinson & Alloy, 2003) and Response Styles Theory (Nolen-Hoeksema, 1991) and suggest that rumination can exert its effects in two steps. First, chronic illness as a stressor may induce stress-related rumination (e.g. ruminative thoughts about the illness, its causes and consequences). Second, once depressed or anxious mood has developed, rumination maintains negative mood or negative emotional states.

As discussed above, rumination in migraine may be a consequence of chronic illness. However, it is also possible that people who are susceptible to, and develop migraine might have higher premorbid trait rumination. Neuroticism has been found to be associated with perseverative cognitions (Roelofs, Huibers, Peeters, Arntz, & van Os, 2008), with rumination acting as a mediator between neuroticism and depression/anxiety (Roelofs et al., 2008). High scores on neuroticism among people with migraine have also been reported (Breslau & Andreski, 1995; Merikangas, Stevens, & Angst, 1993). It is therefore plausible to hypothesise higher rumination among migraineurs.

It has been argued that rumination is a multidimensional construct, and some components might be adaptive while others are maladaptive (Watkins, 2004). For example, Treynor and his co-workers (2003) made a distinction between brooding and reflective pondering. Brooding is considered to be the more maladaptive facet of rumination, defined as a tendency to passively dwell on negative emotions (Treynor et al., 2003). Brooding was associated with concurrent and follow-up depression scores (Cox, Funasaki, Smith, & Mezulis, 2012). Reflection (or reflective pondering), defined as a more purposeful self-reflective response to understanding and solving problems, was also associated with concurrent depression, but did not predict depression prospectively (Cox et al., 2012; Treynor et al., 2003). Reflective pondering may therefore represent a more adaptive strategy. However, it remains unclear how the two components are related to distress in somatic conditions.

The aim of the current analysis was to test whether having migraine predicted rumination. We hypothesised that migraine would be associated with higher rumination since it may increase the occurrence of discrepancies between one's current state and the desired one (see above).

Based on earlier findings, we also expected that migraine would be associated with increased current psychological distress indexed by depression and anxiety scores, and we hypothesised that brooding and reflection will partially mediate this relationship

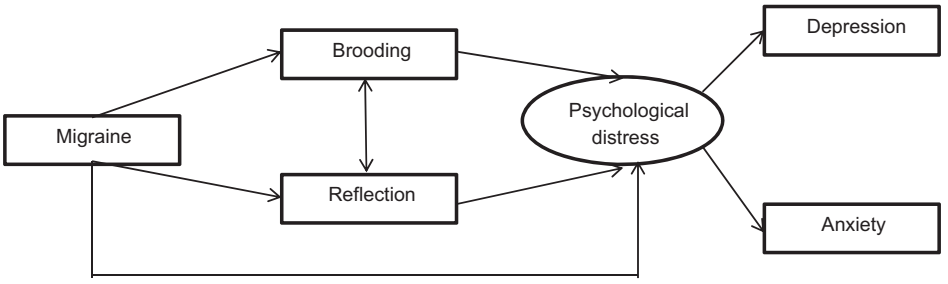

Figure 1. A theoretical model of the mediating effect of brooding and reflection between migraine and current psychological distress.

(see Figure 1). Both brooding and reflection components were expected to be associated with current psychological distress. However, taking into account that reflective pondering is considered to be a less maladaptive response to problems, reflection was expected to explain a smaller amount of the variance of distress than brooding.

Women are considered to ruminate more than men (Nolen-Hoeksema, Larson, & Grayson, 1999), though according to a meta-analysis (Johnson & Whisman, 2013), the effect size is small. Age is related negatively to rumination (Sutterlin, Paap, Babic, Kubler, & Vogele, 2012), but the relationship between rumination and negative health outcomes (e.g. delayed blood pressure recovery) may be stronger (Robinette & Charles, 2014). Elevated brooding and reflection scores are also found among formerly depressed patients compared to control groups (Joormann, Dkane, & Gotlib, 2006). Therefore, we controlled for sex, age and lifetime depression as potential confounders in our analysis.

Methods

Sample

The studies were part of NewMood (New Molecules in mood Disorders), an EU-funded research programme into pathological mechanisms of depression and related conditions (Deakin, Harro, & Anderson, 2011). Participants aged 18–60 years were recruited through general practices and the Internet in Manchester, and through general practices and advertisements in Budapest. In Manchester data from 2004 participants with a mean age of 33.50 (SD = 10.09), and in Budapest data from 1139 participants with a mean age of 31.41 (SD = 10.73) were analysed. We included those who returned the questionnaire either by mail or in person and did not report history of manic or hypomanic episodes, psychotic symptoms or obsessive compulsive disorder. Details of the recruitment strategies and responses have been reported previously (Juhasz et al., 2009). The studies were approved by the local Ethics Committees and the work was conducted in accordance with the Declaration of Helsinki. Written informed consent was provided by all participants. In these two independent cohorts, the proportion of females was almost identical (68 and 69%, respectively).

Measures

In a brief standard questionnaire, basic background information (gender, age and ethnicity) and personal and family psychiatric history were collected. Lifetime depression data were derived from this background questionnaire that has been validated previously and reported elsewhere (Juhasz et al., 2011).

The ID Migraine Questionnaire, a validated screening tool for migraine, was used to assess migraine with three items of the main migraine symptoms: disability, nausea and sensitivity to light (photophobia) (Lipton et al., 2003). According to a recent review and meta-analysis, the diagnostic accuracy of this questionnaire is good: the pooled estimate of sensitivity was 84% and pooled specificity was 76% (Cousins, Hijazze, Van de Laar, & Fahey, 2011). In our analysis, we identified migraineurs as having all the three of the migraine symptoms, and compared them to those who did not report any symptoms in order to test our hypothesis in a relatively homogenous subset of participants.

A short 10-item version of the Ruminative Response Scale (Treynor et al., 2003) was used to measure rumination and its facets: brooding and reflection. Brooding reflects a passive attention to the negative, often self-blaming thoughts related to distress, sad mood or stressors. Reflection (or reflective pondering) is interpreted as a purposeful self-reflective response to understand and solve the problem associated with depressive mood (Treynor et al., 2003). Items are rated on a four-point Likert-type scale ranging from 1 (never) to 4 (always). A continuous weighted score (sum of item scores divided by the number of items completed) was calculated and used in the analyses. Both the brooding and reflection subscales have good reliability in the Budapest (Cronbach αs: 0.71 and 0.73, respectively) and in the Manchester samples (Cronbach αs: 0.80 and 0.80, respectively), as well.

The Brief Symptom Inventory (BSI) subscales (Derogatis, 1993) were used to measure current depression (six items plus four additional symptoms from BSI: poor appetite, trouble falling asleep, thoughts of death or dying and feelings of guilt; Juhasz et al., 2009) and anxiety (6 items). Items are rated on a five-point Likert-type scale ranging from 0 (not at all) to 4 (extremely). A continuous weighted score (sum of item scores divided by the number of items completed) was calculated and used in the analyses. Both subscales of the BSI had good psychometric properties in the Budapest (Cronbach alpha for depression scale: 0.87; and for anxiety scale: 0.81) and in the Manchester samples (Cronbach α for depression: 0.92; for anxiety scale: 0.90).

Statistical analysis

SPSS 20.0 and Mplus 7.11 statistical software packages were used for statistical analyses. Descriptive analyses were used to assess the mean and standard deviation of the scales. For reliability, Cronbach's αs were calculated as indices of internal consistency, which was considered good if the values were at least 0.70. Path analysis was used to test the proposed mediation model within structural equation modelling (SEM). In the analysis, maximum likelihood estimation robust to non-normality (MLR) was used (Muthén & Muthén, 1998–2007). Testing the applicability of the proposed model both for Budapest and Manchester samples, a multi-group analysis was preferred to the two single-group models. Multi-group analysis allows for testing the invariance of the proposed mediation model across both samples. To evaluate the overall fit of our model,

the absolute fit index (χ^2), the comparative fit index (CFI), The Tucker–Lewis index or non-normed fit index (TLI) and the root mean square error approximation (RMSEA) were used. CFI and TLI are related to the total variance accounted by the model and values higher than 0.95 indicate a good fit. RMSEA is related to the variance of the residuals, values below 0.08 are considered an acceptable fit, while values below 0.05 indicate a good fit.

Results

Means and standard deviations are presented in Table 1. The two samples differed from each other on all the scales: the Manchester sample had higher rumination (total, brooding and reflection) and current psychological distress (depression; anxiety) scores. The effect sizes for sample differences in total rumination, brooding and depression were moderate in magnitude (Cohen's *d* above 0.50), and in reflection and anxiety were small (Cohen's $d = 0.36$).

Consistent with this, more participants in the Manchester sample (51.2%) reported lifetime depression in the background questionnaire, than in the Budapest sample (21.2%; $\chi^2 = 287.588$, $p < .001$). The proportion reporting any physical disability was also higher in the Manchester than in the Budapest sample (29.0 and 23.2%, respectively, $\chi^2 = 12.304$, $p < .001$).

In the Budapest sample, 57.7% ($N = 656$) of the participants reported no migraine-related symptoms, 19.3% ($N = 219$) reported only one symptom, 15.4% ($N = 175$) two symptoms and 7.6% ($N = 86$) had all the three symptoms on the ID Migraine Questionnaire. In the Manchester sample, 52.5% ($N = 1053$) of the participants had no symptoms, 18.1% ($N = 362$) reported only one symptom, 15.8% ($N = 317$) indicated two symptoms and 13.6% ($N = 272$) had all the three symptoms.

Therefore, assigning a diagnosis of migraine to participants who reported all three symptoms, the prevalence of migraine was 7.6% in the Budapest sample and 13.6% in the Manchester sample. In both samples, female migraineurs predominated (Budapest sample: 82.6% ($N = 71$) vs. 17.4% ($N = 15$), $\chi^2 = 14.706$ $p < .001$; Manchester sample: 84.6% ($N = 230$) vs. 15.4% ($N = 42$), $\chi^2 = 63.509$ $p < .001$). In both samples, pain conditions other than migraine were reported with a higher prevalence in the migraine group than in the control group (Budapest sample: 27.9% ($N = 24$) vs. 8.4% ($N = 55$),

Table 1. Means, standard deviations (SD) and effect sizes (Cohen's *d*) by sample.

	Hungarian sample ($N = 1139$) Mean (SD)	Manchester sample ($N = 2004$) Mean (SD)	*t* (Cohen's *d*)
Rumination	1.94 (0.48)	2.21 (0.62)	13.99*** (0.49)
Brooding	1.94 (0.56)	2.26 (0.71)	14.04*** (0.50)
Reflection	1.94 (0.58)	2.17 (0.70)	9.88*** (0.36)
BSI – Depression	0.56 (0.68)	1.04 (0.99)	16.30*** (0.57)
BSI – Anxiety	0.68 (0.70)	0.99 (0.99)	10.10*** (0.36)

Notes: BSI: Brief symptom inventory.
For all scales, a continuous weighted score (sum of item scores divided by the number of items completed) was calculated and used in the analyses.
***$p < .001$.

$\chi^2 = 30.461$ $p < .001$; Manchester sample: 21.7% ($N = 59$) vs. 5.3% ($N = 56$), $\chi^2 = 72.893$ $p < .001$).

In both samples, migraineurs had higher scores on rumination and its two facets (brooding and reflection) than the control group with no migraine symptoms. Current depression and anxiety scores were also higher among people with migraine than among controls (Table 2). In both samples, the effect size for differences was at least moderate (Cohen's *d* above 0.50) or large (Cohen's *d* above 0.80) in magnitude on all variables except the reflection scale, where the effect size was small (Cohen's *d* below 0.50).

Brooding and reflection were positively associated in both samples (see Table 3), and both were related positively to anxiety and depression ($r > 0.50$). Since anxiety and depression were highly correlated ($r > 0.73$ and $r > 0.80$, see Table 3), in our mediation model, a single latent variable was used for current psychological distress (see Figure 1).

We hypothesised that migraine would predict brooding and reflection, and the relationship between migraine and current psychological distress (as a latent variable indexing depression and anxiety) would be mediated by the two facets of rumination (see Figure 1). The degree of fit of the proposed mediation model was tested by the means of SEM. In our model sex, age and lifetime depressive episode were controlled for. We ran two analyses: in the first (M1), the structural paths were estimated freely, then in the second (M2), we assumed that structural paths were invariant across both samples. According to the results obtained, relative goodness of fit indices met their corresponding critical values for both M1 ($\chi^2 = 64.485$, d$f = 12$, RMSEA = 0.065 [0.050–0.081], CFI = 0.986, TLI = 0.949) and M2 ($\chi^2 = 107.506$, d$f = 18$, RMSEA = 0.069 [0.057–0.082], CFI = 0.976, TLI = 0.942). Because the degree of fit decreased significantly (Satorra–Bentler-scaled χ^2 difference test = 42.469, d$f = 6$, $p < .001$) when all the path coefficients were constrained to be equal in the two samples, the invariance of the mediation model was not supported. This means that the relationship between variables was not invariant across both samples.

According to the results, in both samples, migraine had a significant direct effect on brooding and reflection and also on psychological distress. Similarly, brooding had a direct effect on psychological distress in both samples, but reflection predicted psychological distress significantly only in the Budapest sample. The path between reflection and psychological distress was non-significant in the Manchester sample (see Figure 2).

In relation to the indirect effect, the 'migraine → brooding → psychological distress' pathway was significant in both the Budapest (standardised indirect effect was 0.129 $p < .001$) and Manchester samples (standardised indirect effect was 0.135 $p < .001$). The 'migraine→reflection→psychological distress' was significant in the Budapest (standardised indirect effect was 0.023 $p < .001$), but not in the Manchester sample (standardised indirect effect was 0.003 $p > .05$). Therefore, migraine had an effect via both brooding and reflection on distress among Hungarian participants, but in the Manchester sample, only brooding mediated the relationship between migraine and distress.

In the Budapest sample, the final model explained 51.9% and in the Manchester sample, 55.3% of the total variance of psychological distress.

Pain conditions other than migraine might influence the relationship between variables; therefore, we reran our analyses while controlling for other pain conditions. Our results did not change at all (M1: $\chi^2 = 68.796$, d$f = 14$, RMSEA = 0.062 [0.047–0.076], CFI = 0.985, TLI = 0.946) and M2 ($\chi^2 = 112.206$, d$f = 20$, RMSEA = 0.067

Table 2. Means, standard deviations and effect sizes (Cohen's *d*) by sample and migraine status.

	Hungarian sample			Manchester sample		
	Migraine group ($N = 86$)	Control group ($N = 656$)	*t/d* (Cohen's *d*)	Migraine group ($N = 272$)	Control Group ($N = 1053$)	*t/d* (Cohen's *d*)
Rumination	2.18 (0.49)	1.83 (0.46)	6.54*** (0.74)	2.46 (0.60)	2.08 (0.60)	9.33*** (0.63)
Brooding	2.24 (0.59)	1.80 (0.52)	6.66*** (0.79)	2.54 (0.73)	2.10 (0.68)	9.09*** (0.62)
Reflection	2.12 (0.58)	1.87 (0.57)	3.78*** (0.43)	2.38 (0.69)	2.06 (0.71)	6.76*** (0.46)
BSI – Depression	0.96 (1.01)	0.40 (0.53)	5.05*** (0.69)	1.56(1.07)	0.76 (0.84)	11.33*** (0.83)
BSI – Anxiety	1.12 (0.91)	0.50 (0.57)	6.21***(0.81)	1.51 (1.10)	0.70 (0.85)	11.29*** (0.82)

Notes: BSI: Brief symptom inventory.
For all scales, a continuous weighted score (sum of item scores divided by the number of items completed) was calculated and used in the analysis.
***$p < .001$.

Table 3. Correlations among scales.

	Rumination	Brooding	Reflection	BSI – Depression	BSI – Anxiety
Rumination	–	0.83	0.85		0.53
Brooding	0.87	–	0.40	0.55	0.56
Reflection	0.87	0.52	–	0.36	0.32
BSI – Depression	0.56	0.62	0.36	–	0.73
BSI – Anxiety	0.53	0.59	0.35	0.80	–

Notes: BSI: Brief symptom inventory.
Above the diagonal correlations among scales in the Manchester sample, and below the diagonal correlations among scales in the Budapest sample are presented. For all scales, a continuous weighted score (sum of item scores divided by the number of items completed) was calculated and used in the analysis. All correlation coefficients are significant at the 0.1 % level ($p < .001$).

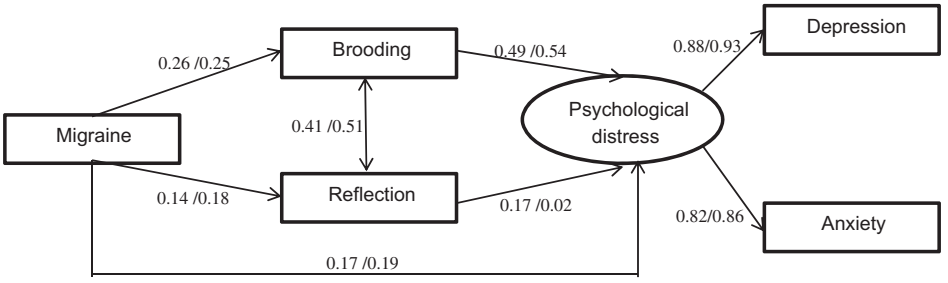

Figure 2. The mediation model and standardised path coefficients. Results of multi-group analysis with factor loadings and path coefficients across both samples (Budapest ($N = 742$)/Manchester ($N = 1325$).
Note: All paths are significant at $p < .001$, except one between reflection and psychological distress in the Manchester sample (standardised path coefficients: 0.02).

[0.055–0.079], CFI = 0.976, TLI = 0.936; Satorra–Bentler-scaled χ^2 difference test = 42.797, d$f = 6$, $p < .001$).

Since our study has a cross-sectional design, multiple equivalent/competing alternative models can be produced. In our model (see Figure 1), migraine explains the variance of psychological distress. We could have formulated our theoretical model differently, such that distress explained the variation in migraine status. Since migraine is a binary variable, we ran path analysis with a categorical dependent variable. For a model where the relationship between migraine and distress was simply reversed (psychological distress → brooding/reflection → migraine), and brooding and reflection mediated this reversed relationship, all the fit statistics indicated a poor fit: $\chi^2 = 762.846$, d$f = 30$, RMSEA = 0.156, CFI = 0.575, TLI = 0.292, after controlling for sex, age and lifetime depression.

Discussion

In two independent samples, we demonstrated that migraine was associated with higher rumination scores. Both rumination components: brooding and reflection, were elevated

among migraineurs, suggesting that a chronic condition like migraine could be associated with both a more passive process (brooding) and with a more purposeful one (reflection) (Treynor et al., 2003). Brooding and reflection partially mediated the relationship between migraine and psychological distress: migraine had both a direct effect and an indirect effect via rumination on psychological distress after controlling for potentially confounding variables of sex, age and lifetime depression. To our knowledge, this is the first study that examines the relationship between migraine and rumination and its components, and our results are in line with Soo et al.'s perspective (2014, 2009) that rumination could be a relevant process in somatic conditions.

Elevated psychological distress in migraine has been detected in several studies (see Introduction). We hypothesised that the relationship between migraine and distress would be mediated by rumination and its components (brooding and reflection). Our results showed that brooding partially mediated the relationship between migraine and psychological distress in both samples, while reflection had a significant effect on psychological distress only in the Budapest sample. The main difference between the two samples in our study was the higher proportion of previously depressed individuals in the Manchester sample that might influence our results. However, the relative importance of brooding in predicting psychological distress was also elevated in the Budapest sample: the standardised beta between brooding and distress (0.49) was higher compared to the coefficient between reflection and distress (0.17). This result is in line with the notion that reflective pondering is a less maladaptive response to negative emotional states including sad mood. Definition of reflective pondering resembles analytical rumination considered as an adaptive response to complex (mainly social) problems (Andrews & Thomson, 2009) with the aim of understanding the meaning, causes and consequences of the problem. Nonetheless, recalling and intentionally (re)processing of negative events are usually accompanied with elevated negative emotions that may explain why reflection is associated with concurrent distress (McLaughlin, Borkovec, & Sibrava, 2007; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013).

Based on the bidirectional relationship between depression and migraine found in longitudinal studies (Breslau et al., 1994, 2003), one could argue that rather than migraine status leading to higher perceived stress, it is the psychological distress that explains migraine status. We cannot exclude this possibility. Our study had a cross-sectional design and our aim was to examine whether rumination partially explained the relationship between migraine and distress, in the direction of process illustrated in the mediation analysis. However, we also tested the reversed relationship between migraine and distress (psychological distress → brooding/reflection → migraine). All the fit statistics of this alternative model indicated a poor fit, though this result might be due to our cross-sectional design.

Current views of migraine suggest that migraine is a primary disorder of the brain's pain modulating circuit, which is supported by neuroimaging evidence (Schwedt, Chiang, Chong, & Dodick, 2015). Therefore, there is a need to determine how psychological processes may affect brain responses to either stress or to migraine attacks. Among healthy people, trait or state rumination after termination of a stressor is related to poor cardiovascular recovery indexed by blood pressure recovery (Gerin et al., 2006; Johnson, Lavoie, Bacon, Carlson, & Campbell, 2012) and heart rate variability recovery (Key et al., 2008; Shumaker, Ockene, & Riekert, 2009). Some study results indicate that the lack of habituation to repeated stressors also contributes to the adverse effect of rumination on somatic

health (Gianferante et al., 2014; Johnson et al., 2012). Based on these results, we speculate that rumination via sustained psychological and physiological arousal may increase the allostatic load (McEwen, 1998) caused by stressors and migraine attacks on the brain in migraine (Borsook et al., 2012). However, this hypothesis should be tested empirically. Rumination has been linked to poor sleep quality (Pillai, Steenburg, Ciesla, Roth, & Drake, 2014; Zoccola, Dickerson, & Lam, 2009) and this may represent another pathway through which rumination may add to the allostatic load.

Limitations

The study has some limitations. Due to our cross-sectional design, we can't draw any conclusions about the causal relationship between migraine and rumination. Furthermore, self-report questionnaires were used, and we assigned migraine to participants who reported three migraine-related symptoms. Nonetheless, we used a valid screening tool for migraine with good specificity and sensitivity (Cousins et al., 2011). However, further studies with clinical samples are needed to confirm and expand our findings. It is worth noting that there was a higher proportion of females in the migraine group which might affect the results when migraineurs were compared with non-migraineurs. However, when sex was controlled for in our mediation model, migraine status still predicted brooding, reflection and psychological distress. Also, there was a higher prevalence of other pain conditions in the migraine group than in the control group; hence, we can't exclude the possibility that pain other than migraine headache affected our results. Again, however, when we controlled for other pain conditions in the SEM analyses, our results did not change. We had no data about pain medication in either group, so we do not know whether it would have affected any of the variables used in our analyses.

We concentrated on the possible mediating effect of rumination between migraine and distress, but other perseverative thoughts, such as worry, may be also relevant in physical illnesses (Brosschot, Gerin, & Thayer, 2006; Kubzansky et al., 1997). Furthermore, the direct and indirect relationship via rumination between migraine and psychological distress could be tested empirically. Psychological and physiological responses to a stressor and in a subsequent period among migraineurs and non-migraineurs could help support our mediation model.

We only investigated if rumination was related to low mood, though other ruminative processes such as ruminative thoughts in response to physical illness (Soo et al., 2014) are also important, and perhaps give insight into the factors relating to the adjustment in chronic conditions. Longitudinal studies need to address whether increased rumination is a reaction to perceived loss due to the impact of migraine, or whether it is observed in the premorbid personality.

Conclusion

Emotion regulation has not been investigated systematically in migraine, in spite of the fact that many features of migraine could be considered in this framework, including: the elevated level of distress (Breslau et al., 1994, 2000, 2003; Ligthart et al., 2013; McWilliams et al., 2004; Modgill et al., 2012; Smitherman, Kolivas, & Bailey, 2013; Swanson, Zeng, Weeks, & Colman, 2013), complex interaction between emotion,

emotion regulation and pain (Lumley et al., 2011), role of stressors (Sauro & Becker, 2009) and the efficacy of stress-releasing methods (e.g. relaxation and/or stress management (Penzien, Irby, Smitherman, Rains, & Houle, 2015)). Our results indicate that as well as genetic factors (Schur, Noonan, Buchwald, Goldberg, & Afari, 2009) and migraine-related variables, inter-individual differences in mental health in migraine might be determined by emotion regulation. It is worth noting that rumination may be relevant in other chronic conditions as well (Fernandez et al., 2010; Galfin & Watkins, 2012; León, Nouwen, Sheffield, Jaumdally, & Lip, 2010; Trick, Watkins, & Dickens, 2014), especially in other pain conditions (Edwards, Tang, Wright, Salkovskis, & Timberlake, 2011). Based on our findings, we suggest that rumination could be an important outcome variable in treatment studies using cognitive behavioural or other techniques or could be a specific target if migraine is comorbid with depression. However, further studies are needed to establish whether rumination affects important migraine outcomes (e.g. severity, frequency and disability).

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Conflict of interest

Prof. Deakin variously performed consultancy, speaking engagements and research for Bristol-Myers Squibb, AstraZeneca, Eli Lilly, Schering Plough, Janssen-Cilag and Servier (all fees are paid to the University of Manchester to reimburse them for the time taken); he has share options in P1vital. Prof. Anderson has received consultancy fees from Lundbeck, Lundbeck/Takeda and Alkermes and grant support from Servier and AstraZeneca. Rebecca Elliott received consultancy fees from Cambridge Cognition and P1vital. The other authors report no biomedical financial interests or potential conflicts of interest.

Disclosure statement

No potential conflict of interest was reported by the authors.

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