

The Impact of Thought Speed and Variability on Psychological State and Threat Perception: Further Exploration of the Theory of Mental Motion

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RUNNING HEAD: IMPACT OF THOUGHT SPEED AND VARIABILITY

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Compliance with Ethical Standards

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Abstract

Thought speed and variability are purportedly common features of specific psychological states, such as mania and anxiety. The present study explored the independent and combinational influence of these variables upon condition-specific symptoms and affective state, as proposed by Pronin and Jacobs' (2008) theory of mental motion. A general population sample was recruited online ($N = 263$). Participants completed a thought speed and variability manipulation task, inducing a combination of fast/slow and varied/repetitive thought. Change in mania and anxiety symptoms was assessed through direct self-reported symptom levels and indirect, processing bias assessment (threat interpretation). Results indicated that fast and varied thought independently increased self-reported mania symptoms. Affect was significantly less positive and more negative during slow thought. No change in anxiety symptoms or threat interpretation was found between manipulation conditions. No evidence for the proposed combinational influence of speed and variability was found. Implications and avenues for therapeutic intervention are discussed.

Introduction

Accumulating evidence suggests that attributes depicting the way one thinks may play a fundamental role in the manifestation of psychological conditions (e.g., Brunyé, Gagnon, Paczynski, Shenhav, Mahoney, & Taylor, 2013; Drost, van der Does, van Hemert, Penninx, & Spinhoven, 2014; Mason & Bar, 2011; McLaughlin, Borkovec, & Sibrava, 2007; Pronin, 2013; Watkins, 2008). Experimental induction of thinking styles symptomatic of specific conditions has been demonstrated to impact on psychological state. For instance, inducing the rumination associated with depression may increase depression levels and inducing the worry associated with anxiety may increase negative affect (McLaughlin et al., 2007; Nolen-Hoeksema & Morrow, 1993). Consequently, the way one thinks may not simply reflect features of specific psychological difficulties; it may directly contribute to those difficulties.

Pronin and Jacobs' (2008) model of mental motion proposes the potential importance of two particular movement-based attributes of thought, namely speed and variability. Thought speed refers to the pace of thinking, whereas thought variability refers to the interconnectivity between thoughts (i.e., their uniqueness or repetitiveness). The mental motion account predicts condition-specific symptoms are causally related to the behaviour of these two attributes of thought.

Experimental manipulation of both attributes has been associated with differences in psychological state (see Pronin & Jacobs, 2008, for example manipulations). Thought acceleration has been associated with increased positive affect (Pronin, Jacobs, & Wegner, 2008; Yang, Friedman-Wheeler, & Pronin, 2014) as well as greater risk-taking compared to slow thought (Chandler & Pronin, 2012). Thought variability has also been associated with specific psychological outcomes

(see Watkins, 2008, for review). Narrow associative thinking (i.e., thoughts revolving around a narrow topic) may decrease positive affect, whereas broad progressive thinking (i.e., thoughts that are related but developing in thematic focus) may decrease negative affect (Mason & Bar, 2011). The role of thought content in psychological conditions is acknowledged; however, the characteristics of mental motion are proposed to exert additional content-independent effects (Pronin & Jacobs, 2008).

The proposed effects of mental motion may be evolutionarily advantageous: facilitating mobilisation and activation in emergency situations that induce quick thinking (Pronin, 2013) and promoting learning/problem-solving through varied thinking by increasing positive affect (Bar, 2009). Conversely, depression, which is associated with slow ruminative thinking, has been hypothesised to encourage inactivity where action is counterproductive (Nesse, 2000).

The influence of thought speed and variability is posited to be both individual and combinational. Individually, specific changes in either thought speed or variability are suggested to cause changes in psychological state consistent with specific conditions (e.g., anxiety, depression, and mania). However, the influence of mental motion may be best understood in terms of specific combinations of thought speed *and* variability (see Pronin & Jacobs, 2008, Fig. 1). The model predicts that depressive states may be induced by slow, repetitive thought; whereas anxious and manic states share increased thought speed but may be differentiated by thought variability. Anxiety is predicted as involving fast, repetitive thought, whereas mania involves fast, varied thought. Although untested as yet, it is plausible that specific cognitive processing biases consistent with specific conditions (e.g., Mogg & Bradley, 2005) may also be induced through manipulation of these variables.

Consequently, the model provides a framework in which individuals experiencing different psychological conditions may exhibit both convergence and divergence in attributes of their thinking style. This proposition has potential relevance to transdiagnostic models of psychological difficulties, which suggest core underlying thought processes may feature across multiple conditions. Consideration of the combinational, as well as individual, influences of different attributes of different thinking styles is one avenue that may contribute to developing our understanding how transdiagnostic psychological risk factors can lead to divergent trajectories and multifinality (see Nolen-Hoeksema & Watkins, 2011).

Whilst the outlined evidence supports the individual influence of thought speed and variability on psychological state, the proposed combinational influence has received limited investigation. In a pace-controlled reading task of neutral trivia statements, Pronin and Jacobs (2008) manipulated combinations of thought speed and variability in 74 participants. No interactive effect of thought speed and variability was demonstrated on affective state. However, repetitive thought was associated with more feelings of depression than anxiety when thought was slow and with more feelings of anxiety than depression when fast. The latter thinking style also predicted higher levels of felt energy. The reported effects using neutral stimuli support the proposed content-independent influence of mental motion. However, the study provided inconclusive support for the proposed combinational influence of these variables as not all predicted outcomes were evidenced and the reported significant interaction effects related to differences *between* levels of anxiety and depression rather than *within* each condition – complicating interpretation of where change occurred.

Research replicating and extending investigation beyond this single study is warranted. Further investigation exploring the differentiating role of thought speed and variability in determining specific psychological conditions is necessary. In particular, the proposed combinational effects of thought speed and variability in generating mania and anxiety symptoms is largely untested and requires empirical evaluation. Providing this evaluation was the primary focus of the present research.

The Present Research

The present study was a larger scale replication of the pace-controlled reading experimental design developed by Pronin and colleagues, outlined above (Pronin & Jacobs, 2008; Pronin & Wegner, 2006). This study extended previous investigation through more detailed focus on the proposed individual and combinational influence of mental motion variables on mania and anxiety symptoms. In addition, affective state has frequently featured in previous research and has provided relatively consistent evidence. Consequently, assessment of affect was included to enable reliability comparison with existing evidence.

The study employed validated clinical self-report measures of condition symptoms alongside indirect symptom assessment through cognitive processing bias. Processing biases are posited to be instrumental in the maintenance of psychological difficulties (e.g., Clark, 1999) and differentiable between conditions (e.g., Mogg & Bradley, 2005). Consequently, this study included assessment of condition-specific threat interpretation bias as an additional, more objective, assessment of condition symptoms. Evidence suggests that processing biases commonly present in anxiety as unconscious, uncontrollable propensities towards threat perception (Teachman, Joormann, Steinman, & Gotlib, 2012). However, whilst

mania may be associated with decreased threat sensitivity, evidence is inconsistent (Carver & Johnson, 2009). Consequently, this study component was exploratory.

Based on theory and evidence outlined, the following primary and secondary research questions were developed:

Primary research question. *Are specific manipulations and combinations of thought speed and variability associated with condition-specific symptoms of mania and anxiety, as predicted by the model of mental motion?*

Hypothesis one. It was predicted that increased thought speed would be associated with increased self-reported levels of mania symptoms, particularly when thought was varied; and increased self-reported levels of anxiety symptoms, particularly when thought was repetitive.

Hypothesis two. As anxiety is associated with increased threat perception (Mogg & Bradley, 2005), it was predicted that fast, repetitive thought would also be associated with increased levels of threat interpretation.

Hypothesis three. Symptoms of mania may be associated with decreased threat sensitivity; however, evidence is inconsistent (Carver & Johnson, 2009). Consequently, it was tentatively predicted that fast, variable thought would be associated with decreased levels of threat interpretation.

Secondary research question. *Are specific manipulations of thought speed and variability associated with affective state consistent with the model of mental motion and previous research findings?*

Hypothesis four. Based on previous research, it was predicted that both faster and more variable thought would be associated with higher level of positive affect.

Hypothesis five. The mental motion model predicted that both slower and more repetitive thought would be associated with higher levels of negative affect (i.e., simulating depressive rumination). However, previous research has inconsistently demonstrated significant association between negative affect and these variables (Pronin et al., 2008).

Method

Participants

Two hundred and sixty-three participants comprised the final sample post data-screening. Participants were an opportunity sample of adults (≥ 18 years) recruited from the general population through online advertising and advert circulation within a selection of UK universities. Demographic information is summarised in Table 1.

A subsection of participants ($n = 78$) were university students recruited through an online participant pool and awarded course credit for participation. Baseline differences between this subsection and remaining participants were explored during data-screening and accounted for in the analyses.

Comparison of final study participant raw data and study non-completers was conducted through Chi-square and independent t-tests – Table 1. Of the 603 participants comprising the full dataset, up to 567 participants provided baseline data and could be employed in this comparison. Significant differences in sex, age, and employment status were found. Levene's test indicated unequal variances for age,

$F(1, 565) = 4.76, p = .030$, and PANAS NA, $F(1, 461) = 13.42, p \leq .001$, only. The adjusted t-test statistic was consulted for these two variables. Overall, no significant differences between completers and non-completers were revealed by t-tests on dependent variables ($ts \leq 1.39; ps \geq .167$), with the exception of PANAS NA, $t(368.78) = 2.24, p = .026$, equal variances not assumed. Non-completers reported higher levels of negative affect ($M = 17.73, SE = .59$) compared to completers ($M = 16.11, SE = .41$). Overall, despite demographic differences between the compared groups, baseline performance on dependent variables used for repeat measurement appeared equivalent.

[INSERT TABLE 1 HERE]

Measures and Materials¹

Internal State Scale (ISS; Bauer et al., 1991). Self-reported activation level was assessed using the ISS. Activation was selected as a mania symptom because it is considered a common and core factor underpinning the range of mania symptoms (see Mansell & Pedley, 2008). Additionally, the ISS: activation subscale correlates positively with clinical assessment of mania.

The ISS includes 15 items comprising four subscales: activation, well-being, depression, and perceived conflict. Participants endorse items (e.g., *Right now I feel impulsive*) on 0-100% agreement scale. Subscales have good internal consistency ($\alpha = .81$ to $.92$). This study utilised activation (5 items) and wellbeing (3 items)

¹ Three additional measures respectively assessing trait vulnerability towards mania (Hypomanic Personality Scale; Eckblad & Chapman, 1986), anxiety (State-Trait Inventory for Cognitive and Somatic Anxiety; Grös, Antony, Simms, & McCabe, 2007), and cognitive fusion (Cognitive Fusion Questionnaire; Gillanders et al., 2014) were also employed at baseline. However, they are not reported here as they pertain to a research question outside of those specified in the present study.

subscales only. ISS: activation subscale scores can be combined wellbeing subscale scores to provide dichotomous categorisation of mood state for (hypo)manic, mixed state, euthymic, and depressive states, respectively (Bauer, Vojta, Kinosian, Altschuler, & Glick, 2000).

Instructions were modified to assess current state rather than last 24 hours. This modification has been employed elsewhere (e.g., Taylor & Mansell, 2008). Cronbach's $\alpha = .82$ (activation) and $.78$ (well-being) in the present study.

Spielberger State-Trait Anxiety Inventory – short-form (STAI-sf; Marteau & Bekker, 1992). Self-reported anxiety symptoms were assessed using the STAI-sf. The STAI-sf is a six-item short-form of the original STAI (Spielberg, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Participants endorse items (e.g., *I feel calm*) on a 4-point likert-scale (1 = *not at all*; 4 = *very much*). Authors report good internal consistency (Cronbach's $\alpha = .82$) and comparability to the full STAI. Compared to alternative short-form versions, the STAI-sf demonstrates the best reliability and validity in correlation with the full STAI (Tluczek, Henriques, & Brown, 2009). Cronbach's $\alpha = .86$ in the present study.

Ambiguous Scenarios Test (AST; Mathews & Mackintosh, 2000). Threat interpretation was assessed using the AST. The test consists of 20 short threat-ambiguous scenarios – 10 pre and 10 post manipulation. For each scenario, participants are presented with two sentences providing differing interpretations of the scenario: one threatening interpretation (*As you speak, people in the audience find your efforts laughable*) and one neutral/benign interpretation (*As you speak, people in the audience laugh appreciatively*). Participants rate both interpretations on a 4-point likert scale in terms of how similar to the meaning of the scenario they

thought each interpretation was (1 = *very different in meaning*; 4 = *very similar in meaning*). Only ratings of the threat interpretation were employed in the present study analyses as these data relate most specifically to the research question. Scenarios in each set of 10 were presented in the same order and the sets counterbalanced between participants (consistent with Hoppitt, Mathews, Yiend, & Mackintosh, 2010).

Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item measure of positive affect (PA) and negative affect (NA). Items are endorsed on the extent that they relate to present mood on a 5-point likert-scale (e.g., *Interested*: 1 = *very slight or not at all*; 5 = *extremely*). Authors report good internal consistency (PA, $\alpha = .89$; NA, $\alpha = .85$) and construct validity through correlation with depression and anxiety measures. Cronbach's $\alpha = .9$ (PA) and $.87$ (NA) for the present study. The PANAS was employed to support the validity of utilising two brief mood items to assess state affect – detailed below.

Brief mood items. Self-reported affect was assessed by single-item assessments of PA and NA, respectively. Participants rated the positivity and negativity of their current mood on two separate 0-100% scales (e.g., *How positive is your mood right now?*). The two single-items were preferred over the PANAS for repeated measurement pre/post manipulation to reduce task burden. For rudimentary validation, baseline scores on the brief mood items were compared to baseline PANAS scores. PA and NA brief mood items demonstrated significant correlation with PANAS PA, $r(261) = .43, p < .001$, and NA, $r(261) = .54, p < .001$, respectively.

Engagement and technical difficulties questions. Items evaluating participant engagement and technical difficulty during study completion were included for data-screening purposes.

Manipulation

The thought speed and variability manipulations replicated Pronin and colleagues' methodology (Pronin & Jacobs, 2008; Pronin & Wegner, 2006). A 2x2 factorial design was employed: thought speed (fast/slow) X thought variability (varied/repetitive). Participants were allocated to one of four manipulation conditions. In each condition, participants read 63 neutral statements presented in a video produced using Microsoft Powerpoint. Thought speed was manipulated by speed of statement presentation: either fast (40ms per letter) or slow (170ms per letter). The interval between statements was 320ms in the fast condition and 4,000ms in the slow condition. Thought variability was manipulated by presenting either a non-repeating sequence of 63 neutral statements (varied thought) or presenting the same three statements 21 times (repetitive thought). Multiple versions of the repetitive condition, with different sets of three statements, were employed across participants to prevent content effects.

Thought speed manipulation check. A single-item utilised by Pronin and Wegner (2006) was employed to evaluate change in perceived thought speed resultant from the manipulation. Participants rated their current speed of thought on a 9-point likert-scale (e.g., *What do you feel is the speed of your thought right now?* 1 = *very slow*; 9 = *very fast*). The original item wording was modified to enable repeated employment pre/post manipulation.

Procedure

Ethical approval was awarded by the departmental ethics committee. Participants completed the study online. Following informed consent, participants completed demographic details and baseline questionnaires – the order of the latter was randomised. Participants then completed the pre-manipulation AST before being allocated to one of four manipulation conditions. Allocation to manipulation condition was determined by the pseudorandom number generator function of the online study software (qualtrics.com), which guarantees relatively equal numbers between conditions. Post-manipulation questionnaires and AST were then administered. Thought speed was assessed at four points across the course of the study – Figure 1.

The study concluded with engagement and technical difficulties questions and a relaxation video aimed to counteract any residual effects of the manipulation. Participants were provided with a full study debrief.

[INSERT FIG 1 HERE]

Data-screening and Reduction

The original dataset ($N = 603$) was screened and reduced to include participants who completed all study components within specific time parameters (e.g., no longer than 1 ½ hours; and remained on the manipulation video webpage for a set minimum time period²). The final data set consisted of 263 participants. To reduce the influence of extreme data points, dependent variables were assessed for outliers, which were replaced using the Winsorising approach.

² Participant time on webpage ≥ 138.6 secs (i.e., 90% of the shortest manipulation video).

T-tests explored baseline differences between participants recruited from the university participant pool and the remaining sample in the final dataset. The adjusted t-test statistic was consulted where Levene's test indicated unequal variances, as was the case for PANAS NA and both brief mood items. No significant baseline differences were found ($ts \leq .87$; $ps \geq .388$), with the exception of threat interpretation score, $t(261) = 2.68$, $p = .008$. Consequently, the sample was collapsed for analysis; however, potential differences between the participant pool subgroup and full sample were considered when analysing threat interpretation data.

Data Transformation

Where data violated statistical test assumptions (e.g., Levene's test) standardised residuals were consulted and/or square-root transformations applied. Subsequently, transformed data were utilised when this action improved model fit. Untransformed data were utilised when fit was found to be acceptable or unimproved by transformation. Instances where test assumptions were violated and model fit could not be improved are highlighted within the results.

Results

Baseline Descriptive Information and Comparisons

Descriptive information for all measures was compiled – Table 2.

[INSERT TABLE 2 HERE]

Baseline comparisons were conducted to assess participant equivalence between the manipulation conditions. Between-groups univariate ANOVAs were employed to investigate baseline differences between the four conditions on each dependent variable. No significant difference was demonstrated between

experimental conditions on these measures ($F_s \leq 1.84$, $p_s \geq .141$), with the exception of single-item NA, $F(3, 259)$, $p = .031$, $\eta_p^2 = .03$. However, baseline scores were accounted for in all subsequent analyses of the dependent variables (including NA), either as covariate or repeated-measures design. Consequently, this finding did not compromise the following results.

Manipulation Check

The impact of the experimental manipulation on perceived thought speed was assessed through repeated-measures ANOVA for slow and fast thought conditions, respectively. Mauchly's test indicated violation of assumption of sphericity (slow thought speed, $X^2(5) = 114.68$, $p < .001$; fast thought speed, $X^2(5) = 31.84$, $p < .001$). Therefore, Greenhouse-Geisser estimates of sphericity are reported as the corrected degrees of freedom.

Analyses demonstrated significantly decreased thought speed in the slow manipulation, $F(1.96, 250.59) = 15.09$, $p < .001$, $\eta_p^2 = .11$, and increased thought speed in the fast manipulation, $F(23.67, 354.66) = 21.87$, $p < .001$, $\eta_p^2 = .14$. Simple contrasts revealed that the initial effect size (between assessment point 1-2) was large for the slow manipulation ($\eta_p^2 = .14$), and larger still for the fast manipulation ($\eta_p^2 = .24$). In both conditions, the manipulation effect remained significant at assessment point 3 ($p < .001$), but decreased in effect size and returned to pre-manipulation state by assessment point 4 ($F_s \leq 1.22$, $p_s \geq .272$).

Primary Research Question: Associations between Mental Motion and Symptoms of Mania and Anxiety

The study was concerned with whether manipulation of thought speed and variability influenced psychological state consistent with predictions based on the model of mental motion. Of primary interest were between-condition differences in symptoms associated with mania and anxiety assessed via self-report and threat interpretation. To explore the primary predictions, a series of 2x2 ANCOVAs were conducted: thought speed (fast/slow) X thought variability (varied/repetitive). The post-manipulation score (assessment point 2/3) on the symptom of interest was entered as the dependent variable. Pre-manipulation score (assessment point 1) was entered as a covariate to account for baseline symptom level. ANCOVA was preferred over alternative tests as it accounts for between-condition differences at baseline, is a powerful test well suited to the study design (Vickers & Altman, 2001), and is consistent with comparable study analyses (Yang et al., 2014). All subsequent reported means are ANCOVA-produced adjusted means – Table 3.

[INSERT TABLE 3 HERE]

These analyses provided assessment of differences *between* manipulation conditions. Where significant between-group effects were found, repeated-measures ANOVA were employed to establish the direction and effect size *within* respective manipulation conditions pre-to-post manipulation. For example, if ANCOVA demonstrated significant difference between fast and slow thought speed conditions for a dependent variable, then change in that dependent variable across assessment points 1-2 was subsequently assessed *within* fast and slow conditions, respectively, by conducting separate repeated-measures ANOVAs for each condition.

Hypothesis one: Self-report symptoms of mania and anxiety. ANCOVA supported the predicted association between the mania symptom ‘activation’ and

increased thought speed and variability. As predicted, activation level was significantly higher in the fast thought condition ($M = 12.6$, $SE = .34$), compared to slow ($M = 10.0$, $SE = .34$), $F(1, 258) = 30.20$, $p < .001$, $\eta_p^2 = .11$. Furthermore, activation level was significantly higher in the variable thought condition ($M = 11.9$, $SE = .34$), compared to repetitive ($M = 10.6$, $SE = .35$), $F(1, 258) = 7.41$, $p = .007$, $\eta_p^2 = .03$.

Assessment of symptom change pre-to-post manipulation within fast and slow thought speed conditions, respectively, clarified these findings. The repeated-measures ANOVAs demonstrated that activation level significantly increased when manipulation induced fast thought speed, $F(1, 133) = 36.32$, $p < .001$, $\eta_p^2 = .21$, but did not significantly change when thought speed was slowed, $F(1, 128) = .79$, $p = .375$, $\eta_p^2 = .01$. Similarly, within the thought variability conditions, only variable thought demonstrated within-group change. Activation increased when variable thought was induced, $F(1, 135) = 19.62$, $p < .001$, $\eta_p^2 = .13$, but not when thought was repetitive, $F(1, 126) = .50$, $p = .482$, $\eta_p^2 < .01$.

Contrary to prediction, ANCOVA demonstrated no significant differences in anxiety symptoms (measured by STAI-sf) between manipulation conditions. No significant main effects were demonstrated between thought speed, $F(1, 258) = 1.02$, $p = .314$, $\eta_p^2 < .001$, or thought variability conditions, $F(1, 258) = .23$, $p = .636$, $\eta_p^2 < .01$.

Contrary to the predicted combinational role of thought speed and variability in differentiating manic and anxious states, the interaction term (Speed X Variability) was non-significant for both activation, $F(1, 258) = 1.08$, $p = .3$, $\eta_p^2 < .01$., and anxiety level, $F(1, 258) = .05$, $p = .826$, $\eta_p^2 < .01$.

Hypotheses two and three: Objective symptoms of mania and anxiety.

Contrary to prediction, no evidence of condition-specific differences in threat interpretation (measured by AST) was found between manipulation conditions. ANCOVA main effects were non-significant for thought speed, $F(1\ 258) = 1.43$, $p = .234$, $\eta_p^2 = .01$, and variability, $F(1\ 258) = 1.40$, $p = .238$, $\eta_p^2 = .01$. Their interaction term was also non-significant, $F(1\ 258) = .19$, $p = .660$, $\eta_p^2 < .01$.

As significant difference in baseline threat interpretation scores were found between participants recruited through the university participant pool and those not, the ANCOVA outlined above was repeated with these two groups included as a covariate. The results remained non-significant, $F_s \leq 1.42$, $p_s \geq .241$.

Secondary Research Question: Associations between Mental Motion and Affective State

Of secondary interest were the predicted differences in affective state dependent on thought speed and variability. The analytic approach employed for the primary research question was repeated with single-item PA and NA as dependent variables: 1) ANCOVA³ assessing differences between manipulation conditions, and

³ Where Levene's test was significant in ANCOVA, square-root data transformation was applied. This action was taken for single-item PA. Transformation exacerbated rather than resolved the violation for PA and did not improve model fit. Consequently, untransformed data were utilised in this instance. Furthermore, the single-item NA ANCOVA and repeated-measures ANOVA for slow thought conditions demonstrated evidence of poorer model fit, which was not resolvable by transformation. Consequently, to improve reliability, PA and NA results should be

2) repeated-measures ANOVA to assess within-condition change pre-to-post manipulation.

Hypotheses four and five: Affective state. Consistent with prediction, ANCOVA demonstrated significant differences between thought speed conditions for both positive, $F(1, 258) = 5.56, p = .019, \eta_p^2 = .02$, and negative affect, $F(1, 258) = 4.33, p = .038, \eta_p^2 = .02$. Participants engaged in fast thought reported significantly higher levels of PA ($M = 61.43, SE = 1.45$) and lower levels of NA ($M = 29.05, SE = 1.56$), compared to the slow condition ($M_{\text{positive affect}} = 56.54, SE = 1.48; M_{\text{negative affect}} = 33.69, SE = 1.58$).

Repeated-measures ANOVAs assessing within-group change pre-to-post manipulation within the slow thought speed condition demonstrated significant decrease in PA, $F(1, 128) = 20.23, p < .001, \eta_p^2 = .14$, and increase in NA, $F(1, 128) = 4.81, p = .030, \eta_p^2 = .04$. No significant changes were demonstrated in the fast thought condition for in either PA, $F(1, 133) = 1.22, p = .271, \eta_p^2 = .01$, or NA, $F(1, 133) = 3.26, p = .073, \eta_p^2 = .02$.

Contrary to prediction, no differences were found between the thought variability conditions for either PA, $F(1, 258) = .62, p = .432, \eta_p^2 < .01$, or NA, $F(1, 258) = .09, p = .766, \eta_p^2 < .01$. Furthermore, no significant interactions (Speed X Variability) were demonstrated ($F_s \leq .46, p_s \geq .497$).

considered with reference to related study findings (e.g., Pronin & Jacobs, 2008; Yang et al., 2014).

Mood State Classification

ISS subscales were employed to generate mood state classifications. Participants were dichotomously categorised as meeting criteria for each of the following categories, respectively: momentary (hypo)mania, depression, euthymia, and mixed-state. Participants were categorised pre and post manipulation. As categorisation included manic and depressive states, the subsequent analyses were relevant to both primary (mania symptoms) and secondary (affective state) research questions.

Four logistic regressions were conducted – one for each mood state category. Participant mood state post-manipulation was entered as the dependent variable. To account for baseline mood, pre-manipulation mood state was entered in the first step as a covariate. Thought speed, thought variability, and their interaction term (Speed X Variability) were entered as predictive variables in the second step. Model fit was good for prediction of (hypo)mania only, consequently the poorer fit of the remaining models should be borne in mind.

Inclusion of the additional mental motion variable predictors produced a significant model against constant only models for (hypo)mania and depression – Table 4. The inclusion of the additional predictors in step two demonstrated significantly improved prediction over baseline covariate for the (hypo)mania and depression models. In both models, only thought speed was found to be a significant additional predictor. Table 4 results indicate that individuals were: 1) more likely to meet criteria for (hypo)mania in the fast thought speed condition compared to slow, and 2) more likely to meet criteria for depression in the slow thought speed condition

compared to fast. However, neither thought variability nor the interaction term, significantly predicted association with either (hypo)mania or depression.

Finally, no significant improvement in model prediction above constant and baseline covariate was demonstrated in step two models for mixed-state, $X^2(3) = 7.13, p = .068$, or euthymia, $X^2(3) = 3.67, p = .299$.

[INSERT TABLE 4 HERE]

Discussion

The present study found supportive evidence for some, but not all, aspects of the mental motion model (Pronin & Jacobs, 2008). The findings most consistently support the mental motion account of manic and affective states. Relationship between mental motion and anxiety was unsupported however, suggesting that the theory requires further development.

Mental Motion and Symptoms of Mania and Anxiety

Based on the mental motion account it was predicted that specific changes in thought speed and variability would be associated with increases in mania and anxiety symptoms, respectively (hypotheses one-three). The predictions were partially supported. Predominantly the mental motion account of manic thinking was supported. Both fast and variable thought were independently associated with increased self-reported activation levels, a core symptom of mania (Mansell & Pedley, 2008). Furthermore, ISS mood state categorisation demonstrated that thought speed predicted momentary (hypo)manic state. However, the predicted between-group differences in anxiety symptoms dependent on mental motion were not observed. Crucially, no support for a combinational influence of thought speed and variability was demonstrated for any dependent variable.

The findings were consistent with previous evidence of increased mania symptoms (e.g., felt levels of energy, power, creativity, and risk-taking) in fast compared to slow thought manipulations (Chandler & Pronin, 2012; Pronin & Wegner, 2006). Thought variability was found to have a significant influence on activation level, whereas in the past a relationship between this variable and 'felt energy' did not meet the significance threshold, $p = .07$ (Pronin & Jacobs, 2008). Collectively, the results support a causal, as well as symptomatic, conception of the thought speed and variability associated with mania. The evidence suggests that rapid stimuli presentation or variable stimuli presentation results in a state of activation. There is potential evolutionary advantage to this relationship as real-world situations requiring rapid processing of information arguably require that the individual is alert and ready for action in the face of potential threat (Pronin, 2013). Arguably, the need to think fast is often accompanied by the need to act fast. The effects of thought variability may serve a similar evolutionary function. Situations in which information is unrelated and consistently changing are difficult to predict, so may also necessitate alertness.

A generic impact of mental motion on activation appears evident. Activation is a core component of mania. However, increased speed and variability does not necessitate clinical levels of mania. Consequently, further factors require consideration to clarify what determines outcome. Factors considered may include individual differences in reactivity to affective change (Gruber, 2011) and the influence of appraisal of internal states (Mansell, Morrison, Reid, Lowens, & Tai, 2007).

Additionally, it was predicted that change in threat perception consistent with decreased threat sensitivity would be evident alongside increased self-reported

activation. However, the evidence did not support this prediction. This study component was acknowledged as exploratory given the mixed evidence of threat sensitivity in mania (Carver & Johnson, 2009; Garcia-Blanco, Salmeron, Perea, & Livianos, 2014) and that existing interpretation bias research has predominantly focused on valence, not threat (e.g., Lex, Hautzinger, & Meyer, 2011; Thomas, Bentall, Knowles, & Tai, 2009). Consequently, the study results cannot be considered conclusive evidence that mania-consistent processing biases are not induced by manipulated changes in thought speed and variability. Further investigation is required.

Contrary to prediction, anxiety symptoms (self-report and threat interpretation) were not related to differences in thought speed and/or variability. Whilst the pace of worried thinking in anxiety has been suggested to demonstrate similarities with manic thinking (Pronin & Jacobs, 2008), anxiety also frequently demonstrates comorbidity with depression (Hirschfeld, 2001) – a condition characterised by slower, ruminative thinking. It has been suggested that anxious worried thought has differently themed content to depressed rumination (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). It may be that association between mental motion and anxiety cannot be accurately observed or understood without considering thought content.

Critically, no evidence was found to support the predicted differentiating role of combined thought speed and variability. This combinational effect has been proposed to be potentially involved in clarifying similarities between some features of anxious and manic thinking styles (Pronin, 2013; Pronin & Jacobs, 2008). Whereas Pronin and Jacobs' (2008) study reported that the combinational effect resulted in significant differences between feelings of anxiety compared to depression. This study found no significant differences when investigating anxiety levels

independently. Consequently, this study failed to support the proposition that the variables of mental motion exert a content-independent *combinational* influence over psychological state on any of the variables investigated (Pronin & Jacobs, 2008). It is possible that the influences of thought speed and variability are solely independent. However, it is also possible that a more nuanced conception of thought variability is required. For example, the conception may incorporate the conceptual thread and progression of thoughts (Mason & Bar, 2011). Thoughts may be variable and non-repeating, whilst also being related through conception associations. This conception is consistent manic episodes as potentially including a “flight of ideas” (American Psychiatric Association, 2013). Further research may include consideration of thematic association in the conception of thought variability.

Mental Motion and Affective State

As predicted (hypothesis four-five), affective experience was influenced by changes in thought speed. Consistent with multiple previous studies (e.g., Chandler & Pronin, 2012; Pronin et al., 2008; Pronin & Wegner, 2006; Yang et al., 2014), fast thought was associated with higher levels of PA and lower levels of NA compared to slow thought. Although within-group change pre-to-post manipulation was consistent with previous results that slow thought may cause decreased PA, this study did not replicate previous findings that fast thought causes significantly increased PA (Pronin et al., 2008 – study six; Yang et al., 2014). However, a recent study that stratified results by depression level only found significant PA change in individuals with mild-moderate depression, whereas change did not meet the threshold for statistical significance in those with minimal or no depression (Yang et al., 2014). Consequently, these findings may suggest that the affective influence of thought speed partially depends on current affective experience (e.g., depression level); and

that, in the general population, slow thought speed demonstrates the strongest influence over mood, decreasing PA and increasing NA. Consistent with this finding, ISS mood state categorisation demonstrated that thought speed predicts depression. The evidence further supports possible relation between thought speed and depression.

Contrary to predictions, affective state was not significantly influenced by thought variability. Previous research which has found an affective influence of thought variability often includes consideration of thought content (Nolen-Hoeksema & Morrow, 1993; McLaughlin et al., 2007) or conceptual interrelationship between thoughts (Mason & Bar, 2011). The present study findings may further indicate the need to consider thought content when investigating the impact of thought variability (Watkins, 2008).

Clinical Implications and Future Research

This research adds to emergent recommendations that psychological intervention may benefit from inclusion of components assessing and manipulating thought speed and variability (e.g., Bar, 2009; Pronin, 2013; Pronin & Jacobs, 2008). A body of literature is demonstrating that thinking style, as well as content, can causally influence psychological experience. This influence may broaden our understanding of the mechanisms of psychological difficulties, such as mania (Pronin & Wegner, 2006) and depression (Yang et al., 2014). Furthermore, this study extends a developing field considering the value of experimental manipulations as interventions as well as research methodologies (Hertel & Mathews, 2011).

Inclusion of therapeutic components targeting mental motion associated with specific psychological difficulties may broaden established cognitive-behavioural

intervention (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). For instance, by increasing thought speed to improve mood in mild-moderate depression (Yang et al., 2014) and, potentially, by inducing slower, more repetitive thought to reduce activation reinforcing factors in individuals with mania.

Furthermore, as these thought processes appear potentially causally related to manifestation of mania symptoms, self-monitoring of thought speed and variability may enhance awareness of current relapse vulnerability and facilitate early intervention (Morriss et al., 2007). Indeed, thought speed (e.g., racing thoughts) is a common prodromal symptom in bipolar disorder (Lam & Wong, 2005). Regular brief assessment of thought speed and variability may enable individuals to be aware of increasing relapse risk factors and engage in targeted intervention to revert thinking to a less elevated pace and level of variability. This advancement is consistent with guidance for relapse prevention in mania (National Institute for Health and Care Excellence, 2006) and may potentially provide additional avenues for support, where currently psychosocial recommendations are limited.

It is stressed, however, that further research is required before intervention development. Investigation in clinical populations is necessary to ascertain if, where, and when manipulation of mental motion provides effective therapeutic change. Existing research suggests that manipulating mental motion may not have the same outcome in severe as in moderate difficulties (Yang et al., 2014).

Limitations

The present study has a number of limitations. First, the study was conducted online rather than in a laboratory. Online research raises concerns regarding experimental control and comparability to existing lab-based evidence. However,

online experiments have demonstrated equivalence with lab-based experiments (Germine et al., 2012). Furthermore, a variation on this study's methodology has been successfully employed online previously (Yang et al., 2014).

Second, consistent with previous research, ISS: activation was utilised as a measure of a symptom of mania. However, increased levels of activity may also present in other difficulties (e.g., Attention Deficit Hyperactivity Disorder). Consequently, whilst levels of activation and positive affect provide preliminary insight into potential mania symptoms, they are not equivalent to (hypo)mania. Future research may expand the present study to include a broader, more representative range of mania symptoms.

Third, a control condition was not included. Although within-group change potentially provides some indication of the impact of deviation of thought speed and variability from 'normal' levels, further investigation may include a non-manipulated comparison control.

Fourth, this study included self-reported thought speed as a manipulation check, but a similar thought variability measure was not included. Although consistent with previous protocol (Pronin & Jacobs, 2008), the omission compromises evaluation of the effectiveness of the variability manipulation. Positively, the significant differences relating to thought variability conditions suggest the manipulation was effective. However, future research should include a specific, repeated measurement to ascertain manipulation effect size and duration. Furthermore, the manipulation check employed was self-report, which is vulnerable to social desirability effects. Inclusion of objective assessment of manipulations (e.g.,

assessing thought speed through response-time in neutral decision-making tasks) would improve future research.

Fifth, in replicating the previous manipulation protocol, manipulation tasks between experimental conditions within this study varied in length. Consequently, the factor of time elapsing may have influenced the results. Furthermore, extraneous variables potentially influenced by the manipulation (e.g., irritation or boredom associated with repetitive or unstimulating tasks) were not assessed. Consequently, their potential contribution to effects observed cannot be discounted. However, relative consistency between present findings and investigation utilising alternative manipulation tasks of varying method and duration (e.g., Pronin et al., 2008) affords confidence that mental motion variables are contributing to the observed effects.

Sixth, the study utilised a convenience, predominantly student sample recruited online and with notable attrition, which may have implications for generalisability. Some demographic differences were observed between study completers and non-completers; however, performance on dependent variables appeared comparable, with the exception of higher PANAS measured negative affective in non-completers. Consistency between the present study and previous research may support generalisability. However, further replication in different populations remains advisable. Additionally, as the present study did not employ a clinical sample, generalisability to a clinical population cannot be assumed.

Conclusion

The present study extends understanding of the independent effects of thought speed and variability on psychological state, specifically focusing of symptoms of mania and anxiety. The study findings suggest that, in its current form,

the model of mental motion may be most relevant to understanding changes in manic and affective states, rather than anxiety. Faster and more varied thought independently contribute to increases in activation, whereas affect appears most significantly influenced slowing thought speed. No significant differences in threat interpretation attributable to changes in mental motion were observed. Critically, the study findings did not support the proposed combinational effects of mental motion variables in differentiating manic and anxious states. Consequently, the theory requires further development, particularly if anxiety is to be incorporated. Future research may aim to generate a more comprehensive theoretical account, incorporating other features of thought, such as content and relationship with that content (e.g., Gillanders et al., 2014; Hayes, Luoma, Bond, Masuda, & Lillis, 2006).

In conclusion, the specificity of the influence exerted by thought speed and variability on psychological state may prove useful for assessment and therapeutic intervention. However, understanding of the specific influence of these variables requires further confirmation and clarification, particularly in clinical populations.

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References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Bar, M. (2009). A cognitive neuroscience hypothesis of mood and depression. *Trends in Cognitive Sciences*, 13, 456-463.
- Bauer, M., Crits-Christoph, P., Ball, W., Dewees, E., McAllister, T., Alahi, P., Cacciola, J., & Whybrow, P. (1991). Independent assessment of manic and depressive symptoms by self-rating: Scale characteristics and implications for the study of mania. *Arch Gen Psychiatry*, 48, 807-812.
- Bauer, M. S., Vojta, C., Kinosian, B., Altshuler, L., & Glick, H. (2000). The Internal State Scale: Replication of its discriminating abilities in a multisite, public sector sample. *Bipolar Disorders*, 2, 340-346.
- Brunyé, T.T, Gagnon, S.A., Paczynski, M., Shenhav, A., Mahoney, C.R., & Taylor, H.A. (2013). Happiness by association: Breadth of free association influences affective states. *Cognition*, 127, 93-98.
- Carver, C., & Johnson, S. (2009). Tendencies Toward Mania and Tendencies Toward Depression Have Distinct Motivational, Affective, and Cognitive Correlates. *Cognitive Therapy & Research*, 33, 552-569.
- Chandler, J., & Pronin, E. (2012). Fast thought speed induces risk taking. *Psychological Science*, 23, 370-374.
- Clark, D. M. (1999). Anxiety disorders: why they persist and how to treat them. *Behavior Research & Therapy*, 37 Suppl 1, S5-27.
- Drost, J., van der Does, W., van Hemert, A.M., Penninx, B.W.J.H., & Spinhoven, P. (2014). Repetitive negative thinking as a transdiagnostic factor in depression

and anxiety: A conceptual replication. *Behaviour Research and Therapy*, *63*, 177-183.

Eckblad, M., & Chapman, L. J. (1986). Development and validation of a scale for hypomanic personality. *Journal of Abnormal Psychology*, *95*, 214-222.

Garcia-Blanco, A., Salmeron, L., Perea, M., & Livianos, L. (2014). Attentional biases toward emotional images in the different episodes of bipolar disorder: An eye-tracking study. *Psychiatry Research*, *215*, 628-633.

Germine, L., Nakayama, K., Duchaine, B., Chabris, C., Chatterjee, G., & Wilmer, J. (2012). Is the Web as good as the lab? Comparable performance from Web and lab in cognitive/perceptual experiments. *Psychonomic Bulletin & Review*, *19*, 847-857.

Gillanders, D. T., Bolderston, H., Bond, F. W., Dempster, M., Flaxman, P. E., Campbell, L., . . . Remington, B. (2014). The development and initial validation of the Cognitive Fusion Questionnaire. *Behavior Therapy*, *45*, 83-101.

Grös, D.F., Antony, M.M., Simms, L.J., & McCabe, R.E. (2007). Psychometric properties of the State-Trait Inventory for Cognitive and Somatic Anxiety (STICSA): Comparison to the State-Trait Anxiety Inventory (STAI). *Psychological Assessment*, *19*, 369-381.

Gruber, J. (2011). Can feeling too good be bad?: Positive emotion persistence (PEP) in bipolar disorder. *Current Directions in Psychological Science*, *20*, 217-221.

Hayes, S.C., Luoma, J.B., Bond, F.W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: Model, processes and outcomes. *Behaviour Research & Therapy*, *44*, 1-25.

- Hertel, P. T., & Mathews, A. (2011). Cognitive bias modification: Past perspectives, current findings, and future applications. *Perspectives on Psychological Science, 6*, 521-536.
- Hirschfeld, R. M. (2001). The comorbidity of major depression and anxiety disorders: Recognition and management in primary care. *Primary Care Companion of the Journal of Clinical Psychiatry, 3*, 244-254.
- Hofmann, S. G., Asnaani, A., Vonk, I. J., Sawyer, A. T., & Fang, A. (2012). The efficacy of cognitive behavioral therapy: A review of meta-analyses. *Cognitive Therapy & Research, 36*, 427-440.
- Hoppitt, L., Mathews, A., Yiend, J., & Mackintosh, B. (2010). Cognitive bias modification: The critical role of active training in modifying emotional responses. *Behavior Therapy, 41*, 73-81.
- Lam, D., & Wong, G. (2005). Prodromes, coping strategies and psychological interventions in bipolar disorders. *Clinical Psychology Review, 25*, 1028-1042.
- McLaughlin, K. A., Borkovec, T. D., & Sibrava, N. J. (2007). The effects of worry and rumination on affect states and cognitive activity. *Behavior Therapy, 38*, 23-38.
- Lex, C., Hautzinger, M., & Meyer, T. (2011). Cognitive styles in hypomanic episodes of bipolar I disorder. *Bipolar Disorder, 13*, 355-364.
- Mansell, W., Morrison, A. P., Reid, G., Lowens, I., & Tai, S. (2007). The interpretation of, and responses to, changes in internal states: An integrative cognitive model of mood swings and bipolar disorders. *Behavioural & Cognitive Psychotherapy, 35*, 515-539.

- Mansell, W., & Pedley, R. (2008). The ascent into mania: A review of psychological processes associated with the development of manic symptoms. *Clinical Psychology Review, 28*, 494-520.
- Marteau, T. M., & Bekker, H. (1992). The development of a six-item short-form of the state scale of the Spielberger State-Trait Anxiety Inventory (STAI). *British Journal of Clinical Psychology, 31*, 301-306.
- Mason, M.F., & Bar, M. (2011). The effect of mental progression on mood. *Journal of Experimental Psychology: General, 141*, 217-221.
- Mathews, A., & Mackintosh, B. (2000). Induced emotional interpretation bias and anxiety. *Journal of Abnormal Psychology, 109*, 602-615.
- Mogg, K., & Bradley, B. (2005). Attentional bias in generalized anxiety disorder versus depressive disorder. *Cognitive Therapy & Research, 29*, 29-45.
- Morriss, R., Faizal, M.A., Jones, A.P., Williamson, P.R., Bolton, C.A., & McCarthy, J.P. (2007). Interventions for helping people recognise early signs of recurrence in bipolar disorder. *Cochrane Database of Systematic Reviews*, CD004854.
- National Institute for Health and Care Excellence. (2006). *Bipolar disorder: The management of bipolar disorder in adults, children and adolescents, in primary and secondary care [CG38]*. London: National Institute for Health and Care Excellence.
- Nesse, R.M. (2000). Is depression an adaptation? *Archives of General Psychiatry, 57*, 14-20.
- Nolen-Hoeksema, S., & Morrow, J. (1993). Effects of rumination and distraction on naturally occurring depressed mood. *Cognition & Emotion, 7*, 561-570.

- Nolen-Hoeksema, S., & Watkins, E.R. (2011). A heuristic for developing transdiagnostic models of psychopathology: Explaining multifinality and divergent trajectories. *Perspectives on Psychological Science, 6*, 589-609.
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*, 400-424.
- Pronin, E. (2013). When the mind races: Effects of thought speed on feeling and action. *Current Directions in Psychological Science, 22*, 283-288.
- Pronin, E., & Jacobs, E. (2008). Thought speed, mood, and the experience of mental motion. *Perspectives on Psychological Science, 3*, 461-485.
- Pronin, E., Jacobs, E., & Wegner, D. M. (2008). Psychological effects of thought acceleration. *Emotion, 8*, 597-612.
- Pronin, E., & Wegner, D. M. (2006). Manic thinking: Independent effects of thought speed and thought content on mood. *Psychological Science, 17*, 807-813.
- Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory (Form Y)*. Palo Alto, CA: Consulting Psychologist Press.
- Taylor, P., & Mansell, W. (2008). "I Get So Energetic and Dominating!" A study of hypomanic personality and conflicting self-perception during activated states in a co-operative task. *Personality & Individual Differences, 45*, 483-487.
- Teachman, B. A., Joormann, J., Steinman, S. A., & Gotlib, I. H. (2012). Automaticity in anxiety disorders and major depressive disorder. *Clinical Psychology Review, 32*, 575-603.
- Tluczek, A., Henriques, J.B., & Brown, R.L. (2009). Support for the reliability and validity of a six-item state anxiety scale derived from the State-Trait Anxiety Inventory. *Journal of Nursing Measurement, 17*, 19-28.

- Thomas, J., Bentall, R. P., Knowles, R., & Tai, S. (2009). Indirect measurement of dysfunctional attitudes in bipolar affective disorder. *Psychology & Psychotherapy: Theory, Research & Practice, 82*, 261-266.
- Vickers, A.J., & Altman, D.G. (2001). Statistical notes: Analysing controlled trials with baseline and follow up measurements. *BMJ, 323*, 1123-1124.
- Watkins, E. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin, 134*, 163-206.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality & Social Psychology, 54*, 1063-1070.
- Yang, K., Friedman-Wheeler, D., & Pronin, E. (2014). Thought acceleration boosts positive mood among individuals with minimal to moderate depressive Symptoms. *Cognitive Therapy & Research*, [Online ahead of print] Retrieved from <http://link.springer.com/article/10.1007/s10608-014-9597-9>.

Table 1

Participant Demographic Information (Including Comparison between Final Sample Participants and Study Non-completers)

Characteristic		% (unless specified)		Chi-square and t-tests
		Final sample <i>n</i> = 263	Non-completers <i>n</i> = 304 (except country, <i>n</i> = 300)	
Sex	Male	27.4	35.9	$X^2(1) = 4.67,$ $p = .031$
	Female	72.6	64.1	
Age	Mean (SD)	27.4 years (11.2)	30.0 years (13.3)	$t(564.52) = 2.55,$ $p = .011$
	Range	51.4 years	81.2 years	
Marital status	Single	52.9	52.3	$X^2(4) = 3.50,$ $p = .478$
	In a couple, not married	27.0	24.3	
	Married	16.3	18.4	
	Divorced	2.7	4.6	
	Widowed	1.1	0.3	
Ethnicity	White	85.6	76.6	N/A ⁺
	Chinese	4.2	2.3	
	Other	10.2	21.1	
Country	UK	62.7	42.3	N/A ⁺
	USA	25.1	40.7	
	Canada	4.2	4.0	
	Other	8	13	
First language	English	89	86.8	$X^2(1) = .60,$

<i>Employment status</i>	Other	29	13.2	$p = .439$
	Full-time employed	22.4	27.3	$\chi^2(6) = 36.32,$ $p < .001$
	Part-time employed	6.8*	18.1*	
	Full-time student	58.9*	36.5*	
	Part-time student	1.5	3.3	
	Unemployed	7.6	8.9	
	Do not work due to disability	1.1	3.6	
	Retired	1.5	2.3	
<i>Diagnosis of mental health difficulties</i>	No	72.6	72.0	
	Depression	16	12.2	
	Anxiety	7.2	6.3	
	Bipolar disorder	.8	4.3	
	Other	3.4	5.3	

*std. residuals $\geq \pm 2.0$; *N/A: Chi-square tests not appropriate as the number of expected counts with a value less than five exceeded 25%.

Table 2

Baseline Raw Data Descriptive Information for Total Sample and Stratified by Manipulation Condition

Manipulation condition:					
<i>Thought Speed X Thought Variability</i>					
Mean variable score (SD)	Slow, repetitive (n = 63)	Slow, varied (n = 66)	Fast, repetitive (n = 64)	Fast, varied (n = 70)	TOTAL SAMPLE (N = 263)
<i>STAI-sf</i>	12.21 (3.92)	11.36 (4.02)	11.63 (4.10)	12.80 (4.05)	12.01 (4.04)
<i>ISS: activation</i>	127.84 (103.23)	122.02 (86.35)	142.06 (104.44)	116.16 (88.04)	126.73 (95.55)
<i>ISS: wellbeing</i>	146.19 (67.46)	157.26 (60.80)	167.59 (64.72)	148.11 (57.18)	154.69 (62.72)
<i>PANAS PA</i>	24.32 (8.50)	26.67 (8.74)	25.72 (8.12)	24.26 (7.27)	25.23 (8.18)
<i>PANAS NA</i>	15.10 (4.66)	15.61 (5.24)	15.53 (5.29)	15.87 (5.26)	15.54 (5.10)
<i>Single-item PA</i>	61.27 (21.57)	67.23 (23.53)	65.84 (22.38)	59.29 (22.87)	63.35 (22.73)
<i>Single-item NA</i>	29.76 (23.75)	25.26 (22.67)*	30.94 (22.44)	37.36 (26.09)*	30.94 (24.10)
<i>AST Threat</i>	20.76 (5.41)	21.59 (5.92)	21.22 (5.85)	21.51 (6.47)	21.28 (5.91)

* Tukey's post-hoc analysis revealed that variable slow and variable fast manipulation conditions significantly differed at baseline on single-item NA, $M = 12.10$ 95% CIs [1.53, 22.67], $SE = 4.09$, $p = .018$.

Table 3

Summary of Estimated Means with 95% Confidence Intervals from ANCOVA Relating to Symptoms of Anxiety, Mania, and Affective State

Dependent variable (post-manipulation)	Thought speed		Sig.	Thought variability		Sig.
	Slow	Fast		Repetitive	Varied	
<i>Estimated Mean [95% CI]</i>						
STAI-sf ⁿ	3.50 [3.44, 3.57]	3.55 [3.49, 3.62]	.314	3.52 [3.45, 3.59]	3.54 [3.47, 3.61]	.636
ISS: activation ⁿ	9.97 [9.29, 10.64]	12.61 [11.95, 13.28]	<.001	10.63 [9.95, 11.32]	11.95 [11.29, 12.61]	.007
Positive affect	56.54 [53.63, 59.46]	61.43 [58.57, 64.29]	.019	59.80 [26.87, 62.74]	58.17 [55.33, 61.01]	.432
Negative affect	33.69 [30.57, 36.82]	29.05 [25.99, 32.12]	.038	31.05 [27.92, 34.17]	31.70 [28.68, 34.73]	.766
Threat interpretation (AST score)	21.45 [20.69, 22.21]	20.81 [20.06, 21.55]	.234	21.45 [20.68, 22.21]	20.81 [20.07, 21.55]	.241
	Interaction					
<i>Estimated Mean [95% CI]</i>	Slow, repetitive	Slow, varied		Fast, repetitive	Fast, varied	Sig.
STAI-sf ⁿ	3.50 [3.40, 3.60]	3.51 [3.41, 3.61]		3.54 [3.44, 3.63]	3.57 [3.48, 3.66]	.836
ISS: activation ⁿ	9.56 [8.60, 10.53]	10.37 [9.43, 11.32]		11.71 [10.74, 12.67]	13.52 [12.60, 14.44]	.300
Positive affect	56.73 [52.56, 60.90]	56.35 [52.27, 60.44]		62.87 [58.73, 67.01]	60.00 [56.02, 63.96]	.551
Negative affect	34.12 [29.68, 38.56]	33.27 [28.90, 37.64]		27.97 [23.56, 32.37]	30.14 [25.89, 34.39]	.497
Threat interpretation (AST score)	21.89 [20.80, 22.97]	21.01 [19.95, 22.07]		21.01 [19.93, 22.08]	20.61 [19.58, 21.64]	.658

ⁿ = Square-root transformed data; Estimated means = Adjusted group means accounting for dependent variable pre-manipulation score as covariate.

Table 4

Logistic Regression Exploring Mood State Predicted by Thought Speed and Variability

Dependent variable	Predictors	95% CI for exp <i>b</i>		
		B (SE)	Lower CI	Upper CI
(Hypo)mania	Constant	-1.84*** (.35)		.16
	Baseline ISS mania classification	2.22*** (.35)	4.66	9.19
	Thought speed	1.07* (.44)	1.23	2.90
	Thought variability	.21 (.46)	.50	1.23
	Speed X Variability	-.07 (.60)	.29	.93
				3.01

$R^2 = .18$ (Hosmer & Lemeshow); $.21$ (Cox & Snell); $.29$ (Nagelkerke)

Model $X^2(4) = 61.71$, , $p < .001$; Step $X^2(3) = 12.70$, $p = .005$

Depression	Constant	-1.21 (.32)		.30
	Baseline ISS depression classification	-2.70*** (.38)	7.13	14.93
	Thought speed	-.94* (.48)	.15	.39
	Thought variability	-.09 (.43)	.39	.92
	Speed X Variability	-.42 (.68)	.17	.66
				2.48

$R^2 = .23$ (Hosmer & Lemeshow); $.24$ (Cox & Snell); $.35$ (Nagelkerke)

Model $X^2(4) = 71.77$, $p < .001$; Step $X^2(3) = 13.00$, $p = .005$

*** $p < .001$; ** $p < .01$; * $p < .05$

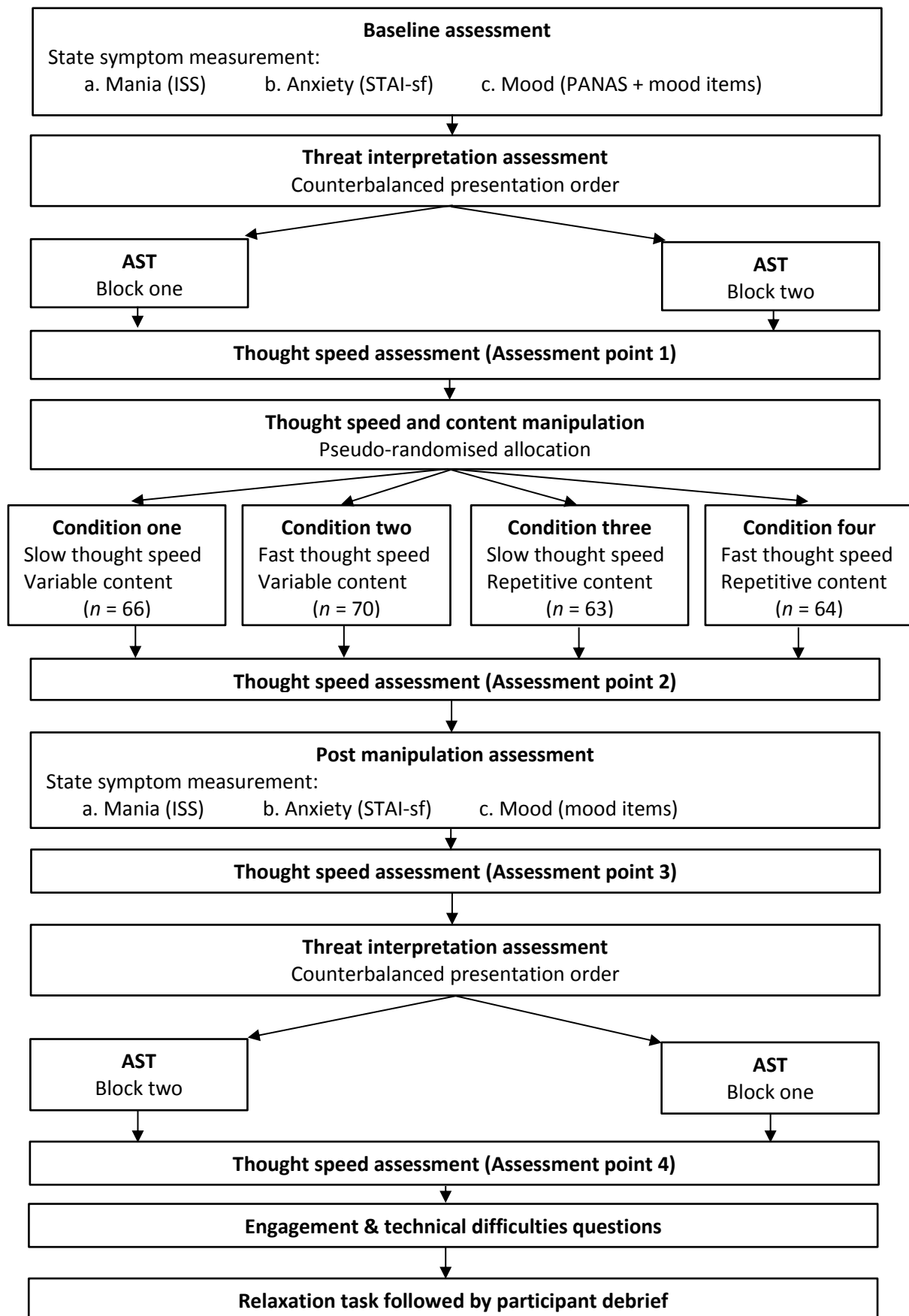


Figure 1. Experimental design and procedural flow.