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Impact of depressive symptoms, self-esteem and neuroticism on trajectories of overgeneral autobiographical memory over repeated trials

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The present study examined trajectories of change in the frequency of overgeneral autobiographical memory (OGM) over the course of repeated trials, and tested whether particular dimensions of depressive symptomatology (somatic and cognitive-affective distress), self-esteem, and neuroticism account for individual differences in these trajectories. Given that depression is associated with impairments in effortful processing, we predicted that over repeated trials depression would be associated with increasingly OGM. Generalised Linear Mixed Models with Penalised Quasi-Likelihood demonstrated significant linear and quadratic trends in OGM over repeated trials, and somatic distress and self-esteem moderated these slopes. The form of these interactions suggested that somatic distress and low self-esteem primarily contribute to OGM during the second half of the trial sequence. The present findings demonstrate the value of a novel analytical approach to OGM that estimates individual trajectories of change over repeated trials.

Overgeneral autobiographical memory (OGM) has been repeatedly demonstrated in depressed and suicidal patients (Dalgleish, Spinks, Yiend, & Kuyken, 2001; Kuyken & Brewin, 1995; Kuyken & Dalgleish, 1995; Moore, Watts, & Williams, 1988; Williams & Broadbent, 1986; Williams & Dritschel, 1988; Williams & Scott, 1988), as well as in a number of additional clinical conditions including posttraumatic stress disorder (McNally, Lasko, Macklin, & Pitman, 1995), acute stress disorder (Harvey, Bryant, & Dang, 1998), obsessive-compulsive disorder (Wilhelm, McNally, Baer, & Florin, 1997), and possibly

borderline personality disorder (Jones et al., 1999; although see also Arntz, Mereen, & Wessel, 2002; Kremers, Spinhoven & van der Does, 2004). When asked to retrieve a specific autobiographical memory (AM; one with a distinct time and place), these individuals instead tend to respond with memories that are general (ones that occurred over a period of time greater than 24 hours or actually consisted of numerous similar memories combined into one). Some data suggest that OGM prospectively predicts a more prolonged course of disorder, at least in the case of episodes of depression (Brittlebank, Scott, Williams, & Ferrier, 1993; Dalgleish et al., 2001: Peeters, Wessel, Merckelbach, & Boon-Vermeeren, 2002; although see also Brewin, Reynolds, & Tata, 1999) and acute stress disorder (Harvey et al., 1998). It may also act as a vulnerability to depressive symptoms in combination with life stressors (Gibbs & Rude, 2004).

Although OGM has been most consistently reported in depressed and suicidal patients, the fact that similar difficulties have been demonstrated in other mental disorders raises the possibility that shared features across these conditions contribute to OGM. For example, there is evidence that a major dimension of psychopathology reflects degree of internal distress (as opposed to ''acting-out'' behavioural problems; Krueger, 1999). These so-called internalising disorders, such as depression and various anxiety disorders, likely emerge from a common dispositional tendency that may be associated with high levels of neuroticism or low self-esteem. For example, self-esteem deficits appear to be involved in a number of disorders (Roberts, in press), while neuroticism has been proposed as a potential diathesis for both depression and anxiety (Clark, Watson, & Mineka, 1994). It may be that these shared characteristics are involved in OGM, and therefore may account for the presence of this memory anomaly across different clinical disorders. On a related note, it is unclear whether particular con-stellations of depressive symptoms play a greater or lesser role in OGM. For example, is OGM primarily associated with the affective components of depression, such as sad mood and low positive affect, or is it more strongly associated with somatic symptoms, such as sleep disturbance, agitation, and fatigue? In this regard, Dalgleish et al. (2001) suggest that OGM prospectively predicts the maintenance of somatic symptoms of depression, but not the affective and cognitive symptoms. The present research was designed to examine the role of self-esteem, neuroticism, and various dimensions of depressive symptomatology in predicting OGM.

In some sense it is not surprising to find that depression is associated with poor performance on the Autobiographical Memory Task (AMT), a task that asks participants to retrieve specific AMs to various cue words. It is well recognised that depression involves cognitive impairment, and numerous studies have demonstrated that depression can be associated with substantial deficits in effortful processing (see Hartlage, Alloy, Vazquez, & Dykman, 1993). Depressed individuals tend to perform poorly on a variety of demanding tasks. As summarised by Gotlib, Roberts, and Gilboa (1996), cognitive performance

may suffer in depression as the result of lowered motivation (Miller, 1975), decreased cognitive capacity or resources (Ellis & Ashbrook, 1988), failure to initiate cognitive strategies (Hertel, 1994; Hertel & Hardin, 1990), failures to inhibit intrusive distracting thoughts (Hertel & Rude, 1991), or more extensive processing of certain stimuli (usually negatively valenced) at the expense of other stimuli (usually positively valenced; Williams, Watts, MacLeod, & Mathews, 1997). Any of these factors on their own or in combination could contribute to poor cognitive performance in depression.

In the context of AM, it may be that OGM is the result of some of these same factors. For example, lowered motivation, deficient attentional resources, and distracting negative thoughts all might make it difficult for depressed individuals to go through the mental operations involved in retrieving specific memories. Over the course of the task, they may fail to maintain the instruction set or find it increasingly difficult to muster the energy to retrieve specific memories. Consistent with this perspective, recent research suggests that reductions in AM specificity may be related to ''secondary goal neglect'' (Dalgleish, 2004). In other words, depressed individuals may sufficiently attend to the primary goal of verbalising memories from their own past experience (autobiographical), while failing to sufficiently attend to the secondary goal of retrieving *specific* memories. For example, Dalgleish (2004) reported that OGM was correlated with poor performance on the WAIS Block Design. Likewise, depressed individuals performed poorly when the instruction set was altered and they were asked to produce OGMs. In this case, they produced more specific memories than nondepressed control participants. Accordingly, it may be valuable to frame the AMT (Williams & Broadbent, 1986) as a cognitive task that requires effortful processing.

From this perspective we would expect that mental fatigue and concentration difficulties would be associated with increased frequency of OGMs and that performance would decline over repeated trials as a function of decreasing attention and concentration. In other words, we would expect a linear trend over trials in which the frequency of OGMs progressively increases. Of most relevance to the present research, we would expect individual differences in these trajectories that vary as a function of factors such as the severity and nature of symptomatology. In particular, according to this perspective, individuals with higher levels of depressive symptomatology would likely show steeper declines in performance over time and would become increasingly overgeneral. This perspective also suggests that specific symptoms of depression are going to be most strongly associated with reductions in specificity. In particular, difficulties with sustained attention, concentration, and motivation would be most closely associated with OGM, particularly over time and repeated trials.

Recent research suggests that the association between depression and reduced AM specificity can be moderated by the nature of self-focused attention (Park, Goodyer, & Teasdale, 2004; Watkins & Teasdale, 2001, 2004). When depressed

individuals engage in highly analytical self-focused processing, in which they think about the causes and meaning of their internal states, they tend to maintain low levels of specificity. In contrast, when they engage in low analysis self-focused processing, in which they simply experience those internal states, they tend to become more specific. The present study tested whether these two modes of self-focused processing moderate the effects of depressive symptoms, self-esteem, or neuroticism on OGM.

The present study was designed to investigate trajectories of OGM over repeated trials, and to determine if specific dimensions of depressive symptoms, self-esteem, or neuroticism are associated with individual differences in these trajectories. Given that depression is associated with impairment in effortful processing, we predicted that these symptoms would be associated with steeper increases in OGM over repeated trials. In order to examine individual differences in trajectories of OGM over repeated trials, we treated data from the AMT (Williams & Broadbent, 1986) as multilevel in nature with repeated trials being nested within participants. Such multilevel level approaches are increasingly used to model individual growth curves (Kenny, Bolger, & Kashy, 2002). Within this framework we can examine both the estimated level of specificity at the start of the trial sequence (intercept) and the rate of change over the sequence (slope). We also tested whether the nature of self-focused attention (analytical vs. experiential) would moderate the effects of depressive symptoms, self-esteem, or neuroticism on trajectories of OGM across trials. Based on previous research demonstrating that experiential processing attenuates the effects of depression on AM specificity (Park et al., 2004; Watkins & Teasdale, 2001), we predicted that depressive symptoms would be associated with steeper increases in the frequency of OGM among those in the high analytical processing condition.

METHOD

Participants

Participants were 204 undergraduate students (112 female) at the University at Buffalo who received credit in partial fulfillment of a course requirement. Participants' mean age was 19.8 ($SD = 5.1$).

Self-report measures

Depressive symptomatology.   The Beck Depression Inventory-II (BDI; Beck, Steer & Brown, 1996) was used to assess depressive symptoms. The BDI is a widely used self-report measure of depressive severity and has been shown to have strong psychometric properties (see Gotlib & Cane, 1989, for a review of this literature). In the present sample, coefficient alpha was .90.

Self-esteem.   The Rosenberg Self-Esteem Scale (Rosenberg, 1979) is a 10-item scale designed to measure global self-regard (e.g., ''On the whole, I am satisfied with myself''). Responses were made on 7-point Likert scales (1 = *strongly agree*; 7 = *strongly disagree*). In the present sample, coefficient alpha was .90.

Neuroticism.   The Eysenck Personality Questionnaire—Revised Short Scale (Eysenck, Eysenck, & Barrett, 1985) was used to measure neuroticism. The neuroticism scale consists of 12 items, such as: ''Does your mood often go up and down?'' and ''Are you an irritable person?'' Participants indicate ''yes'' or ''no'' to each item. In the present sample, coefficient alpha was .83.

Procedures

The study was described to all participants before receiving their informed consent to participate. Participants were run individually. After completing a packet of self-report questionnaires, they were randomly assigned to either a high or low analytical processing condition (Watkins & Teasdale, 2001). In both conditions, participants read the same set of 28 items, such as ''how hopeful or hopeless you are feeling'', ''the way your body feels right now'', and ''how quick or slow your thinking is right now''. Participants focused on these statements for 8 min. In the high analysis condition, participants were instructed to ''think about'' each of the different items (e.g., ''Think about your character and who you strive to be''). In the low analysis condition, participants were asked to focus their attention on the experience of the items (e.g., ''Focus your attention on the experience of the physical sensations you feel in your body''). In contrast to Watkins and Teasdale (2001) we did not include a distraction condition.

Next, the AMT (Williams & Broadbent, 1986) was administered. Participants were asked to recall a specific personal memory in response to six positive, six negative, and six neutral cue words (e.g., *friendly*, *hurt*, and *uncle*, respectively). One list of 18 words was used in which we interspersed words based on valence. Specifically, we presented a positive word, followed by a neutral word, followed by a negative word, followed by a positive word, and so forth. Participants were given 30 s per cue word to recall a memory. Each memory was rated on its specificity (specific for memories with a distinct time and place and that lasted less than 1 day and general for memories that demonstrated a summary of many similar events, that lacked a distinct time and place, or that lasted for over 1 day). Categoric overgeneral memories (i.e., those that summarised many similar events) and extended overgeneral memories (i.e., those that lacked a distinct time and place or that lasted for over 1 day) were not distinguished in our coding. Participants were not prompted with a reminder to retrieve specific memories after incorrectly responding with an OGM. If a participant did not

retrieve a memory in the allotted time period, it was considered an omission. If a participant responded with a semantic or verbal associate rather than a real memory, they were prompted for a memory; if they failed to respond with one, the trial was coded as an omission. Likewise, if the participant responded with a memory from that day they were asked to respond with one from at least the day before, whereas if they responded with an anticipated future event they were asked to retrieve a memory from the past. Of the 3626 scoreable AMT presentations in the sample, there were 2189 (60.4%) specific memories, 1144 (31.5%) overgeneral memories, and 293 (8.1%) omissions. A total of 46 trials could not be scored due to experimenter error. In order to determine reliability, a total of 90 responses were recoded by another rater who listened to audiotapes. Cohen's kappa was .78.

RESULTS

Factor analysis of depressive symptomatology

On average, our sample reported mild levels of depressive symptoms on the BDI (M = 9.9; SD = 8.0, range 0–49). To examine the structure of depressive symptomatology in our sample, a Maximum Likelihood Factor Analysis with Varimax Rotation was conducted on the BDI. Examination of the scree plot, as well as a parallel analysis (Horn, 1965), suggested that the data were best represented by two factors. These factors accounted for 36.2% of the total variance. Factor scales were constructed based on items that loaded greater than or equal to .4 on only a single factor. Items were summed with unit weighting. Factor 1 consisted of 12 items (α = .86) and was labelled ''Cognitive-affective distress'' (items 1, 2, 3, 4, 5, 7, 8, 9, 10, 12, 13, and 14). Item content included sad mood, pessimism, feelings of failure, loss of pleasure, suicidality, and worthlessness. It accounted for 18.8% of unique variance. Factor 2 was composed of 6 items (α = .80) and was labelled ''Somatic distress'' (items 11, 15, 16, 17, 19, and 20). Item content included agitation, loss of energy, sleep disturbance, irritability, concentration difficulties, and fatigue. This factor accounted for 17.4% of the unique variance.

As can be seen in Table 1, the two dimensions of depressive symptomatology were moderately to highly correlated with each other, as well as with self-esteem and neuroticism.

Multilevel analysis of trajectories

Although the number of overgeneral memories is typically summed to create a single total score in studies using the AMT, data can also be viewed as a series of repeated measures that can be modelled with multilevel data-analytical procedures (Kenny et al., 2002; Nezlek, 2001). Viewing our data in this way has the advantage of allowing the modelling of trajectories of change over our

TABLE 1
Correlation matrix, means, and (standard deviations) of dimensions of depressive
symptoms, self-esteem, and neuroticism

	Cognitive-affective distress	Somatic distress	Self-esteem	M	(SD)
Cognitive-affective distress				4.8	(4.7)
Somatic distress	.66			3.9	(3.0)
Self-esteem	−.74	−.59		53.0	(10.9)
Neuroticism	.62	.55	−.61	5.5	(3.5)

*Note*.   All correlations are statistically significant at $p < .001$.

sequence of 18 trials. Within our data, repeated trials are nested within parti-
cipants. From this perspective, each individual has an intercept, reflecting the
participant's estimated level of OGM at the start of the trial sequence, and a
slope, reflecting the participant's estimated linear rate of change over trials. In
addition to the linear slope, higher order polynomials can be estimated, most
commonly the quadratic term. This term reflects the participant's estimated rate
of acceleration of change at a given trial. Given that our dependent variable was
dichotomous (overgeneral vs. specific),[1] primary analyses were conducted with
Generalised Linear Mixed Models with Penalised Quasi-likelihood. Specifically,
we used the glmPQL function of the MASS package (Venables & Ripley, 2002)
run in R 2.0 (R Core Development Team, 2004). Significant interactions were
graphically displayed by plotting estimated scores for each trial conditioned at
1.5 SD above and below the mean for the moderator variable. In the case of
somatic distress, which was a positively skewed variable with a negative value
at 1.5 SD below the mean, we plotted at a value of 0 instead.

Linear and quadratic effects of trial sequence.   Preliminary analyses were
conducted to test the linear and quadratic effects of trial sequence on specificity
in the sample as a whole. We first tested the linear effects of trial, while
statistically controlling for cue valence of stimuli (treated as a dummy variable
with three levels: negative, neutral, and positive). A significant linear trend was
found, $\beta = .082$, $t(3126) = 10.73$, $p < .001$, such that likelihood of retrieving an
OGM increased over time. In order to test whether effects were fixed or random,
we estimated models using the NLME package (Pinehiero & Bates, 2000) run in
R 2.0 (R Core Development Team, 2004), and compared the fit of models in
which intercepts and slopes were fixed vs. random. The linear effect of Trial had
both a random intercept, Likelihood Ratio (LR) = 92.01, $p < .001$, and a random

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[1] Omissions were excluded from the analyses reported below. Results were virtually identical
when OGMs were contrasted with a category that included both specific memories and omissions.

slope, $LR = 9.76$, $p < .01$. In other words, there was significant variability across participants in terms of their baseline probability of retrieving an OGM at the first trial (intercept) and their rate of change over trials (slope). We next tested the quadratic effect of trial sequence (Trial-squared), while statistically controlling for the linear effects of trial and cue valence. The quadratic effect was statistically significant, $\beta = -0.012$, $t(3125) = 6.90$, $p < .001$. Furthermore, the model including the quadratic component provided a better overall fit than the model limited to the linear component, $LR = 38.99$, $p < .001$.

As seen in Figure 1, across participants there was an inverted U-shaped function in which the probability of recalling an OGM increased rapidly during the first half of the trial sequence, and then decelerated and began to decrease during the second half. Although this quadratic effect was statistically significant for the sample as a whole, it was also a random effect, $LR = 8.44$, $p < .05$. In other words, there was significant variability across participants in the degree to which their performance fit this quadratic curve. Our next set of analyses tested whether different dimensions of depressive symptomatology, self-esteem, and neuroticism were associated with variability in this quadratic effect, and whether mode of processing (analytical vs. experiential) would moderate these effects.
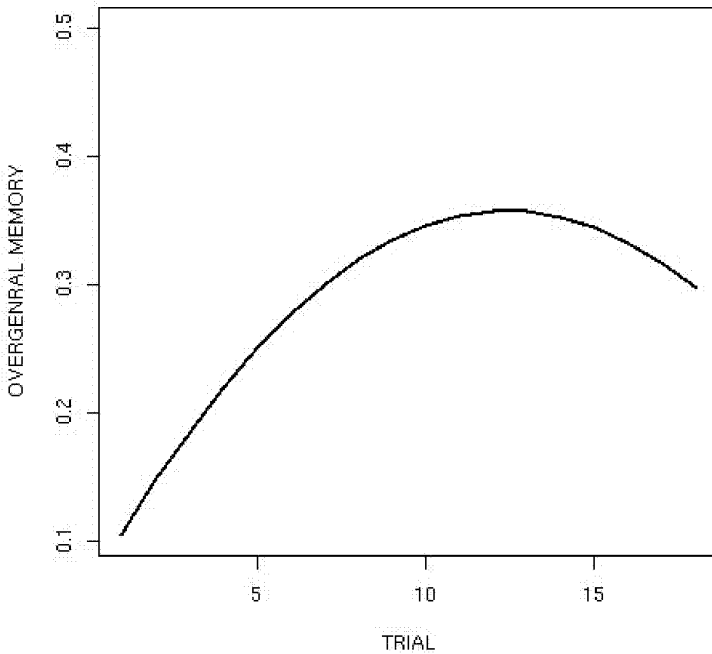
Figure 1.   Quadratic effect of trial number on frequency of overgeneral memory.

Predictors of individual differences in trajectories.   In order to examine factors that might moderate the quadratic effect of trial sequence, models were constructed in a stepwise manner. Step 1 included the main effects of the putative moderator (the two dimensions of depression, self-esteem and neuroticism each tested individually), Trial and cue valence (Val) dummy coded (negative, neutral, and positive). Step 2 included the two-way interaction between the putative moderator and Trial, as well as the quadratic component of trial number (Trial-squared), whereas Step 3 included the interaction between the putative moderator and the quadratic component of Trial. In each of these models, the linear and quadratic components of Trial were treated as random. Initial models included Condition (analytical vs. experiential self-focused processing) as a factor. None of the main effects of Condition or its higher order interactions with the linear or quadratic components of Trial or the dimensions of depression, self-esteem or neuroticism were statistically significant (all ps>.10). Therefore Condition was not included in any of the models reported below.

As can be seen in Table 2, there were significant main effects of Trial and cue valence across analyses. The probability of retrieving an OGM increased over trials, $t = 10.71$, $p < .001$. Likewise, the probability of retrieving an OGM increased with neutral cue words relative to negative cue words, $t = 3.82$, $p < .001$, as well as with positive cue words relative to negative cue words, $t = 8.15$, $p < .001$. In contrast, none of the main effects of the two dimensions of depression (self-esteem or neuroticism) were significant. Likewise, the two-way interactions between these predictors and the linear effects of Trial were not statistically significant.

The primary theoretical question of interest was whether the quadratic trend of Trial is moderated by dimensions of depressive symptomatology, self-esteem or neuroticism. This question is addressed by the interaction term between $Trial^2$ and each of these variables. Given that the quadratic term is represented by Trial $\times$ Trial, these effects are triple interactions (Trial $\times$ Trial $\times$ Putative Moderator). As can be seen at the lower row of Table 2, the $Trial^2 \times$ Somatic distress, $t = 3.27$, $p < .01$, and $Trial^2 \times$ Self-esteem, $t = 3.02$, $p < .01$, interactions were statistically significant. These results indicate that somatic distress and self-esteem each accounted for a statistically significant amount of variance across participants in the quadratic effects of trial number. In other words, the shape of these quadratic curves differed depending on participants' level of somatic distress and self-esteem.

The two significant interactions are graphically displayed in Figures 2 and 3. As can be seen in Figure 2, individuals who were low in somatic distress show a similar inverted U-shaped curve to the one seen in the sample as a whole; specifically they had a rapid increase in the frequency of overgeneral memories, followed by a deceleration and eventual decline of these memories. In contrast, individuals high in somatic distress showed a steady linear increase in the frequency of overgeneral memories over trials that was not followed by

TABLE 2
Multilevel analysis testing individual differences in trajectories of overgeneral memory over repeated trials

| | Somatic distress | | Cognitive-affective | | | | Neuroticism | |
| | | | Distress | | Self-esteem | | | |
	β	t	β	t	β	t	β	t
Step 1: Main effects								
PM	.008	0.30	.011	0.63	-.011	1.42	-.032	1.32
Trial	.087	11.04***	.087	11.04***	.086	11.02***	.087	11.05***
Valence (neutral)	.377	3.97***	.377	3.97***	.377	3.97***	.377	3.97***
Valence (positive)	.786	8.29***	.786	8.28***	.787	8.29***	.787	8.29***
Step 2: w-way interactions								
Cue × Cue	-.013	7.17***	-.013	7.17***	-.012	6.56***	-.013	7.15***
Cue × PM	.002	0.53	.001	0.62	-.000	0.12	-.002	0.61
Step 3: 3-way interactions								
Cue × Cue × PM	.002	3.06**	.000	1.06	-.001	2.88**	-.001	1.39

PM, putative moderator variable; PA, positive affect; β, unstandardised beta coefficient. $*p < .05$; $**p < .01$; $***p < .001$.
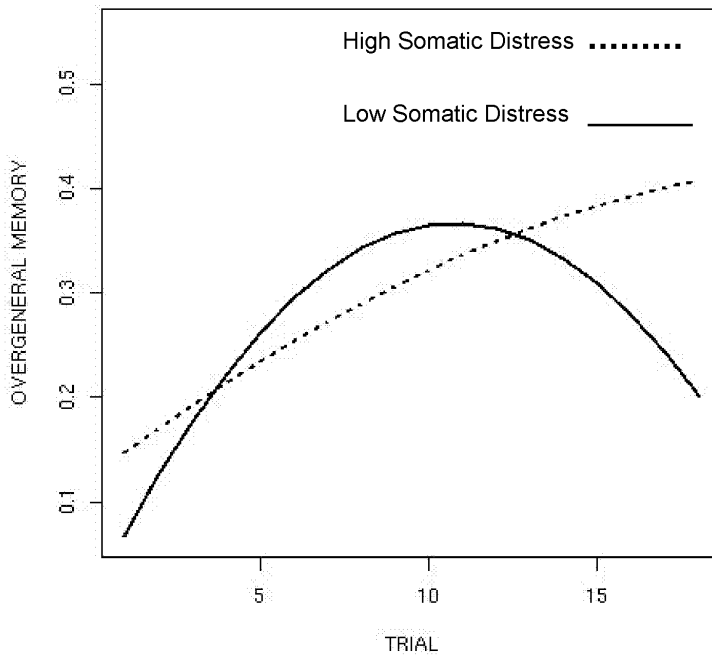
Figure 2.   Depressive Somatic Distress $\times$ Trial^2 interaction on frequency of overgeneral Memory. High Somatic Distress reflects scores estimated from the full equation conditioned at 1.5 standard deviations above the mean. In contrast, because Somatic Distress had a nonmeaningful negative value at 1.5 standard deviations below the mean, low Somatic Distress was conditioned at a value of 0.

deceleration. As can be seen in Figure 3, self-esteem exhibited a similar pattern. Individuals high in self-esteem exhibited a rapid increase in overgeneral memories, followed by a deceleration and decline, whereas those with low self-esteem never decelerated.

   Separate tests across cue valence.   Because cue words were administered in a single fixed order, it is possible that our quadratic effects were driven by the particular words that appeared toward the end of the list. In order to address this concern, we ran follow-up analyses to test the linear and quadratic effects of Trial with each of the three valences of cue words (positive, neutral, negative) separately. Furthermore, we tested whether somatic distress and self-esteem moderated the quadratic terms. Because the original 18-item word list was subdivided into three 6-item lists, any major idiosyncrasies of word order that existed in the original list would likely be eliminated. If effects were generally similar across the three lists, then it would be difficult to argue that our findings were simply due to word order.
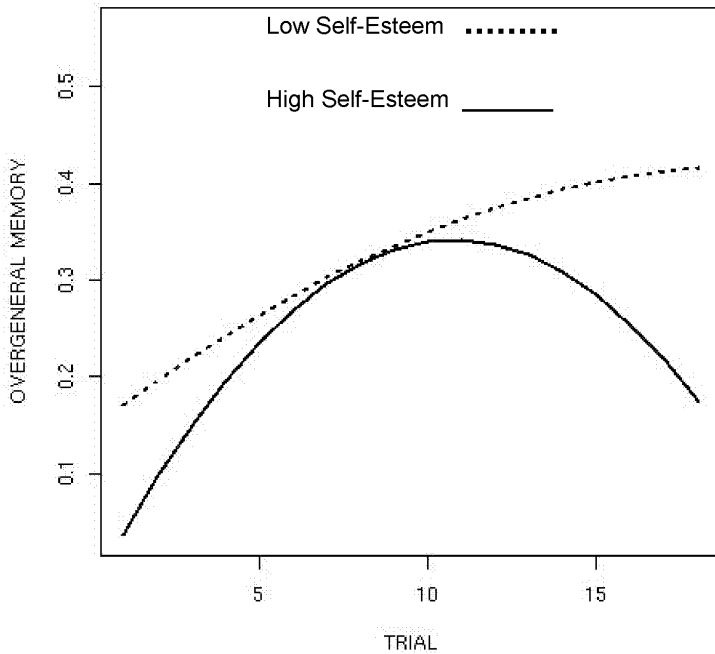
Figure 3.   Self-esteem × Trial^2 interaction on frequency of overgeneral memory. Graph reflects scores estimated from the full equation conditioned at 1.5 standard deviations above and below the mean on Self-esteem.

The linear effect of Trial was statistically significant for positive, $t = 7.13$, $p < .001$, neutral, $t = 2.33$, $p < .05$, and negative words, $t = 8.56$, $p < .001$. Likewise, the quadratic term was statistically significant for positive, $t = 5.76$, $p < .001$, and negative words, $t = 5.59$, $p < .001$, but not for neutral words, $t = 0.76$, $p = .45$. The quadratic term was moderated by somatic distress with neutral, $t = 2.73$, $p < .01$, and negative words, $t = 1.90$, $p < .06$, but not positive words, $t = 1.21$, $p = .22$. Likewise, the quadratic term was moderated by self-esteem with positive, $t = 2.08$, $p < .05$, and negative words, $t = 2.09$, $p < .05$, but not neutral words, $t = 1.48$, $p = .14$. Overall, this pattern suggests generally similar effects across word types, and therefore does not support the word order interpretation.

DISCUSSION

The present study was designed to explore two novel questions related to overgeneral memory in depression. First, this research examined the trajectory of OGM over repeated trials and tested whether or not there was significant variability in these trajectories across participants. Second, this research tested whether particular dimensions of depressive symptomatology, self-esteem and

neuroticism moderated the shape and form of these curves. In other words, if there were individual differences in these trajectories, would characteristics, such as dimensions of symptomatology account for this variability?

The sample as a whole showed both linear and quadratic effects of trial number on OGM. The linear effect suggested that participants became more overgeneral over time. However, this linear effect was qualified by a higher order quadratic effect. The latter was in the shape of an inverted U, and suggested that the frequency of OGMs rapidly increased over the first half of the trial sequence, stabilised during the middle of the sequence, and began to decrease toward the end of the sequence. Although this quadratic effect was significant for the sample as a whole, it was random. In other words, there was significant variation across participants in the magnitude and shape of this effect. The next logical question is what accounts for these individual differences? In the present study we tested whether various dimensions of depressive symptomatology, self-esteem and neuroticism, would moderate the trajectory of OGM over repeated trials.

In our sample, a Maximum Likelihood Factor Analysis with Varimax Rotation suggested that depressive symptomatology was represented by two major dimensions that we labelled cognitive-affective distress and somatic distress. Of these two dimensions, somatic distress was a statistically significant moderator of AM trajectories. Likewise, self-esteem moderated the shape of these trajectories. The form of these interactions suggests that individuals low in somatic distress or high in self-esteem exhibit a rapid increase in the frequency of overgeneral memories during the first half of the trial sequence, followed by a deceleration and decline during the second half. In contrast, among individuals high in somatic distress or low in self-esteem, frequency of OGMs progressively increases over the entire trial sequence without any period of deceleration. Consequently, performance between those low and high in somatic distress or self-esteem is fairly comparable during the first half of the trial sequence—they all rapidly increase in the frequency of retrieving OGMs. In contrast, during the second half of the sequence, participants high in somatic distress or low in self-esteem continue to accelerate in their frequency of OGMs, whereas the trend is reversed among those low in somatic distress or high in self-esteem. In contrast, interactions between the quadratic component of trial sequence and cognitive-emotional distress and neuroticism were not statistically significant.

As discussed in the introduction, it makes sense that symptoms related to somatic distress would be associated with an increased frequency of OGMs, particularly in the later half of the trial sequence. Our dimension of somatic distress included symptoms of psychomotor agitation, concentration difficulties, sleep disturbance, and fatigue that are likely to have a major impact on effortful cognitive tasks. Likewise, low self-esteem appears to impact these processes. In contrast, other types of depressive symptoms, such as those reflecting low positive affect (sad mood, loss of pleasure, loss of interest), and negatively

valenced cognitions (self-criticism, feelings of failure, guilt, and punishment), and the personality dimension neuroticism were not related to trajectories of OGM in our study. These findings suggest that future research on OGM in depression might benefit from examining more fine-grained dimensions of symptomatology. Certain types of depressive symptoms appear to have a greater impact than others. In addition, certain personality correlates of depression, such as low self-esteem, appear to have greater impact than others.

As described above, the frequency of OGMs increased through Trial 10 or so in the sample as a whole, and then began to decrease. It is interesting to consider what might be happening at this point in the trial sequence. One possibility is that the effect is simply an artifact that resulted from the particular cue words that were presented in the final half of the sequence. Although we interspersed positive, negative, and neutral cue words, our study used a single list that was presented in a fixed order. Our analyses statistically controlled for word valence, but it is possible that the linear and quadratic effects of trial number were an artifact of the particular word order used. In other words, effects may have had more to do with the particular words used in the early, middle and late phases of the trial sequence, rather than trial number per se. On the other hand, this possibility would not explain why somatic distress and self-esteem moderated these effects. Furthermore, follow-up analyses demonstrated similar linear and quadratic effects in analyses conducted separately on cue words of each valence (positive, negative, neutral), and in general, somatic distress and self-esteem, moderated these quadratic effects across word types. Nonetheless, it would be important for future studies to use multiple word lists and to vary the order of word presentation across trial numbers to control for this possible confound.

What else might be happening at this point in the sequence? It is possible that participants develop a mnemonic strategy for generating responses early in the task. For example, a participant might try to generate responses based on their work experiences. As the trial sequence continues and the pool of work-related experiences becomes depleted, this particular mnemonic device is likely to become progressively less successful. Perhaps at some point many individuals learn to ''shift gears'' and use a different mental framework, such as generating responses based on their experiences within their families. Given that this pool of memories has not yet been tapped, it would likely prove to be more useful in generating specific autobiographical memories. This account would explain the curvilinear effect with performance steadily worsening up to a certain point and then improving. The point of inflection would be when individuals arrive at a new more effective mnemonic device. Within this account, individuals with greater somatic distress and lower self-esteem would be less able to flexibly switch from one mnemonic device to another. They would stick to their initial strategy despite its ever-decreasing utility, and consequently their performance would decrease in a linear manner with no point of inflection. This account is clearly speculative and little is known about the actual strategies participants use

in the AMT. It would be valuable for future studies to test whether manipulations that change participants' mnemonic devices at various points in the trial sequence subsequently lead to increased specificity in the AMT, particularly among high somatic distress and low self-esteem participants.[2]

Interestingly, it seems likely that symptoms of somatic distress are a common feature of the various clinical disorders in which reductions in AM specificity have been demonstrated; not only would these symptoms be present in depression, they would also likely characterise posttraumatic stress disorder, obsessive-compulsive disorder, and borderline personality disorder. It may be that somatic distress with its concomitant difficulties with concentration and attention is responsible for the increased levels of OGM across these conditions. It also seems likely that low self-esteem is a common feature of these conditions that in turn could contribute to OGM across a range of mental disorders. It should also be noted that nontrauma-related anxiety disorders typically do not demonstrate OGM (e.g., Burke & Matthews, 1992; Wenzel et al., 2002; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001) and several studies have failed to find OGM in borderline personality disorder (Arntz et al., 2002; Kremers et al., 2004). These findings are problematic for our transdiagnostic formulation as it is likely that these conditions are also marked by elevated levels of somatic distress. It would be important for future studies to directly test the degree to which symptoms of somatic distress, such as loss of energy, fatigue, concentration difficulties, and agitation, as well as low self-esteem, are associated with OGM across a range of different mental disorders. It may be that various conditions, such as borderline personality disorder and OCD, are only associated with OGM when they are marked by high levels of comorbid somatic distress symptoms or low self-esteem (see also Kremers et al., 2004; Wilhelm et al., 1997).

Previous studies have shown that OGM has important correlates and functional consequences. For example, OGM is more common among individuals with a history of childhood trauma (Dalgleish et al., 2003; de Decker, Hermans, Raes, & Eelen, 2003; Hermans et al., 2004; Kuyken & Brewin, 1995; although also see Wessel et al., 2001), leading some to speculate that OGM helps regulate negative moods resulting from these adverse experiences. Consistent with this hypothesis, a recent study found that reduced AM specificity was associated with better short-term negative mood regulation (Raes, Hermans, de Decker, Eelen, & Williams, 2003). On the other hand, there is evidence that OGM is associated with problem-solving deficits (Goddard, Dritschel, & Burton, 1996). It is possible that trajectories of change in specificity over trials might serve as a stronger predictor of these and related outcomes. It may prove valuable for future studies to estimate each participant's intercept and slope coefficients and to use these as predictor variables of these and other outcomes (see Young et al.,

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[2] We thank Phil Barnard for suggesting this explanation to us.

1996 for an example based on individual intercepts and slopes involving the association between depressive symptomatology and hopelessness). It may be that estimated baseline levels (intercepts) and estimated rates of downward performance over trials (slopes) are differentially related to outcomes, such as mood regulation and problem solving. Future research should also examine whether the intercepts, linear slopes or quadratic terms of OGM data prospectively predict the course of depressive disorders or risk for the onset of future depressive symptoms. Such work would be informative as to whether OGM plays an etiological role in depression.

Recent research has suggested that a specific cognitive process, analytic self-focused processing (which involves analysing and trying to understand, as opposed to simply ''experiencing'') may contribute to difficulties retrieving specific autobiographical memories in depression (Park et al., 2004; Watkins & Teasdale, 2001, 2004). In contrast, in our study this processing style failed to interact with our two dimensions of depressive symptomatology, self-esteem or neuroticism, in predicting trajectories of OGM. Of note, past research demonstrating the effects of analytical processing was based on clinically depressed samples in contrast to our college sample. It may be that analytical processing has a greater impact on more severely depressed individuals with major depressive disorder compared to individuals with elevated levels of subclinical symptoms. Furthermore, the past studies focused on categoric overgeneral memories in particular, whereas our study collapsed across categoric and extended overgeneral memories.

In terms of methodology, our findings suggest that depression may be more strongly associated with OGM after approximately 12–14 trials. Following this logic, future research may benefit from using lengthier sequences of cue words. Traditionally, 18 cue words are presented on the AMT (Williams & Broadbent, 1986). Our results suggest that differences between individuals with high levels of somatic distress and low self-esteem only emerge with the final few cue words. Increasing the number of cue words potentially will yield a more sensitive index of OGM regardless of whether investigators explore trajectories, as we have done, or simply create an aggregate score.

Given our findings suggesting that OGM in part results from poor concentration and fatigue, it will be important for future research to determine whether OGM is distinct from more general cognitive deficits and poor performance. Does OGM predict psychopathology, early childhood abuse, mood regulation, or problem-solving controlling for performance on other effortful cognitive tasks? Would the addition of secondary cognitive tasks, which increase demand on cognitive resources, amplify the effects of depression and other mental conditions on AM specificity? Does the downward trajectory of memory specificity reflect participants' gradually forgetting the instruction to recall *specific* memories or developing secondary goal neglect? To test this latter possibility, it may be useful to examine changes

in these trajectories following a reminder in which instruction are restated at some point in the series of trials. It also would be useful to examine whether the trajectories are similar when the instruction set is reversed and participants are asked to produce overgeneral memories rather than specific memories (Dalgleish, 2004). It would also be important to examine if similar trajectories arise when participants are given reminder prompts following overgeneral responses. Finally, future research should determine whether symptoms of somatic distress and low self-esteem are the key contributors to OGM in more serious diagnosable depressive disorders, and other mental conditions, such as posttraumatic stress disorder. It may be that this constellation of symptoms helps account for the range of mental disorders that are associated with OGM.

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