

## Executive Function Deficits, Rumination and Late-Onset Depressive Symptoms in Older Adults

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**Abstract** Empirical evidence indicates that late-onset depression (i.e., age of onset  $\geq 60$  years) is associated with executive function decline. This relationship suggests the possibility that executive dysfunction (ED) may contribute to depressive symptoms because it leads to decreased ability to inhibit ruminative thinking. This hypothesis was tested in a sample of 44 older adults reporting depressive symptoms with onset either late in adulthood or earlier in life. Consistent with hypotheses, older adults suffering from late onset, but not early onset, depressive symptoms showed an association between ED and depressive symptomatology. Furthermore, this selective relationship between ED and depressive symptomatology was mediated by ruminative tendencies. These results suggest that executive function deficits may contribute to late-onset of depressive symptoms by interfering with the ability to control ruminative thoughts.

**Keywords** Late-onset depression · Rumination · Executive function · Inhibition

Late adulthood is marked by major life changes. Many of these changes are positive, such as the birth of grandchildren and the increased leisure time that comes with retirement. However, other changes may be significant sources of stress and unhappiness, such as frequent health problems, relocation, death of peers and loved ones, and loss of social and financial status. As in other stages of life, such negative life events bring increased risk for depressive symptoms (Fiske, Gatz, & Pedersen, 2003; Nolen-Hoeksema & Ahrens, 2002), especially when they are associated with increased ruminative thinking (Bonanno et al., 2002; Bonanno, Papa, Lalande, Zhang, & Noll, 2005; Nolen-Hoeksema, Parker, & Larson, 1994). It is particularly poignant that such changes coincide with a stage of life at which notable deficits in

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inhibitory ability and other executive functions are likely to emerge (Hasher & Zacks, 1988; Persad, Abeles, Zacks, & Denburg, 2002). It is our hypothesis that this unfortunate convergence in cognitive, social, and physical events may be a potent mixture in the genesis of depressive symptoms, by providing triggers for depression while simultaneously robbing the individual of important mental capacities involved in the control of depressogenic cognitive tendencies.

Just as declines in executive functions—including inhibitory ability, attentional control, cognitive flexibility, working memory, and problem-solving—have been associated with aging (e.g., Hasher & Zacks, 1988; Persad et al., 2002), so too are such deficits associated with depression (Channon & Baker, 1996; Channon, Baker, & Robertson, 1993; Merriam, Thase, Haas, Keshavan, & Sweeney, 1999; Mialet, Pope, & Yurgelun-Todd, 1996; Trichard et al., 1995; Sweeney, Strojwas, Mann, & Thase, 1998). Although depression may itself cause such cognitive deficits, there is also reason to believe that at least some aspects of executive dysfunction (ED) may contribute to vulnerability to and maintenance of depression by impairing control of depressogenic patterns of thinking (Crews & Harrison, 1995; Davis & Nolen-Hoeksema, 2000; Hester & Garavan, 2005; Martin, Oren, & Boone, 1991; Muraven, 2005; Watkins, Teasdale, & Williams, 2000). For example, Davis and Nolen-Hoeksema (2000) reported evidence consistent with the hypothesis that rumination is a manifestation of a more general pattern of cognitive perseveration or inflexibility, suggesting that people who lose the ability to think flexibly may increase in their ruminative tendencies.

Executive dysfunction is particularly apparent in depression having its initial onset in older adulthood (typically defined as at or after 60 years of age; for a review see Alexopoulos, 2003). This suggests the possibility that ED may contribute to depressive symptoms because it brings decreased capacity for self-regulation in the face of negative life events. Additionally, age-related ED may increase vulnerability to depression among older adults who may have been prone to depressogenic patterns of thinking throughout their lives. For example, in keeping with the hypothesis of Davis and Nolen-Hoeksema, individuals prone to ruminative responses to negative life events may become increasingly prone to rumination as they age due to declines in their ability to inhibit such thoughts or redirect their attention.

In sum, we propose that an inability to exert effective executive control over persistent negative or ruminative thoughts may contribute to depressive symptomatology in older adulthood. According to this hypothesis, late-onset depressive symptoms are more likely to be associated with deficits in inhibitory function and executive control than are early-onset depressive symptoms (for which poor inhibition is less likely to be a central causal factor). As such, among depressed older adults, late onset of symptoms should be associated with poor inhibitory ability, whereas early onset of symptoms may or may not be associated with inhibitory ability. Moreover, the relation between ED and late-onset depressive symptoms should be mediated by rumination. Thus, the present study tested the prediction that age of depressive symptom onset (early or late) should interact with ED such that symptoms should be more strongly related to ED in the late-onset group. Furthermore, rumination was predicted to mediate the relation between this interaction and depressive symptoms. That is, we predict a pattern of mediated moderation (see Baron & Kenny, 1986) in which the stronger association between ED and depressive symptoms in older adults with late rather than early onset of depressive symptoms is mediated by ruminative responses.

To test these hypotheses, we assessed depressive symptom severity, age of onset of depressive symptoms, and ED in a group of older adults who were recruited because they suffer from depressive symptoms.

## Method

### Participants

Forty-four older adults (age 66–92 years,  $M = 82.2$ ,  $SD = 7.0$ ; 38 female) in Sydney, Australia participated in return for 30 Australian dollars (at the time, ~\$20 USD). Participants were recruited from retirement communities and similar settings by distributing fliers that solicited participation from older adults who were “feeling blue or depressed.” These fliers were followed up by contacting relevant program staff persons to identify participants and to help determine eligibility of volunteers. Staff persons and potential participants were asked for information concerning the following inclusion criteria: (1) age 65 years or older; (2) adequate eyesight for reading; (3) fluent in English; (4) no diagnosis of dementia; and (5) free from major psychoactive medications that may interfere with cognitive functioning (e.g., anti-psychootics and sedatives).<sup>1</sup> Staff persons identified qualifying potential participants and approached them to determine if they were willing to participate. As a consequence of this recruitment strategy, we lack data regarding how many potential participants were approached but were unwilling or unable to participate. Additionally, one participant began the experiment but discontinued due to difficulties with the materials.

Age of onset cut-offs for defining late-onset depression have varied (Lyness, Pearson, Leboitz, & Kupfer, 1994). In the present investigation, we chose onset at age 60 years or older because it appears to be the most commonly used cut-point (e.g., Alexopoulos, Young, & Meyers, 1993; Baldwin, 1990; Elderkin-Thompson et al., 2003; Holroyd & Duryff, 1997; Kiosses, Klimstra, Murphy, & Alexopoulos, 2001; Lavretsky, Lesser, Wohl, & Miller, 1998). Based on this cut-off, 20 participants (18 female) fell in the late-onset depressive symptom group ( $M_{\text{onset age}} = 76.2$  years,  $SD = 9.1$ ) and 24 participants (20 female) fell in the early-onset group ( $M_{\text{onset age}} = 28.4$  years,  $SD = 15.6$ ).

### Measures

#### *Ruminative Responses Scale—Short form (RRS; Davis & Nolen-Hoeksema, 2000)*

This scale assesses the tendency to respond to negative emotions with ruminative thoughts. Participants respond to items (e.g., “Think about why you always react this way.”) on a 4-point scale anchored by “almost never” and “almost always”. Davis and Nolen-Hoeksema (2000) developed the short form of the RRS (Nolen-Hoeksema & Morrow, 1991) by selecting the ten items from the original 22-item RRS that correlated most strongly with the total RRS score in a large community sample and for which at

<sup>1</sup> Although we screened for major psychoactive drugs, there are a very large number of other drugs that may interfere to some extent with cognitive processing for which we did not screen.

least 15% of the sample endorsed a response other than “almost never.” The RRS-Short Form has good internal consistency ( $\alpha = .85$  in the present sample) and Davis and Nolen-Hoeksema report excellent correspondence with the full 22-item version of the RRS ( $r = .93$ ).

*Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996)*

The BDI-II is a 21-item self-report measure used to assess depressive symptomatology. For each item, respondents choose from among four statements the one that best describes their experience of depressive symptoms over the past 2 weeks. Scores may range from 0 to 63. BDI-II scores are commonly divided into four ranges: 0–13 = *minimal depression*, 14–19 = *mild depression*, 20–28 = *moderate depression*, and 29–63 = *severe depression* (Beck et al., 1996). Beck et al. (1996) report high test–retest reliability and internal consistency for the BDI (in the present sample,  $\alpha = .88$ ).

*Geriatric Depression Scale-15 (GDS; Sheikh & Yesavage, 1986)*

The GDS-15 is a shortened version of the full GDS (Yesavage et al., 1983). The GDS-15 has acceptable psychometric properties (D’Ath, Katona, Mullan, Evans, & Katona, 1994; Leshner & Berryhill, 1994; Sheikh & Yesavage, 1986) and high sensitivity as a screening measure of depression (Almeida & Almeida, 1999; D’Ath et al., 1994; de Craen, Heeren, & Gussekloo, 2003). GDS-15 scores are commonly divided into three ranges: 0–4 = *not depressed*, 5–10 = *mild to moderate depression*, and 11–15 = *severe depression*. Consistent with past studies, the GDS had acceptable internal consistency in the present sample ( $\alpha = .76$ ).

*Stroop color naming task (Stroop, 1935)*

A typical card format Stroop task was used. Participants were first shown a laminated A4 sized card containing 44 color blocks arranged in three columns, and were then shown a similar card with the color blocks replaced by 44 color words in 28 point font. The words were color names (red, blue, green, purple, and brown). Words were printed in these five colors such that almost all color names were printed in an incongruous ink color. Participants were instructed to name the color of the ink while ignoring the meaning of the words. The index of color-naming interference was the latency to name words minus the latency to name blocks (Serra-Mestres & Ring, 2002).

*Paragraph reading task (Connelly, Hasher, & Zacks, 1991)*

In this task participants were presented with seven paragraphs describing everyday events. The paragraphs were written in italic script in 16 point font, and four of them contained distracting phrases interspersed throughout. The distracting phrases were written in upright script. Participants were asked to read the paragraphs out loud as close to a normal speed as possible. They were told to read only the text in italics, and to avoid reading any text in upright script. The paragraphs were intermixed such that those containing no distracting text were presented in between those containing distracting text. Participants were audio-recorded as they read the paragraphs, so that they could

later be timed. Paragraph reading scores were computed as the average difference in reading time between the paragraphs with distracting text and the paragraphs without distracting text.

*Working memory test (Daneman & Carpenter, 1980)*

This task requires participants to read simple sentences and recall the last word of each sentence after reading a series of sentences. The task consisted of 15 trials: 5 trials each containing a series of 2, 3, or 4 sentences. Each sentence ranged from 10 to 13 words long, and the last word of each sentence was different. The sets were presented beginning with 5 sets of 2 sentences, followed by 5 sets of 3 sentences and finally 5 sets of 4 sentences. The sentences were typed on individual cards in 16 point font and were held for participants to read aloud at a comfortable distance. The task was demonstrated using two practice sentences. Participants were told they would be required to remember more words as the task progressed. After participants read all of the sentences in each series (only one sentence was visible at a time), the experimenter covered the sentences and asked the participant to recall the last word of each sentence in the series. After each series, participants were told they could forget the last set of words and focus on the next sentences presented. Working memory was scored as the total number of words that participants correctly recalled on trials in which they made no errors (May, Hasher, & Kane, 1999).

*Wisconsin Card Sorting Task-64 (WCST-64; Greve, 2001)*

The WCST presents participants with a series of cards for which the participant must discover a rule according to which the cards are to be sorted (i.e., according to the shape, color, or number of symbols on each card). Limited instructions are available from the experimenter, who responds only “right” or “wrong” to each answer. The participant must deduce from this feedback the correct sorting of cards. After ten consecutive correct placements, the examiner shifts the sorting rule, indicating the shift to the participant only by a changed pattern of feedback. This is continued until six category shifts are achieved or all cards are used.

The shortened 64 card version of the WCST was utilized due to concern about potential fatigue of the participants. Several studies have provided good support of the validity and reliability of the WCST-64 (Greve, 2001; Purdon & Waldie, 2001). The number of perseverative errors (defined as a repetition of an incorrect response despite feedback) on the WCST provides a measure of inhibitory functioning. The WCST was administered manually, and perseverative errors were scored according to the standardized scoring system (Heaton et al., 1993; Berry, 1996).

*Shipley vocabulary test (Zachary, 1986)*

Due to the substantial number of tests being administered, a 10-item version of the Shipley vocabulary task (Zachary, 1986) was included as a very brief measure of vocabulary. In this shortened version of the Shipley vocabulary test, every fourth item was chosen from the original test. Ten target words were presented, with participants required to choose for each target word which of four possible words is most similar in meaning. Level of difficulty increased with each item.

This shortened version of the Shipley has the advantage that it minimizes fatigue compared to the full test. Additionally, it has been used with community dwelling and institutionalized (i.e., residents of an assisted-living elder hostel) older adults, where it was found to correlate with short term memory span, age, and whether participants were institutionalized (von Hippel et al., 2005). It should be noted, however, that the Shipley is but one of many tests that would ideally be given to measure general cognitive functioning, in order to isolate the effect of executive function decline from overall cognitive decline. Nevertheless, because such cognitive tests are fatiguing, and because the current testing session was already long, the decision was made to focus on the proposed relationship between ED and late-onset depression, leaving for future research the goal of disentangling the effects of ED from other types of cognitive decline.

### Procedure

Assessments were conducted by a trained psychology student at a place of the participants' choosing, typically in their apartment or in a private area of their retirement village. Participants were initially interviewed regarding demographic information to ensure that they did not meet any exclusion criteria for the study. Next, they completed the following tasks in a fixed order: (1) Stroop color-naming task, (2) the Paragraph Reading Task, (3) the RRS, (4) the GDS, (5) the BDI-II, (6) the WCST, (7) the Working Memory Test, and (8) the Shipley Vocabulary Test.

Age of onset of first significant episode of depression was assessed based on participant self-report. Specifically, immediately following their completion of the two depression questionnaires, participants were asked to report the age at which they first experienced significant levels of the sort of depressive symptoms described in the GDS and BDI-II. Thus, we used the items on the GDS and BDI-II to provide a consistent context in which participants would have a clear understanding of depressive symptoms when reporting the first time they experienced significant levels of depression.

## Results

### Preliminary analyses

Descriptive statistics for the main study variables, separated by age of onset, appear in Table 1. Correlations among the study variables appear in Table 2. The age of onset groups did not differ on age or RRS scores. Neither did they differ on BDI-II or GDS scores. On the BDI-II, based on ranges specified by Beck et al. (1996), 32% fell in the minimal depression range ( $\leq 13$ ), 22% in the mild (14–19), 23% in the moderate (20–28), and 23% in the severe depression range ( $> 28$ ). On the GDS, based upon ranges specified by Brink and Yesavage (1982), 37% of the sample scored in the non-depressed range ( $\leq 4$ ), 52% scored in the mild range (5–9), and 11% scored in the moderate to severe range ( $\geq 10$ ) of depressive symptoms. Because BDI-II and GDS scores were strongly correlated ( $r = .77$ ), they were standardized and averaged to create a composite measure of depressive symptoms. As shown in Table 1, the groups also did not differ on this composite measure. With regard to ED measures, the age of onset groups differed on the Working Memory measure as well as a composite measure of ED (see below), with the late-onset group performing worse in each case. The groups also differed in their scores on the Shipley vocabulary test with the late-onset group performing worse

**Table 1** Descriptive statistics by age of onset group

Variable	Early-onset group mean ( <i>SD</i> )	Late-onset group mean ( <i>SD</i> )
Age	78.2 (9.1)	82.6 (7.3)
Age at onset*	28.4 (15.6)	76.2 (9.1)
Shipley vocabulary*	8.1 (1.8)	6.6 (1.8)
Working memory*	20.2 (8.8)	12.9 (7.5)
Stroop index	34.3 (14.7)	50.1 (39.5)
Paragraph reading index	69.0 (46.6)	80.9 (63.5)
WCST perseverative errors	19.0 (9.1)	21.2 (13.0)
ED composite*	-.24 (.71)	.39 (.98)
RRS rumination	2.5 (.61)	2.4 (.56)
BDI-II	19.8 (9.1)	18.3 (11.1)
GDS	5.8 (3.7)	5.7 (3.0)
Depressive symptom composite	.48 (.93)	.39 (.92)
BDI-II cognitive affective index	6.8 (4.7)	6.5 (5.4)
BDI-II somatic/motivation index	13.0 (5.1)	11.8 (6.4)

Asterisked variables differ between groups  $P < .05$

than the early-onset group. Finally, the groups did not differ significantly with regard to gender (Fisher's exact test  $P = .67$ ).

#### *Derivation of the ED composite score*

The four measures of ED were combined into a single index by standardizing each and averaging. This score demonstrated moderate internal consistency ( $\alpha = .65$ ). However, reliability analysis also revealed that the internal consistency of the scale would increase if WCST perseverative errors score were dropped. Additionally, the WCST was missing for several participants. Therefore, to improve internal consistency and include all participants' data, a three-element composite was computed. This score possessed acceptable internal consistency ( $\alpha = .70$ ) and was highly correlated with the four element composite score ( $r = .93$ ). Therefore, the three-element composite was used in all remaining analyses.

#### Main analyses

The relation between the age of onset Group (early onset = 0, late onset = 1) and depressive symptoms was examined through a series of hierarchical regression analyses. In each analysis, Gender, Age, Shipley Vocabulary, and a variable indicating whether a participant was widowed or not (widowed = 1, not widowed = 0) were entered into the model in Step 1, along with Group and ED Composite. In Step 2, a product term was entered representing the Group  $\times$  ED interaction. It should be noted that the Group and ED main effects were standardized prior to computing this interaction, according to the guidelines provided by Aiken and West (1991). Finally, in Step 3, the RRS score was added to the model.

In the first model tested, the dependent variable was the depressive symptom composite score. As shown in Table 3, the addition of the Group  $\times$  ED interaction produced a significant increment in  $R^2$ . Finally, the addition of the RRS score also produced a significant increment in  $R^2$ . Consistent with predictions, RRS scores appeared to mediate the Group  $\times$  ED interaction. As depicted in Fig. 1, the addition of RRS caused the regression coefficient for the Group  $\times$  ED interaction to become non-significant.

**Table 2** Correlations among measures

Variable	Gender	Age	Widow	Shipley vocab.	Working memory	Stroop index	Paragraph reading index	ED comp	Age of onset group.	RRS	BDI-II	GDS	Dep. comp.	Cognitive-affective index
Age	-.06													
Widowed	-.25	.61*												
Shipley vocabulary	.23	-.49*	-.28											
Working memory	-.10	-.37*	-.09	.37*										
Stroop index	.08	.21	-.03	-.17	-.40*									
Paragraph reading index	.09	.15	-.08	-.28	-.38*	.53*								
ED comp	.13	.34*	.04	-.37*	-.78*	.81*	.82*							
Age of onset group	-.10	.26	.12	-.39*	-.41*	.27	.15	.36*						
RRS	.15	-.14	-.06	.06	-.02	.26	.04	.11	-.08					
BDI-II	.18	-.11	-.10	.03	-.04	.22	.14	.15	-.08	.64*				
GDS	-.01	.07	.04	.02	.00	.09	.10	.10	-.01	.52*	.77*			
Depressive symptom composite	.09	-.02	-.03	.03	-.02	.17	.13	.13	-.05	.62*	.94*	.94*		
Cognitive-affective index	.13	-.19	-.16	.02	.06	.22	.12	.13	-.03	.69*	.93*	.66*	.84*	
Somatic/motivation index	.19	-.03	-.04	.04	.01	.20	.14	.14	-.11	.53*	.95*	.78*	.92*	.76*

\**P* < .05



**Table 3** Regression analysis predicting composite depressive symptoms

	Step and predictor				SE	
	<i>B</i> <sup>a</sup>	<i>B</i>	$\beta$	<i>t</i>	<i>R</i> <sup>2</sup>	$\Delta R^2$
Step 1					.05	.05
Step 2					.15*	.10*
Constant	2.10	2.27		.92		
Gender	.43	.47	.16	.91		
Age	-.03	.03	-.23	-.94		
Widowed	.34	.43	.18	.78		
ShIPLEY vocabulary	-.02	.10	-.03	-.16		
ED composite	.10	.18	.11	.53		
Group	-.08	.17	-.08	-.46		
Group $\times$ ED composite	.34	.17	.35	2.03*		
Step 3					.41*	.25**
Constant	-1.98	2.22	-.89			
Gender	.09	.41	.03	.22		
Age	.00	.02	.01	.02		
Widowed	.08	.37	.04	.21		
ShIPLEY vocabulary	.01	.08	.02	.10		
ED composite	.02	.15	.02	.14		
Group	-.01	.14	.01	-.07		
Group $\times$ ED composite	.15	.15	.16	1.01		
Rumination (RRS)	.90	.24	.57	3.76**		

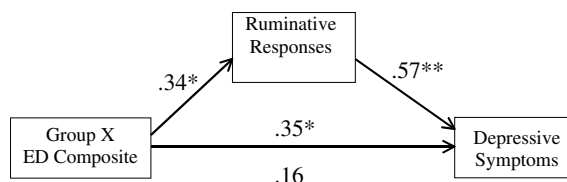
\**P* < .05; \*\**P* < .01<sup>a</sup>Coefficients omitted for Step 1

Note: Coding for Widowed: 0 = not widowed; 1 = widowed; Coding for Gender was 0 = female; 1 = male

The significance of this mediation effect was tested using the  $z' = \alpha\beta/\sigma_{\alpha\beta}$  method described by MacKinnon, Lockwood, Hoffman, West, and Sheets (2002). In their comparison of methods to test mediation effects, MacKinnon et al. (2002) identified this approach as being superior in statistical power to most alternatives (e.g., the Sobel test). In this case,  $z' = 1.79$ ,  $P < .05$ .<sup>2</sup> and thus the Group  $\times$  ED interaction's association with depressive symptoms was significantly mediated by rumination. To examine the nature of this mediated moderation, we examined correlations separately for the late-onset and early-onset groups. In the late-onset group, ED was marginally significantly correlated with depressive symptoms ( $r = .42$ ,  $p = .063$ ) and rumination ( $r = .41$ ,  $P = .075$ ). When rumination scores were partialled out, the correlation between ED and depressive symptoms was reduced (semi-partial  $r = .18$ , ns). In contrast, in the early-onset group ED was not significantly correlated with either depressive symptoms ( $r = -.14$ , ns) or rumination ( $r = -.12$ , ns). Moreover, the relation between ED and depressive symptoms was largely unchanged when rumination was partialled out (semi-partial  $r = -.07$ , ns).

To further explore this pattern of associations, we computed separate scores representing cognitive-affective and somatic-motivational symptoms of depression based on the BDI-II as described by Beck et al. (1996). Similar dimensions have been found in a number of factor analytic studies of the BDI-II (Beck et al., 1996; Storch, Roberti, & Roth, 2004; Whisman, Perez, & Ramel, 2000). Separating these two aspects of depressive symptoms may be especially important when studying depression in older adulthood because depressive symptom scores may potentially be inflated due to age-related somatic-motivational complaints that may reflect medical conditions rather

<sup>2</sup> As discussed by MacKinnon et al. (2002),  $z'$  uses a distribution other than the normal distribution. Thus, critical values differ from those associated with typical  $z$  tests. Critical values are available at <http://www.public.asu.edu/~davidpm/ripl/methods.htm>.



**Fig. 1** Mediated moderation model depicting relationship between the Group  $\times$  ED Composite interaction, ruminative responses, and depressive symptoms composite. Path coefficients are standardized beta weights. The path coefficient above the arrow from Group  $\times$  ED Composite to depressive symptoms represents the effect when the mediator is not included in the model. The path coefficient below the arrow represents the effect when rumination is included as a mediator. \* $P < .05$ ; \*\* $P < .01$

than depression (Fiske, Kasl-Godley, & Gatz, 1998; McNeil & Harsany, 1989; Zemore & Eames, 1979).

When the regression analysis was repeated on the cognitive-affective symptom index, the pattern of results was very similar to that observed for the depressive symptom composite. Again, the test of the mediation effect indicated that rumination was a significant mediator of the relation between the Group  $\times$  ED interaction and depressive symptoms,  $z' = 1.86$ ,  $P < .05$ . This same pattern of results was observed when the somatic/motivational symptom index was used as the dependent variable ( $z' = 1.63$ ,  $P < .05$ ).

## Discussion

The results of this study provide support for a link between rumination and ED in late-onset depressive symptoms. This conclusion is supported by three related results. First, consistent with other research, older adults reporting late-onset depressive symptoms demonstrated poorer performance on a composite measure of executive function than those reporting onset of symptoms earlier in life. Second, age of onset interacted with ED in predicting rumination scores, such that ED was positively correlated with rumination in the late-onset group whereas it was not related in the early-onset group. As an aside, it is noteworthy that past studies reporting an association between ED and depression in older adults have focused on individuals meeting diagnostic criteria for a depressive disorder. Results of the present study suggest that this association exists even in a non-clinical sample. Third, when rumination scores were added to the regression model predicting depressive symptoms, the interaction between ED and age of onset became non-significant. This reduction in the interaction's relationship to depressive symptoms when rumination was added to the model was itself significant. This pattern of results was also found when depressive symptoms were divided into cognitive-affective symptoms versus somatic-motivational symptoms.

These results are consistent with a model wherein age-related declines in inhibitory ability and other executive functions lead to impaired ability to regulate ruminative responses to negative affect and thereby contribute to the development, maintenance, and/or intensification of depressive symptoms in older adulthood. However, it should be noted that the current study was cross-sectional and correlational in design. Thus, although the results are consistent with such a model, they are insufficient by themselves to establish causality. Consequently, it is possible that late-onset depressive symptoms

lead to ED and ruminative tendencies, or that some third factor causes ED, late-onset depressive symptoms, and rumination. To rule out such possibilities, prospective studies will be necessary to establish the causal role of inhibitory deficits in late-onset depressive symptoms among older adults. The current results provide an evidentiary basis for such an investment in longitudinal research.

It should be noted, however, that at least one recent longitudinal study of older adults found that cognitive impairment predicted increases in depressive symptoms over a 4-year-period, whereas depression did not predict later cognitive impairment (Vinkers, Gussekloo, Stek, Westendorp, & van der Mast, 2004). Should further longitudinal research yield similar results (and also provide support for the mediating role of rumination), such findings would suggest that interventions directed at improving executive function in older adults may be effective in preventing or treating depressive symptoms in late life. For example, increased exercise is associated with significant gains in cognitive function among older adults (see Colcombe & Kramer, 2003), and exercise also appears to be effective in reducing depression in older adults (Mather et al., 2002; Singh, Clements, & Fiatarone Singh, 2001). The results of the present study suggest that the latter effect may, at least in part, reflect the former. Similarly, Mohlman (2005) has suggested that executive function rehabilitation training may improve response to cognitive-behavioral or problem-solving therapies in older adults. The current results suggest that in addition to training executive functioning, clinical interventions targeting ruminative thought patterns might be particularly useful in treating late-onset depression.

Several additional limitations of this study warrant consideration. First, because we screened only for major psychoactive medications, it is possible that some participants may have been taking drugs posing some risk of cognitive impairment. Although it seems unlikely that such participants would cluster predominantly in the late-onset group, the possibility remains that various drugs may account for some of the effect. Second, the study is limited by its small sample. It will be important for future research to replicate our findings in a larger sample, and extend them to samples with more severe depression. Third, the study is limited by our reliance on self-reports of age of depression onset. The reliability and validity of these self-reports is unclear. Other studies of depression in older adults typically have assessed age of onset in the context of a structured diagnostic clinical interview, occasionally corroborating age of onset reports with family members (e.g., see Ballmaier et al., 2004). Although such an approach seems likely to provide high quality information, it should be noted that there are little data available regarding its reliability and validity. Nevertheless, the approach taken in this study is open to question, and it remains possible that ED might interfere with the ability to accurately recall age of onset. However, one indication of the validity of our approach is that it produced evidence of the same relationship between age of onset of depression and executive dysfunction as has been observed in other studies using more elaborate methods. That is, significantly higher levels of executive dysfunction were seen in participants reporting late-onset depression (e.g., see Ballmaier et al., 2004).

In summary, the findings of the current research provide further evidence that late-onset depression differs from early-onset depression by virtue of the greater prominence of executive dysfunction in the former than the latter. The current findings also go beyond previous research by highlighting the important role of rumination in the relationship between executive dysfunction and late-onset depressive symptoms. Specifically, the current findings suggest that one important consequence of deficits in

executive functioning is that some older adults may no longer be able to inhibit their ruminative thought patterns. It is this increased rumination that seems to play a critical role in the development of late-onset depression. Thus, the current results point to a potentially productive avenue of future research that will assume even greater importance as an increasing percentage of the population moves into late adulthood.

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