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ORIGINAL INVESTIGATION

Higher levels of neuroticism in older adults predict lower executive functioning across time: the mediating role of perceived stress

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Abstract

Neuroticism has been associated with individual differences across multiple cognitive functions. Yet, the literature on its specific association with executive functions (EF) in older adults is scarce, especially using longitudinal designs. To disentangle the specific influence of neuroticism on EF and on coarse cognitive functioning in old adulthood, respectively, we examined the relationship between neuroticism, the Trail Making Test (TMT) and the Mini-Mental State Examination (MMSE) in a 6-year longitudinal study using Bayesian analyses. Data of 768 older adults (M_{age} = 73.51 years at Wave 1) were included in a cross-lagged analysis. Results showed no cross-sectional link between neuroticism and TMT performance at Wave 1 and no longitudinal link between neuroticism at Wave 1 and MMSE at Wave 2. However, neuroticism at Wave 1 predicted TMT performance at Wave 2, indicating that the more neurotic participants were, the lower they performed on the TMT six years later. Additional analyses showed that this relation was fully mediated by participants' perceived stress. Our results suggest that the more neurotic older adults are the more stress they may perceive six years later, which in turn negatively relates to their EF. In sum, this study demonstrates that neuroticism may lead to lower EF in older age across six years. It further suggests older adults' perceived stress as mediator, thereby providing novel insights into the mechanisms underlying this relation. Possible intervention approaches to counter these effects are discussed.

Keywords Neuroticism · Executive functions · Aging · Perceived stress · Cross-lagged modeling · Longitudinal study

As the population's longevity is continuously increasing, age-related cognitive decline has become one of the main issues in the study of aging (Kramer, 2012). Indeed, it is well established that many cognitive abilities decline with increasing age, particularly those related to executive functions (EF; Clarys et al., 2009; Fjell et al., 2017; Isingrini et al., 2008). Thus, uncovering which factors contribute to the maintenance or loss of EF across the lifespan is crucial. Not only does this allow to understand which factors can lead to a decrease in EF, but also to set up possible interventions to support individuals in preserving autonomy in their daily life as long as possible.

EF are defined as top-down cognitive processes that contribute to planning, reasoning, managing goals, dealing with unexpected challenges and self-regulation (Diamond, 2013). As they contribute to older adults' daily functioning and health-related behaviors (e.g., management of medication and chronic illness), EF are considered of major importance in successful aging and are crucial to maintain the autonomy of older adults (Engel-Yeger and Rosenblum, 2021; Suchy et al., 2020; Vaughan and Giovanello, 2010). In addition, EF

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are also indicators of subsequent neurocognitive diseases such as mild cognitive impairment and Alzheimer's disease (Allain et al., 2013; Espinosa et al., 2009; Storandt, 2008). Previous studies have gained important insights regarding factors that affect whether and to which degree EF decrease with age. For example, they have highlighted that lifestyle and socioeconomic factors (such as higher levels of education, higher socioeconomic status, or longer engagement in professional and non-professional occupations) can help preserve older adults' cognitive abilities in general and EF in particular (Delgado-Losada et al., 2019; Ihle et al., 2018; Moorman et al., 2018; Roldán-Tapia et al., 2012). Of key interest for the present study, personality traits have also been associated with age-related changes in EF (Booth et al., 2006; Chapman et al., 2017; DeYoung et al., 2010; Hall et al., 2014; Waggel et al., 2015).

Personality traits can be defined as an individual's disposition to behave in a similar, consistent way across different situations (Costa and McCrae, 1980). According to the Big Five Model, the following personality traits can be distinguished: openness to experience, conscientiousness, extraversion, agreeableness, and neuroticism (Costa and McCrae, 1992). The relationship to EF has been repeatedly examined for openness to experience, conscientiousness, extraversion, agreeableness (Bell et al., 2020; Buchanan, 2016; Campbell et al., 2011; Chapman et al., 2017; DeYoung et al., 2010; Hall et al., 2014; Ihle et al., 2019c; Jensen-Campbell et al., 2002; Murdock et al., 2013; Roye et al., 2020; Schretlen et al., 2010; Unsworth et al., 2009; Williams et al., 2010). In contrast, so far, very few studies investigated the association between neuroticism and EF, and particularly how EF develop as a function of neuroticism in old age. According to the Big Five Model, neuroticism is characterized by irrationality of thoughts, low ability to manage impulsions and stress, emotional instability, and consequently, a higher risk of developing psychiatric disorders (Costa and McCrae, 1992). However, while the negative relationship between neuroticism and EF seems well established in younger adults (Crow, 2019; Murdock et al., 2013; Purić and Pavlović, 2012; Schretlen et al., 2010), research on older adults is still inconsistent. Indeed, certain studies did not find a significant link between neuroticism and EF (Boyle et al., 2010; Hill et al., 2015; Turunen et al., 2020; Waggel et al., 2015), whereas others found that neuroticism was negatively associated with EF in older adults (Bell et al., 2020; Booth et al., 2006; Chapman et al., 2017; Denburg et al., 2009; Kim et al., 2021; Williams et al., 2010). Except for Kim et al.'s (2021) study, which demonstrates that sleep quality moderates the relationship between neuroticism and EF, little is known about mechanisms through which neuroticism may be associated with EF.

Thus, the potential mechanisms through which neuroticism may affect EF largely have yet to be examined. One possible mechanism involved in the relationship between neuroticism and EF might be stress. Indeed, higher levels of neuroticism have been associated with experiencing more negative affect, such as stress (Costa and McCrae, 1992; Curtis et al., 2015). Stress, in turn, has been associated with more intrusive thoughts, reduced levels of interference control, decreased attention, and thereby can impair performance on cognitive tasks and lead to everyday cognitive failures (Boals and Banks, 2012; Braunstein-Bercovitz, 2003; Caswell et al., 2003; Curtis et al., 2015; Lupien et al., 2009). Previous research on older adults further indicated that higher levels of stress are negatively associated with EF and positively to accelerated cognitive decline (Aggarwal et al., 2014; Diamond, 2013; Ihle et al., 2020; Liston et al., 2009; Oaten and Cheng, 2005; Orem et al., 2009; Wolff et al., 2021). So far, there has been no longitudinal study exploring whether the relationship between neuroticism and EF may be mediated by stress among older adults. This is unfortunate because if perceived stress indeed mediates the link between neuroticism and EF, targeting stress perception with interventions like mindfulness could reduce both the stress and the risk of adults' cognitive decline. Therefore, we aimed to explore this mechanism in the present study in a longitudinal design.

Indeed, research on neuroticism and EF in older adults also lacks longitudinal studies as most previous studies were cross-sectional (but see Waggel et al., 2015). One of the shortcomings of cross-sectional studies is that they do not provide information on how neuroticism and EF develop over time (Sedgwick, 2014). Moreover, discrepancies have been observed between the results of cross-sectional versus longitudinal studies that targeted other cognitive processes. Specifically, previous studies suggest cross-sectional studies usually depict a more negative image of older adults' abilities than results of longitudinal studies showed (Damon, 1965; Hedden and Gabrieli, 2004; Schaie, 1996; Schaie and Strother, 1968). Therefore, it is relevant to explore neuroticism and EF in a longitudinal design to explore whether cross-sectional effects are maintained over time or whether a different pattern appears longitudinally. It is noteworthy that, when assessing longitudinal mediation effects, it has been indicated that three waves of data collection should be favored over two. Specifically, compared to two waves, including three or more waves is more likely to avoid bias on the estimates of the mediation effects by accounting more precisely for previous values of the dependent variable (Cole and Maxwell, 2003; O'Laughlin et al., 2018). However, although examining data from even more waves does provide certain advantages, two waves longitudinal mediation analyses have been frequently used to examine potential links between different psychological constructs (Bilevicius et al., 2018; Butt et al., 2020; Frazier et al., 2017; Ihle

et al., 2019a; Ju and Lee, 2018; Kearns et al., 2016; Kwon et al., 2018; Naeem et al., 2020; Royuela-Colomer and Calvete, 2016; Weber and Exner, 2013). Further, previous studies show that two waves can be sufficient to examine such links using structural equation modeling (Bilevicius et al., 2018; Frazier et al., 2017; Ihle, et al., 2019a; Ju and Lee, 2018; Kwon et al., 2018; Royuela-Colomer and Calvete, 2016). Indeed, previous research shows that two waves of data still provide results that go beyond cross-sectional analyses. For example, Jose (2016) applied both cross-sectional and longitudinal analyses to the same dataset and found that of six significant cross-sectional mediations only four remained significant when examined longitudinally. Thus, even two-wave longitudinal studies may provide important information on abilities' development over time and demonstrate that cross-sectional conclusions should be taken cautiously when considering long-term implications such as interventions.

To the best of our knowledge, only one longitudinal study focusing on older adults has investigated the relationship between neuroticism and EF in older adults. Waggel and colleagues (2015) investigated performance on the TMT (as key indicator of EF) in a longitudinal study design of two years among 493 older adults and demonstrated that higher neuroticism scores were cross-sectionally associated with poorer performance on the TMT but did not predict future decline among older adults (Waggel et al. 2015). The authors explained that longer follow-up periods may be more appropriate to examine potential associations between neuroticism and cognitive measures because neuroticism could start influencing cognition earlier in life and potential effects may thus be more difficult to detect within old age. Considering this empirical and theoretical framework, we longitudinally investigated the relationship between neuroticism and EF with perceived stress as a potential mediator. Specifically, the first goal of the present study was to examine whether neuroticism is associated with EF (as indexed by performance on the TMT) beyond its possible relationship with coarse cognitive functioning (as indexed by performance on the Mini-Mental State Examination; MMSE, Folstein et al., 1975) among older adults in a longitudinal study across 6 years. As previous studies are lacking longitudinal results to assess the joint development of neuroticism and EF in late adulthood, we hypothesized a link between neuroticism and TMT (and between TMT and neuroticism) across six years without specifying the direction (positive vs. negative) of this link. The second goal of the present study was to explore whether perceived stress mediates the link between neuroticism and TMT across our two waves of data. Related to this, we further aimed to test a potential reverse causation by examining possible evidence for EF mediating the relationship between neuroticism and perceived stress.

Method

Data stem from the first and second waves of the longitudinal study "Vivre – Leben – Vivere" (VLV), which investigated cognitive aging across the lifespan in Switzerland. Participants were assessed in 2011 and 2017 in a face-to-face computer-assisted personal interview (for more details on the procedure, see Ihle et al., 2018; Künzi et al., 2021; Mella et al., 2018; Vallet et al., 2020).

The initial sample consisted of 3080 participants at the first wave and 1059 at the second wave (six years later). For the subsequent analyses, we only included participants who completed both waves and who had at least one answer on TMT or Neuroticism at Wave 1 or 2, who completed at least half of the questions of the MMSE at Wave 2 and who provided data for age, gender, and education. Thus, our final sample for the present analyses consisted of 768 participants (49% females) with a mean age of 73.51 years ($SD = 6.09$; range = 64–91 years) at Wave 1, 79.51 years (SD = 6.10; range = 70.21–97.94 years) at Wave 2 and a mean level of education of 3.92 (SD = 1.54; range = 0–6) at Wave 1, which indicates that, on average, participants' educational level was between "professional apprenticeship" and "high school graduation." All participants gave their written informed consent for inclusion before they participated in the study. The study was conducted in accordance with the Declaration of Helsinki (World Medical Association 2000), and the ethics commission of the Faculty of Psychology and Social Sciences of the University of Geneva had approved the protocol (project identification codes: CE_FPSE_14.10.2010 and CE_FPSE_05.04.2017).

Measures

Mini-mental state examination (MMSE)

Coarse cognitive functioning was assessed at Wave 2 with the MMSE (Folstein et al., 1975). The MMSE aims to assess cognitive functioning in the general population and represents one of the most used tests in research. It comprises subtests of spatial and temporal orientation, immediate and delayed recall, calculation and language. We calculated a total score of MMSE for each participant. The maximum score possible is 30 points. Higher scores reflect better coarse cognitive functioning.

Trail making test (TMT)

EF were assessed at both waves with the TMT. It has been previously used in several studies to assess cognitive flexibility using a visual scanning task. It is subdivided into two subtests: the TMT-A and the TMT-B (Gallant, 2016; Godefroy, 2001; Ihle et al., 2019b; Moll et al., 2002; Oosterman et al., 2010; Perrochon and Kemoun, 2014; Senior et al., 2018; Zhang et al., 2019). Both subtests consist of twenty-five circles that participants have to connect to one another. In TMT-A, each circle contains a number (from 1 to 25) and participants have to connect the circles in ascending order as fast as possible. In TMT-B, there are numbers (from 1 to 13) and letters (from A to L) that participants have to connect in ascending order while alternating between numbers and letters as fast as possible. In detail, they have to connect 1 to A, A to 2, 2 to B, etc. Thereby, TMT-A provides a baseline in a simple condition, whereas TMT-B requires participants to alternate between numbers and letters and thus deploys EF (Reitan, 1958). The TMT provided completion times in seconds for both TMT-A and TMT-B. It is important to know that based on multiple studies (Ashendorf et al., 2008; Goul and Brown, 1970; Tombaugh, 2004), we used cutoff criteria for the results to include in our study to avoid unlikely and biased results. More precisely, for TMT-A, we included participants who scored between 15 and 180 s (as it is generally very unlikely to be able to complete the test in less than 15 s, and 180 s is the stop time used in the VLV study). For TMT-B, we included participants who scored between 29 and 180 s (for the same reasons as previously mentioned, also see Ashendorf et al., 2008; Goul and Brown, 1970; Tombaugh, 2004). For information, a higher score on both parts of the TMT represents a worse performance: the higher the score, the slower the participants performed the task.

Neuroticism

Neuroticism was assessed at both waves with two questions of the BFI-10, an abbreviated self-assessment version of the Big Five Inventory (BFI-44; Rammstedt and John, 2007). The two questions were "I see myself as a person who…" (1) "… is relaxed, copes well with stress" and (2) "… is easily anxious," and participants had to answer on a 5-point Likert scale ranging from 1 "strongly disagree" to 5 "strongly agree." On both items, participants' score could range from 1 to 5. The score of the first item was inverted.

Perceived stress

Perceived stress was assessed at Wave 2 with the PSS-4 based on the Perceived Stress Scale (Cohen et al., 1983). Its aim is to measure the perception of stress participants had during the last month in their daily life (how the participants felt and experienced stress, how overwhelmed with or uncontrollable they consider their life during the last month). This scale includes four items related to general situations (e.g., "in the last month, how often have you felt that you were unable to control the important things in your life?") ranging from 0 "never" to 4 "very often." Two of the four items' scores had to be inverted. This questionnaire was introduced to the participants explaining that they will answer questions about their feelings and thoughts during the last month.

Statistical analyses

Following Lifshitz-Vahav et al. (2017), we applied three successive two-wave cross-lagged models, using the SPSS extension AMOS (Arbuckle, 2014). We used a latent variable of TMT performance constructed from scores in TMT parts A and B and a latent variable of neuroticism constructed from the two neuroticism items of the BFI-10. Specifically Model 1 included (a) autoregressive paths from neuroticism and TMT performance at Wave 1 to the same variables at Wave 2, (b) cross-lagged reciprocal paths between neuroticism and TMT performance over time, and paths from neuroticism and TMT performance at Wave 1 to MMSE at Wave 2. In Model 2, we added the latent variable of perceived stress to the previous model to test whether stress mediated the relationship between neuroticism at W1 and TMT performance at Wave 2. In Model 3, we tested the reverse causation hypothesis that is whether TMT mediated the relationship between neuroticism and perceived stress. In all models, we controlled for the effects of gender, education, and age on neuroticism, TMT, and MMSE of Wave 2. All predictors of interest and control variables were allowed to correlate. All three models presented a posterior predictive *p*-value close to 0.50, indicating a good model fit (see van de Schoot et al., 2014).

We tested the normality of our data which showed that the normality was not fulfilled, indicated by *p*-values of both Kolmogorov–Smirnov and Shapiro–Wilk tests being lower than 0.05. For the subsequent analyses, we applied Bayesian structural equation modeling (Bayesian SEM), as Bayesian analyses do not require normality to obtain robust results (Kelava et al., 2012).[1] Moreover, Bayesian analyses allow firm conclusions for maintaining the null hypothesis, meaning that we can conclude that there is no effect between

[1] Results presented in this paper are based on Bayesian estimation because normality assumptions for maximum likelihood estimation were not fulfilled. However, post hoc analyses show that classical inferential estimation would provide the same pattern of results, both for the cross-lagged and the mediation cross-lagged models.

Table 1 Descriptive statistics and Bayesian t tests between each variable for both waves with their credibility interval

Variables	*M*	*SD*	*T* tests Bayes factor$_{10}$	Estimated median effect size	95% Credibility interval
Wave 1 Neuroticism	5.32	1.90	0.358	−0.079	[−0.154;−0.004]
Wave 2 Neuroticism	5.24	1.79			
Wave 2 MMSE	28.47	1.82	–	–	–
Wave 1 TMT-A completion time	51.12	20.81	0.366	0.087	[0.002;0.172]
Wave 2 TMT-A completion time	53.12	22.18			
Wave 1 TMT-B completion time	102.7	29.7	43,247.593	0.239	[0.149;0.328]
Wave 2 TMT-B completion time	111.3	32.8			
Wave 2 Perceived stress	5.88	2.47	–	–	–

"M" = means and "SD" = standard deviation. Neuroticism mean scores are based on the sum of the two items of neuroticism for each wave divided by 2. A value of Bayes Factor $_{10}$ (BF_{10}) higher than 3 indicates that data are in favor of the alternative hypothesis rather than the null hypothesis. The credibility interval indicates significance of the BF_{10}: when the confidence interval does not contain the value 0 (meaning that both ends of the interval are either larger or smaller than 0), we reject H0 and conclude that the path is significant. Analyses indicate anecdotal evidence for H0 for neuroticism and TMT-A, meaning there was no significant difference between mean scores at W1 and W2. Analyses indicate extreme evidence for H1 for TMT-B, meaning there was a significant difference between mean scores at W1 and W2

variables. This is a great advantage compared to the frequentist approach because in our case, it would help to conclude whether there is or not a longitudinal relationship between neuroticism and the TMT. In sum, Bayesian analyses seem to be the best statistical tools because they will be less sensitive to the size of our sample and will help us determine whether there is a link between neuroticism, TMT, MMSE, and perceived stress. The analysis of our model resulted in a posterior predictive *p*-value of 0.50, which indicates a good model fit (see van de Schoot et al., 2014). We assessed significance of posterior means (further called estimates) for model paths using bounds (95% credible intervals; e.g., van de Schoot et al. 2014). When the confidence interval does not contain the value 0 (meaning that both ends of the interval are either larger or smaller than 0), we reject H0 and conclude that the path is significant.

Results

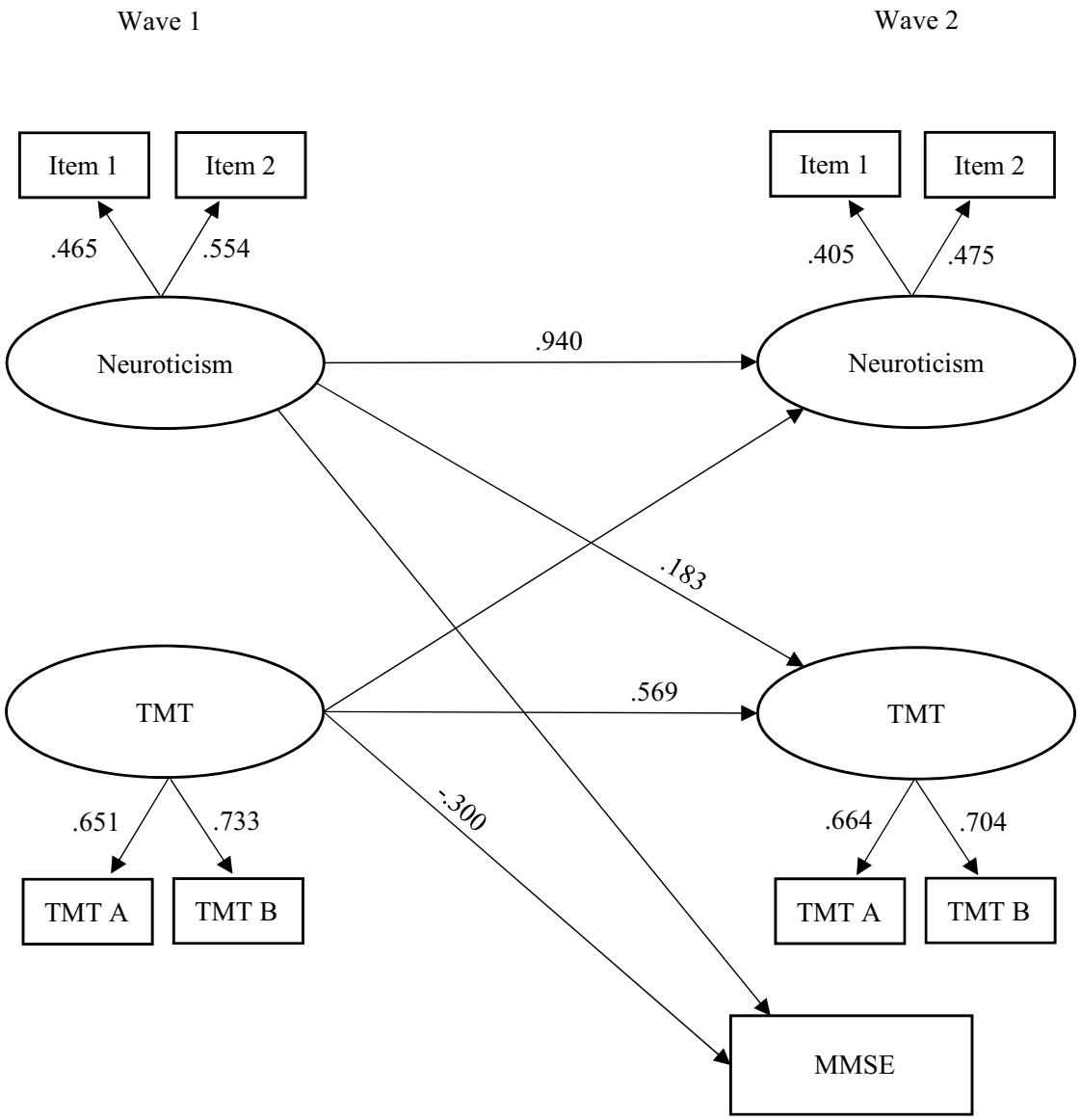
Descriptive statistics

Table 1 shows descriptive statistics of the variables of interest and the results of Bayesian *t* tests of variables to analyze change between both waves. These tests indicate anecdotal evidence supporting that there was no difference between TMT-A in W1 and TMT-A in W2 scores (BF_{10} = 0.366; 95% CI [0.002 ; 0.172]). Bayes factor indicates extremely strong evidence supporting a difference between TMT-B at Wave 1 and TMT-B at Wave 2, meaning that performance was lower at Wave 2 than at Wave 1 (BF_{10} = 43,247.593; 95% CI [0.149; 0.328]). Bayes factor indicates moderate evidence supporting no difference for participants' neuroticism across time (BF_{10} = 0.358; 95% CI [− 0.154; − 0.004]).

We also conducted a Bayesian repeated-measures ANOVA to explore whether there were differences between Wave 1 and Wave 2 on neuroticism scores at an individual level. Results indicate anecdotal evidence supporting H0 (BF_{10} = 0.304; 95% CI [− 0.004; 0.064]), thereby suggesting stability of neuroticism (rather than change) between Wave 1 and Wave 2. Further, we examined within-person changes in neuroticism (i.e., participants' scores of Wave 2 minus Wave 1). This showed that mean change between the two measurement waves was small ($M = -0.14$, $SD = 1.79$). Indeed, 74% of the sample changed by 2 points or less, and only 5% changed by more than 3 points. Taken together, although a small number of individuals displayed larger changes in neuroticism, overall this again suggested that neuroticism was rather stable across six years and that there was relatively little within-person variability.

Relationships between neuroticism and TMT across the two waves (Model 1)

Figure 1 depicts a synthesized version of Model 1, whereas Table 2 details estimates, standard errors, standard deviations, convergence, and the lower and upper bounds for credible intervals. Results showed that neuroticism at Wave 1 positively predicted neuroticism at Wave 2 (β = 0.940; 95% CI [0.740; 1.174] and that TMT at Wave 1 positively predicted TMT at Wave 2 (β = 0.569; 95% CI [0.431; 0.716]). Neuroticism and TMT at Wave 1 did not correlate (β = 0.087; 95% CI [− 0.076; 0.250]). Neuroticism at Wave 1 predicted TMT at



Note. This figure illustrates the results for Model 1. Reported values correspond to standardized posterior means for regression coefficients. For the purpose of readability, covariances between predictors, residual variances and covariances between residual variances are not depicted. Similarly, control variables (age, sex, education) and corresponding paths and estimates are not depicted. Further, only significant parameter estimates are reported. Parameter estimates that are not depicted here are reported in Table 2. "Neuroticism" = self-reported neuroticism on items of Big Five Inventory-10; "TMT" = Trail Making Test as index of executive functions; "MMSE" = Mini-Mental State Examination as index of coarse cognitive functioning.

Fig. 1 Two-Waves Cross-Lagged Model Between Neuroticism, Executive Functions, and Coarse Cognitive Functioning

Table 2 Estimates, standard errors, standard deviations, convergence statistic, lower and upper bounds of the paths of our cross-lagged model

	Estimate	*SE*	*SD*	Convergence statistic	95% lower bound	95% upper bound
Loadings						
Neuro1 → Neuro1Item1	0.465*	0.005	0.081	1.002	0.331	0.656
Neuro1 → Neuro1Item2	0.554*	0.004	0.098	1.001	0.360	0.745
Neuro2 → Neuro2Item1	0.405*	0.004	0.078	1.001	0.276	0.592
Neuro2 → Neuro2Item2	0.475*	0.004	0.090	1.001	0.306	0.658
TMT1 → TMT1A	0.651*	0.001	0.044	1.000	0.565	0.738
TMT1 → TMT1B	0.733*	0.001	0.054	1.000	0.625	0.837
TMT2 → TMT2A	0.664*	0.001	0.042	1.000	0.583	0.745
TMT2 → TMT2B	0.704*	0.001	0.043	1.000	0.620	0.789
Direct effects for variables of interest						
Neuro1 → Neuro2	0.940*	0.002	0.108	1.000	0.740	1.174
TMT1 → TMT2	0.569*	0.001	0.073	1.000	0.431	0.716
Neuro1 → TMT2	0.183*	0.002	0.072	1.000	0.044	0.324
TMT1 → Neuro2	−0.064	0.001	0.094	1.000	−0.249	0.117
Neuro1 → MMSE2	0.020	0.001	0.063	1.000	−0.099	0.146
TMT1 → MMSE2	−0.300*	0.001	0.059	1.000	−0.419	−0.186
Neuro1 ↔ TMT1	0.087	0.002	0.082	1.000	−.076	0.250
Control variables						
Age → Neuro2	0.052	0.001	0.069	1.000	−0.079	0.189
Age → TMT2	−0.237*	0.001	0.052	1.000	0.134	0.336
Age → MMSE2	−0.019	0.001	0.042	1.000	−0.099	0.064
Sex → Neuro2	−0.017	0.001	0.066	1.000	−0.150	0.112
Sex → TMT2	0.081	0.001	0.046	1.000	−0.171	0.009
Sex → MMSE2	0.063	0.001	0.038	1.000	−0.012	0.136
Educ → Neuro2	−0.043	0.002	0.065	1.000	−0.169	0.088
Educ → TMT2	−0.098*	0.001	0.048	1.000	−0.192	−0.002
Educ → MMSE2	0.144*	0.001	0.037	1.000	0.069	0.215
Age ↔ Neuro1	−0.041	0.001	0.057	1.000	−0.154	0.070
Age ↔ TMT1	0.313*	0.001	0.049	1.000	0.213	0.408
Age ↔ Sex	−0.047	0.001	0.037	1.000	−0.121	0.025
Age ↔ Educ	−0.037	0.001	0.036	1.000	−0.107	0.035
Sex ↔ Neuro1	0.204*	0.001	0.058	1.000	0.089	0.316
Sex ↔ TMT1	0.036	0.001	0.051	1.000	−0.063	0.137
Sex ↔ Educ	−0.175*	0.001	0.035	1.000	−0.244	−0.106
Educ ↔ Neuro1	−0.028	0.001	0.058	1.000	−0.144	0.082
Educ ↔ TMT1	−0.171*	0.001	0.051	1.000	−0.271	−0.068
Residual covariances						
Neuro1Item1 ↔ Neuro2Item1	0.184	0.005	0.091	1.002	−0.036	0.326
Neuro2Item1 ↔ Neuro2Item2	0.447*	0.006	0.136	1.001	0.153	0.686
TMT1A ↔ TMT2A	0.004*	0.001	0.002	1.000	0.001	0.008
TMT1B ↔ TMT2B	0.003	0.001	0.005	1.000	−0.007	0.012

"Neuro1" = neuroticism at Wave 1, "Neuro2" = neuroticism at Wave 2, "TMT1" = executive functions at Wave 1, "TMT2" = executive functions at Wave 2, "MMSE2" = MMSE at Wave 2, "Age" = control variable Age, "Sex" = control variable Sex, "Educ" = control variable Education. Single-headed arrows → indicate regression weights. Double-headed arrows ↔ indicate correlations between control variables, while they indicate covariances for residual covariances. The direct paths from Neuro1 to Neuro2, Neuro 1 to TMT2, TMT1 to TMT2 are significant, which is not the case for the path from TMT1 to Neuro2.* indicates significance based on the credibility intervals

Wave 2 ($\beta = 0.183$; 95% CI [0.044; 0.324]), but TMT at Wave 1 did not predict neuroticism at Wave 2 ($\beta = -0.064$; 95% CI [−0.249; 0.117]). Hence participants who had a higher level of neuroticism at Wave 1 had longer completion times on the TMT and thus worse EF performance at Wave 2. Regarding the predictors of coarse cognitive functioning, longer TMT completion times at Wave 1 predicted poorer MMSE performance ($\beta = -0.300$; 95% CI [−0.419; −0.186]), while the level of neuroticism at Wave 1 did not predict MMSE performance at Wave 2 ($\beta = 0.020$; 95% CI [−0.099; 0.146]).

Mediation via *perceived stress (Model 2)*

As a preliminary step to Model 2, we examined whether neuroticism at Wave 1 and perceived stress at Wave 2 were not redundant predictors of TMT performance at Wave 2. As indicated by tolerance value greater than 0.10 and a VIF value smaller than 10, there was no collinearity between neuroticism and perceived stress. We therefore proceeded with examining a potential mediation of the relationship between neuroticism and TMT through perceived stress. Figure 2 depicts a synthesized version of Model 2, whereas Table 3 details estimates, standard errors, standard deviations, convergence, and the lower and upper bounds for credible intervals. As expected, neuroticism at Wave 1 positively predicted perceived stress at Wave 2 ($\beta = 0.520$; 95% CI [0.367; 0.708]), meaning that the more neurotic participants were at Wave 1, the higher was their perceived stress during the last month before Wave 2. In turn, perceived stress negatively predicted TMT ($\beta = 0.204$; 95% CI [0.044; 0.394]), meaning that the more stressed participants were at Wave 2, the lower their cognitive performance at that same wave. Critically, while the indirect effect was significant ($\beta = 0.108$; 95% CI [0.022; 0.254]), the direct effect from neuroticism at Wave 1 to TMT at Wave 2 was no longer significant ($\beta = 0.029$; 95% CI [−0.223; 0.224]). Hence, perceived stress fully mediated the relationship between neuroticism at Wave 1 and the TMT performance at Wave 2 and explained 78.83% of the direct path from neuroticism at Wave 1 to TMT at Wave 2.

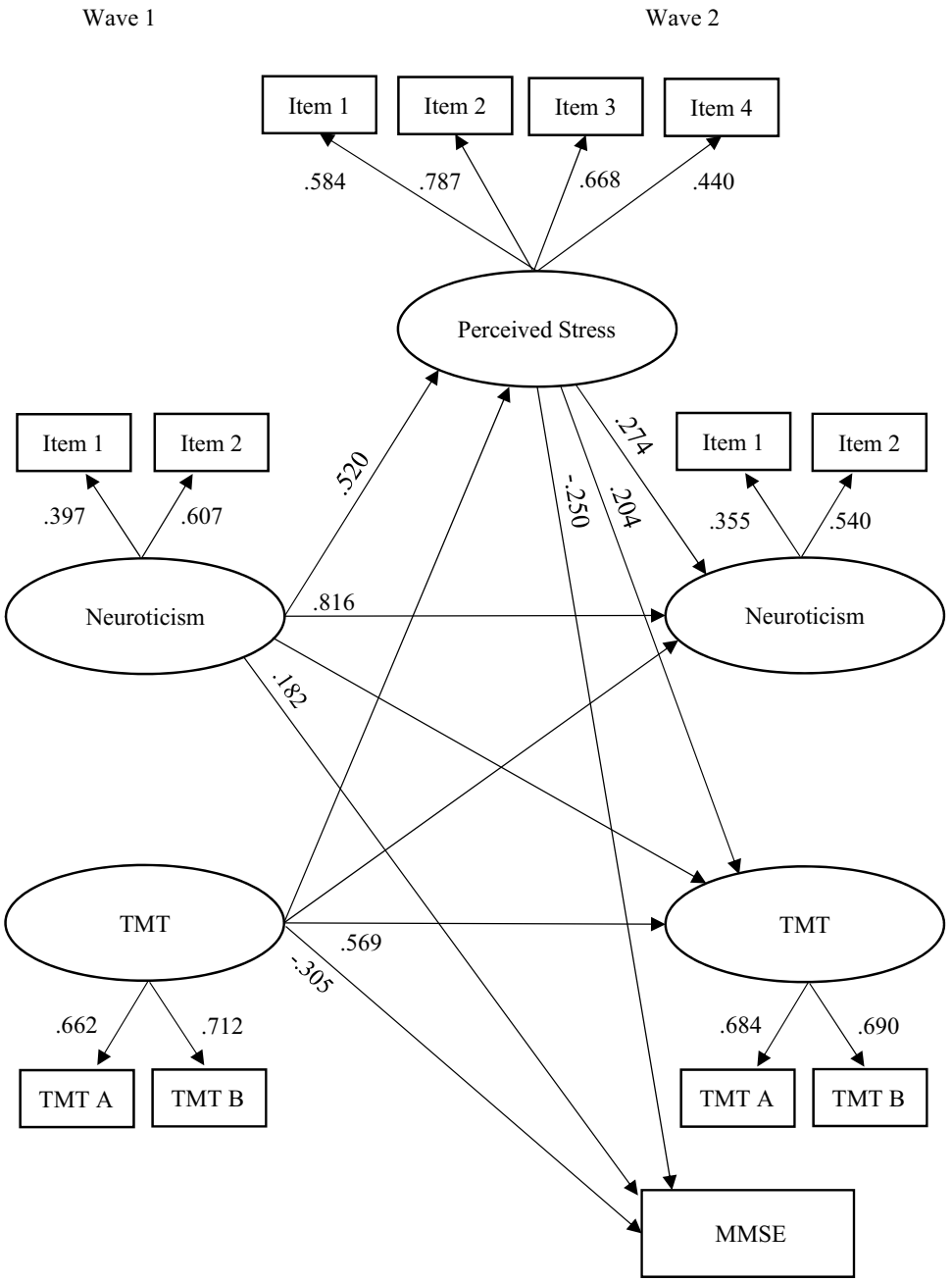
Reverse causation (Model 3)

To examine potential reverse causation, we tested an alternative model in which the relationship between neuroticism at Wave 1 and perceived stress at Wave 2 was mediated by TMT at Wave 2 (see Table 4). Neuroticism at Wave 1 positively predicted perceived stress at Wave 2 ($\beta = 0.467$; 95% CI [0.312; 0.648]) and TMT at Wave 2 positively predicted perceived stress ($\beta = 0.301$; 95% CI [0.064; 0.568]). However, neither the direct effect of neuroticism at Wave 1 on TMT at Wave 2 nor the indirect effect of neuroticism at Wave 1 on perceived stress at Wave 2 was significant ($\beta = 0.141$; 95% CI [− 0.009; 0.281] and $\beta = 0.039$; 95% CI [− 0.006; 0.094], respectively). Hence, these results exclude a reverse causation hypothesis.

Discussion

The present study examined the relationship of neuroticism and cognitive abilities (evaluated with the TMT and MMSE) in older adults across six years. The first aim of this study was to determine the existence of a link between neuroticism and EF in older adults. Although certain of the previous studies reported negative cross-sectional links between neuroticism and EF in younger adults (Crow, 2019; Murdock et al., 2013; Purić and Pavlović, 2012; Schretlen et al., 2010), other cross-sectional studies reported inconsistent results in older adults (Bell et al., 2020; Booth et al., 2006; Boyle et al., 2010; Chapman et al., 2017; Denburg et al., 2009; Hill et al., 2015; Kim et al., 2021; Turunen et al., 2020; Williams et al., 2010). Importantly, the only longitudinal study so far did not find a longitudinal link between neuroticism and EF among older adults when assessed across a relatively short period of time (i.e., two years, Waggel et al., 2015).

The present findings indicate that neuroticism at Wave 1 predicted TMT performance at Wave 2, meaning that the more neurotic participants were at the first wave, the lower their cognitive performance was six years later. This is in contrast to the study by Waggel et al. (2015) who investigated the link between neuroticism and TMT in a longitudinal design of two years. Interestingly, their results showed that neuroticism was negatively correlated cross-sectionally to TMT performance, but there was no significant 2-year longitudinal prediction from neuroticism to TMT performance. In contrast, in our study neuroticism and EF were unrelated at Wave 1, but higher levels of neuroticism predicted worse TMT performance six years later. A possible explanation for this difference of results between Waggel's study (2015) and ours might be the difference in time intervals (two versus six years) and in populations. In the latter regard, our participants were community-dwelling older adults randomly drawn from the general population aged 65 and above for Wave 1 (see Ludwig et al. 2014 for a description of the study), while in Waggel's study (2015) certain participants already had mild cognitive impairment (MCI) at Wave 1, others importantly declined in cognitive status and were then diagnosed with MCI or dementia before/at Wave 2 (note that this may also have compromised the validity of their assessment in using self-report personality questionnaires). Although our sample was composed of non-clinical participants, performance on TMT was significantly lower at Wave 2 than Wave 1, indicating indeed a decline in performance at Wave 2. Regarding longitudinal links, Waggel and colleagues (2015) indicated in their study that a longer time



Note. This figure illustrates the results of path analyses for Model 2. The direct cross-lagged path between neuroticism at Wave 1 and TMT at Wave 2 is not significant anymore after the addition of perceived stress as a mediator. Reported values correspond to standardized posterior means for regression coefficients. For the purpose of readability, covariances between predictors, residual variances and covariances between residual variances are not depicted. Similarly, control variables (age, sex, education) and corresponding paths and estimates are not depicted. Further, only significant parameter estimates are reported. Parameter estimates that are not depicted here are reported in Table 3. "Neuroticism" = self-reported neuroticism on items of Big Five Inventory-10; "TMT" = Trail Making Test as index of executive functions; "MMSE" = Mini-Mental State Examination as index of coarse cognitive functioning. "Perceived stress" = self-reported perceived stress on PSS-4 items of the Perceived Stress Scale.

Fig. 2 Analysis with Perceived Stress as a Mediator of the Relationship Between Neuroticism at Wave 1 and Executive Functions at Wave 2

Table 3 Estimates, standard errors, standard deviations, convergence statistic, lower and upper bounds of the paths of our cross-lagged model with perceived stress as a mediator

	Estimate	*SE*	*SD*	Convergence statistic	95% Lower bound	95% Upper bound
Loadings						
Neuro1 → Neuro1Item1	0.397*	0.002	0.043	1.001	0.312	0.483
Neuro1 → Neuro1Item2	0.607*	0.002	0.065	1.001	0.480	0.737
Neuro2 → Neuro2Item1	0.355*	0.001	0.044	1.000	0.268	0.442
Neuro2 → Neuro2Item2	0.540*	0.002	0.061	1.000	0.419	0.660
TMT1 → TMT1TMTA	0.662*	0.001	0.043	1.000	0.575	0.747
TMT1 → TMT1TMTB	0.712*	0.002	0.051	1.001	0.609	0.809
TMT2 → TMT2TMTA	0.684*	0.001	0.038	1.000	0.607	0.759
TMT2 → TMT2TMTB	0.690*	0.001	0 .039	1.000	0.612	0.769
Stress2 → Stress2Item1	0.584*	0.001	0.032	1.000	0.518	0.644
Stress2 → Stress2Item2	0.787*	0.001	0.027	1.000	0.734	0.838
Stress2 → Stress2Item3	0.668*	0.001	0.028	1.000	0.611	0.722
Stress2 → Stress2Item4	0.440*	0.001	0.036	1.000	0.369	0.510
Direct effects for variables of interest						
Neuro1 → Neuro2	0.816*	0.005	0.143	1.000	0.554	1.123
TMT1 → TMT2	0.569*	0.002	0.074	1.001	0.427	0.718
Neuro1 → TMT2	0.029	0.006	0.111	1.002	−0.223	0.224
TMT1 → Neuro2	−0.102	0.003	0.084	1.001	−0.272	0.060
Neuro1 → Stress2	0.520*	0.004	0.085	1.001	0.367	0.708
TMT1 → Stress2	0.011	0.003	0.077	1.001	−0.149	0.153
Neuro1 → MMSE2	0.182*	0 .006	0.108	1.002	0.009	0.448
TMT1 → MMSE2	−0.305*	0.002	0.063	1.001	−0.436	−0.186
Stress2 → TMT2	0.204*	0.005	0.088	1.001	0.044	0.394
Stress2 → Neuro2	0.274*	0.004	0.122	1.000	0.011	0.499
Stress2 → MMSE2	−0.250*	0.005	0.084	1.002	−0.441	−0.110
Neuro1 ↔ TMT1	0.141	0.003	0.085	1.000	−0 .021	0.312
Indirect effect for variables of interest						
Neuro1 → TMT2	0.108*	0.004	0.060	1.002	0.022	0.254
Control variables						
Age → Neuro2	−0.003	0.002	0.070	1.000	−0.136	0.140
Age → TMT2	0.182*	0.002	0.057	1.001	0.065	0.292
Age → MMSE2	0.040	0.009	0.049	1.001	−0.051	0.144
Age → Stress2	0.219*	0.002	0.051	1.001	0.119	0.324
Sex → Neuro2	−0.026	0.002	0.062	1.001	−0.154	0.092
Sex → TMT2	−0.065	0.002	0.047	1.001	−0.156	0.029
Sex → MMSE2	0.047	0.002	0.043	1.001	−0.043	0.125
Sex → Stress2	−0.030	0.002	0.052	1.000	−0.137	0.064
Educ → Neuro2	−0.047	0.002	0.058	1.001	−0.162	0.066
Educ → TMT2	−0.081	0.001	0.048	1.000	−0.174	0.016
Educ → MMSE2	0.123*	0.001	0.040	1.000	0.042	0.196
Educ → Stress2	−0.064	0.001	0.048	1.000	−0.158	0.027
Age ↔ Neuro1	−0.039	0.002	0.057	1.000	−0.152	0.074
Age ↔ TMT1	0.314*	0.001	0.049	1.000	0.216	0.411
Age ↔ Sex	−0.049	0.001	0.037	1.000	−0.122	0.024
Age ↔ Educ	−0.037	0.001	0.037	1.000	−0.108	0.035
Sex ↔ Neuro1	0.214*	0.002	0.056	1.000	0.104	0.325
Sex ↔ TMT1	0.034	0.001	0.049	1.000	−0.063	0.131
Sex ↔ Educ	−0.174*	0.001	0.036	1.000	−0.245	−0.105

Table 3 (continued)

	Estimate	*SE*	*SD*	Convergence statistic	95% Lower bound	95% Upper bound
Educ ↔ Neuro1	−0.021	0.001	0.057	1.000	−0.134	0.092
Educ ↔ TMT1	−0.175*	0.001	0.051	1.000	−0.276	-0.076
Residual covariances						
Neuro1Item1 ↔ Neuro2Item1	0.243*	0.001	0.051	1.000	0.144	0.345
Neuro2Item1 ↔ Neuro2Item2	0.359*	0.001	0.092	1.000	0.170	0.534
TMT1A ↔ TMT2A	0.004*	0.001	0.002	1.000	0.001	0.008
TMT1B ↔ TMT2B	0.004	0.001	0.005	1.000	−0.005	0.013
Stress2Item1 ↔ Stress2Item4	0.132*	0.001	0.028	1.000	0.078	0.188

"Neuro1" = neuroticism at Wave 1, "Neuro2" = neuroticism at Wave 2, "TMT1" = executive functions at Wave 1, "TMT2" = executive functions at Wave2, "Stress2" = perceived stress at Wave 2, "MMSE2" = MMSE of Wave 2, "Age" = control variable Age, "Sex" = control variable Sex, "Educ" = control variable Education. Single-headed arrows → indicate regression weights. Double-headed arrows ↔ indicate correlations between control variables, while they indicate covariances for residual covariances. The direct path from Neuro1 to TMT2 is not significant anymore, while the direct paths from Neuro1 to Stress2, Stress2 to TMT2 are significant. The indirect path from Neuro1 to TMT2 is significant

*Indicates significance based on the credibility intervals

interval seems to be necessary for neuroticism effects to longitudinally manifest its effect. In contrast to Waggel et al.'s (2015) suggestion, our study did not demonstrate significant differences in neuroticism scores at the group level across six years. Similarly, on an individual level, results showed that only few individuals importantly changed in neuroticism across six years and that, in general, there was relatively little within-person variability. However, although neuroticism was rather stable between Wave 1 and Wave 2, results demonstrate a longitudinal effect of neuroticism on EF. To our knowledge, this is the first study to demonstrate a significant longitudinal relationship from neuroticism to EF among older adults. As EF are related to older adults' daily functioning (Engel-Yeger and Rosenblum, 2021; Suchy et al., 2020; Vaughan and Giovanello, 2010), this finding is of relevance because it could enable to develop prevention programs to help older adults preserving their autonomy in daily life. Further, our initial findings do not indicate a link between neuroticism at Wave 1 and MMSE at Wave 2. This is in line with previous studies finding no relationship between neuroticism and coarse cognitive functioning (Arbuckle et al., 1998; Baker and Bichsel, 2006; Jelicic et al., 2003; Pearson, 1993), but is in contrast with other studies demonstrating that neuroticism is negatively associated with the performance on coarse cognitive functioning. Such differences may partially be related to differences in assessment, and future studies will therefore have to replicate our results using similar measures of coarse cognitive functioning. Further, although our participants were healthy community-dwelling older adults who lived autonomously, were willing to participate in the study, and who managed to participate in a rather long cognitive assessment, the mean age at Wave 2 was 79.53 years, with the oldest participants being up to 97 years old. Thus, although the oldest individuals may have still been rather autonomous and cognitively functioning in their daily life, other negative aspects of old age may have impacted their daily living and well-being, such as loneliness, reduction of positive affect, and of life satisfaction (Baltes and Smith, 2003; Gilleard and Higgs, 2010; Higgs and Gilleard, 2021; Yang et al., 2018). It would therefore be important for future studies to control for or examine in more detail these and other factors that are linked to older age and may impact cognition.

Our second aim was to explore the link between neuroticism and EF with perceived stress as a mediator in our model. Bayesian analyses revealed a full mediation. Results indicate that the more neurotic older adults were, the more they perceived stress six years later, and in parallel, the worse their cognition at that time. This full mediation between neuroticism and EF across time is congruent with previous studies showing that stress can have a negative influence on EF performance (Diamond, 2013; Ihle et al., 2020; Liston et al., 2009; Oaten and Cheng, 2005; Orem et al., 2009; Wolff et al., 2021). Our results support the idea that individuals with higher levels of neuroticism experience more stress, which finally has an influence on their EF performance. In our analyses, we also examined a potential reverse causation between neuroticism at Wave 1, perceived stress and TMT at Wave 2. Specifically, we tested an alternative model in which the relationship between neuroticism at Wave 1 and perceived stress at Wave 2 was mediated by EF. This model enabled us to rule out a potential reverse causation mechanism. Hence, our findings support our hypothesis that perceived stress mediates the effect of neuroticism on EF across time, and are in accordance with previous studies demonstrating an association between EF and stress

Table 4 Estimates, standard errors, standard deviations, convergence statistic, lower and upper bounds of the paths of the reverse causation cross-lagged model with executive functions as a mediator of the relationship between neuroticism and perceived stress

	Estimate	*SE*	*SD*	Convergence statistic	95% lower bound	95% upper bound
Loadings						
Neuro1 → Neuro1Item1	0.401*	0.001	0.044	1.000	0.315	0.488
Neuro1 → Neuro1Item2	0.613*	0.001	0.063	1.000	0.488	0.739
Neuro2 → Neuro2Item1	0.357*	0.001	0.044	1.000	0.270	0.443
Neuro2 → Neuro2Item2	0.542*	0.001	0.061	1.000	0.419	0.661
TMT1 → TMT1A	0.660*	0.001	0.044	1.000	0.572	0.744
TMT1 → TMT1B	0.714*	0.001	0.051	1.000	0.610	0.812
TMT2 → TMT2A	0.678*	0.001	0.040	1.000	0.599	0.756
TMT2 → TMT2B	0.688*	0.001	0 .040	1.000	0.607	0.766
Stress2 → Stress2Item1	0.586*	0.001	0.033	1.000	0.520	0.648
Stress2 → Stress2Item2	0.789*	0.001	0.027	1.000	0.735	0.840
Stress2 → Stress2Item3	0.669*	0.001	0.028	1.000	0.611	0.722
Stress2 → Stress2Item4	0.445*	0.001	0.036	1.000	0.371	0.514
Direct effects for variables of interest						
Neuro1 → Neuro2	0.821*	0.002	0.141	1.000	0.556	1.113
TMT1 → TMT2	0.574*	0.001	0.074	1.000	0.434	0.722
Neuro1 → TMT2	0.141	0.001	0.074	1.000	−0.009	0.281
TMT1 → Neuro2	−0.103	0.001	0.086	1.000	−0.280	0.062
Neuro1 → Stress2	0.467*	0.002	0.085	1.000	0.312	0.648
TMT1 → Stress2	−0.165	0.003	0.128	1.000	−0.439	0.061
Neuro1 → MMSE2	0.173*	0 .003	0.099	1.000	0.008	0.388
TMT1 → MMSE2	−0.302*	0.001	0.061	1.000	−0.427	−0.185
TMT2 → Stress2	0.301*	0.003	0.128	1.000	0.064	0.568
Stress2 → Neuro2	0.274*	0.002	0.116	1.000	0.040	0.497
Stress2 → MMSE2	−0.245*	0.002	0.077	1.000	−0.407	−0.112
Neuro1 ↔ TMT1	0.141	0.001	0.082	1.000	−0.019	0.304
Indirect effect for variables of interest						
Neuro1 → Stress2	0.039	0.001	0.025	1.000	−0.006	0.094
Control variables						
Age → Neuro2	−0.004	0.001	0.068	1.000	−0.133	0.135
Age → TMT2	0.230*	0.001	0.053	1.000	0.123	0.331
Age → MMSE2	0.037	0.001	0.048	1.000	−0.052	0.136
Age → Stress2	0.150*	0.001	0.054	1.000	0.043	0.257
Sex → Neuro2	−0.024	0.001	0.062	1.000	−0.149	0.097
Sex → TMT2	−0.075	0.001	0.046	1.000	−0.165	0.016
Sex → MMSE2	0.050	0.001	0.041	1.000	−0.033	0.128
Sex → Stress2	−0.003	0.001	0.051	1.000	−0.107	0.095
Educ → Neuro2	−0.043	0.001	0.058	1.000	−0.160	0.070
Educ → TMT2	−0.096*	0.001	0.047	1.000	−0.187	−0.002
Educ → MMSE2	0.124*	0.001	0.039	1.000	0.045	0.199
Educ → Stress2	−0.036	0.001	0.048	1.000	−0.129	0.058
Age ↔ Neuro1	−0.038	0.001	0.057	1.000	−0.150	0.073
Age ↔ TMT1	0.314*	0.001	0.049	1.000	0.217	0.411
Age ↔ Sex	−0.048	0.001	0.036	1.000	−0.119	0.022
Age ↔ Educ	−0.037	0.001	0.037	1.000	−0.109	0.035
Sex ↔ Neuro1	0.212*	0.001	0.056	1.000	0.103	0.322
Sex ↔ TMT1	−0.036	0.001	0.050	1.000	−0.063	0.135
Sex ↔ Educ	−0.175*	0.001	0.035	1.000	−0.243	−0.105

Table 4 (continued)

	Estimate	*SE*	*SD*	Convergence statistic	95% lower bound	95% upper bound
Educ ↔ Neuro1	−0.022	0.001	0.057	1.000	−0.133	0.089
Educ ↔ TMT1	0.175*	0.001	0.051	1.000	−0.274	−0.075
Residual covariances						
Neuro1Item1 ↔ Neuro2Item1	0.241*	0.001	0.052	1.000	0.141	0.344
Neuro2Item1 ↔ Neuro2Item2	0.355*	0.001	0.095	1.000	0.160	0.531
TMT1A ↔ TMT2A	0.004*	0.001	0.002	1.000	0.001	0.008
TMT1B ↔ TMT2B	0.004	0.001	0.005	1.000	−0.005	0.013
Stress2Item1 ↔ Stress2Item4	0.131*	0.001	0.028	1.000	0.076	0.187

"Neuro1" = neuroticism at Wave 1, "Neuro2" = neuroticism at Wave 2, "TMT1" = executive functions at Wave 1, "TMT2" = executive functions at Wave2, "Stress2" = perceived stress of Wave 2, "MMSE2" refers to MMSE at Wave 2, "Age" = control variable Age, "Sex" = control variable Sex, "Educ = control variable Education. Single-headed arrows → indicate regression weights. Double-headed arrows ↔ indicate correlations between control variables, while they indicate covariances for residual covariances. The direct paths from Neuro1 to Stress2, Neuro1 to TMT2, TMT2 to Stress2 are significant. The indirect path from Neuro1 to Stress2 is significant. * indicates significance based on the credibility intervals

(Diamond, 2013; Ihle et al., 2020; Liston et al., 2009; Oaten and Cheng, 2005; Orem et al., 2008; Wolff et al., 2021). In the present study, perceived stress explained 78.83% of the effect of neuroticism at Wave 1 on EF at Wave 2.

As our findings suggest that perceived stress plays a major role in the relationship between neuroticism and the loss of EF abilities, this could further be crucial for daily autonomy in older adults. For example, when considering real-life implications of the current findings and future directions, it seems essential to prevent older adults from being affected by stress to avoid the potential decline in EF. Previous research suggests that mindfulness-based interventions can reduce stress in younger and in older adults (Armstrong and Rimes, 2016; Berk et al., 2018; Hanley et al., 2019). For example, Armstrong and Rimes (2016) presented an intervention that indirectly targets neuroticism through training sessions that aim to make participants understand why some individuals are more sensitive to certain types of stressful events, to improve how they react to stress, and how they can manage stress. Two meta-analyses and two more recent studies demonstrated that mindfulness-based programs are able to reduce stress levels in healthy younger and older adults as well as in those suffering from chronic conditions like cardiovascular diseases and diabetes, but also anxiety, depression, and stress disorders (Chiesa and Serretti, 2009; Felsted, 2020; Grossman et al., 2004; Perez-Blasco et al., 2016). Moreover, previous research and reviews also demonstrate that mindfulness-based stress reduction programs can improve performance on different components of EF, including cognitive flexibility (Gallant, 2016; Hazlett-Stevens et al., 2019; Moynihan et al., 2013). These intervention programs are promising and may help older adults to improve or preserve their EF as they allow simultaneously targeting EF as direct outcome as well as stress as mediator between neuroticism and EF and thereby might help them maintain their daily autonomy.

Although our study presents a large sample with both cross-sectional and longitudinal data, it also has certain limitations. First, neuroticism was assessed with the BFI-10, which includes only two items to estimate participants' neuroticism levels. Even though this test is standardized and should reflect actual levels of participants' neuroticism (Courtois et al., 2020; Rammstedt et al., 2013), future studies will have to further examine and replicate the role of neuroticism using a more comprehensive test battery. Second, in the present study, EF were assessed with the TMT, which is a reliable tool used in neuropsychological screening to assess cognitive flexibility. However, according to Miyake et al.'s (2000) conception, EF are composed of multiple separate yet related core facets: updating (i.e., enabling individuals to update and monitor the content in working memory), inhibition (i.e., enabling individuals to inhibit predominant responses), shifting (also referred to as cognitive flexibility; i.e., enabling individuals to shift between mental sets, tasks, or instructions). Although the different facets are interrelated and EF tasks typically deploy more than one facet at a time (Burgess, 1997; Hughes and Graham, 2002; Miyake et al., 2000; Miyake and Friedman, 2012; Phillips, 1997), the TMT mainly focuses on assessing cognitive flexibility (Gallant, 2016; Godefroy, 2001; Ihle et al., 2019b; Moll et al., 2002; Oosterman et al., 2010; Perrsochon and Kemoun, 2014; Senior et al., 2018; Zhang et al., 2019). In the future, it would be important to examine additional measures that target other components of EF (i.e., working memory updating or inhibitory control) and explore their relationship to neuroticism in a longitudinal design to understand whether our pattern of results is specific to cognitive flexibility or present for other EF components. Third, when considering the mediation via perceived stress, further research across at least three

waves would be necessary to replicate and investigate in more detail how perceived stress influences the relationship between neuroticism and EF across longer time intervals. This would also enable to confirm the absence of a potential reverse causation.

Taken together, the present study adds to the literature on personality, EF, and stress in older age. To our knowledge, this is the first longitudinal study to investigate neuroticism, TMT, MMSE, and perceived stress in a cross-lagged model across six years using Bayesian analyses. In detail, it introduces neuroticism as a longitudinal predictor of EF six years later and perceived stress as a relevant mediator of this relationship, thereby providing novel insights into the possible mechanisms behind personality and EF interaction. Further, our study supports the importance of developing prevention and intervention trainings to counteract cognitive decline due to neuroticism and stress using promising mindfulness-based programs.

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