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Conscientiousness is associated with less amyloid deposition in cognitively normal aging

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Abstract

Little is known about the association between personality and Alzheimer's disease (AD) biomarkers, and existing results are inconsistent. We aimed to determine whether personality was associated with β -amyloid ($A\beta$) accumulation in cognitively normal aging. One hundred twenty-nine participants were included in this cross-sectional study. Personality was measured with the Big Five Inventory (BFI) and brain $A\beta$ deposition was assessed with [¹¹C] Pittsburgh compound B (PiB)-positron emission tomography (PET) imaging. Conscientiousness scores had a negative association with global PiB distribution volume ratio (DVR) in all participants after adjusting for age, sex, education, and vascular risk factors (β [SE] = -0.19 [0.09], 95% CI = $[-0.35, -0.02]$, $p = 0.031$), while agreeableness, extraversion, neuroticism, and openness had no association with global PiB DVR. Assuming the relative stability of personality traits, these findings suggest that conscientiousness may protect against $A\beta$ accumulation in cognitively normal aging through mechanisms that are as yet unknown.

Keywords

personality; conscientiousness; amyloid; positron emission tomography

It is well known that β -amyloid ($A\beta$) accumulation in Alzheimer's disease (AD) progresses decades before symptoms occur. For this reason, there is a growing interest in factors that can affect AD risk before it begins. Personality has been linked to the risk for AD, but the mechanisms that might underlie this association are unknown. The five-factor

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Conflict of interests

Dr. Jagust serves as a consultant to Genentech, CuraSen, Grifols, and Bioclinica. Dr. Baker serves as a consultant to Genentech. Dr. Landau has served as a paid consultant for Cortexyme and Neurovision.

With regard to prior dissemination of these results, the majority of this work was presented as a poster at the Human Amyloid Imaging conference in Miami FL in January 2020. No results have appeared in any other format, printed or verbal.

model (FFM) of personality is a well-known personality construct that defines five traits: neuroticism, extraversion, openness, agreeableness, and conscientiousness (McCrae & John, 1992). Several studies have investigated the association not only between personality and cognition but also between personality and dementia. They have consistently reported that higher conscientiousness was associated with better cognitive status and less decline, while higher neuroticism was associated with worse performance on multiple cognitive measures and a greater decline in memory (Caselli et al., 2016; Luchetti, Terracciano, Stephan, & Sutin, 2016). With respect to dementia, Chapman et al. (2019) indicated that adolescent personality traits were associated with later-life dementia and therefore might be a true independent risk factor for dementia by age 70 years. Additionally, several previous studies including a meta-analysis have provided evidence that low conscientiousness and high neuroticism have the most consistent associations with increased risk of incident dementia (Low, Harrison, & Lackersteen, 2013; Terracciano, Stephan, Luchetti, Albanese, & Sutin, 2017). Meta-analysis studies also indicate that individuals who scored higher on neuroticism or lower on conscientiousness had a greater risk for incident AD (Terracciano & Sutin, 2019; Terracciano et al., 2014) and several observational studies suggest that personality is a risk or protective factor for AD through unknown pathways (Duberstein et al., 2011; Johansson et al., 2014).

Personality may affect AD risk through lifestyle, health behaviors, and adaptation to stress throughout life. These factors, in turn, might influence a key pathology of AD, the A β plaque. A β pathology can be detected in vivo in the brain, using positron emission tomography (PET) with radiopharmaceutical ligands that bind to and image the quantity and distribution of A β plaques. Higher PET measures of A β plaque burden correlate with lower levels of the A β protein in cerebrospinal fluid reflecting sequestration of the protein in plaques in the brain. A recent investigation found that individuals with CSF biomarkers indicative of AD showed a significant increase in neuroticism and a decrease in conscientiousness over time as compared to a non-AD CSF biomarker group (Tautvydaite, Antonietti, Henry, von Gunten, & Popp, 2017). One study using PET imaging reported a positive association between neuroticism scores and global measures of PET A β deposition, but no relationship with extraversion, openness, agreeableness, or conscientiousness (Schultz et al., 2019). These conflicting results might reflect relatively small sample sizes or heterogeneous participants.

Several mechanisms could link personality to A β deposition. First, personality may affect health behaviors, lowering the risk of cardiovascular disease, which has been linked to A β accumulation (Allen, Magee, Vella, & Laborde, 2017; Stephan, Sutin, Luchetti, Bosselut, & Terracciano, 2018; Sutin et al., 2016; Sutin & Terracciano, 2016, 2017). Second, because chronic stress is associated with increased AD risk (Crowe, Andel, Pedersen, & Gatz, 2007; Johansson et al., 2010; Wilson et al., 2003), stress responses may be managed differently by those with different personality characteristics, which might affect A β deposition (Besser & Shackelford, 2007; Ebstrup, Eplöv, Pisinger, & Jørgensen, 2011; Kim et al., 2016). Finally, “cognitive style” may influence how neural resources are used for cognitive computations, and could affect activity in brain networks, thereby resulting in differential amyloid release with neural activity (Jagust & Mormino, 2011). Based on both empirical data linking AD risk to personality, along with an interest in trying to explain underlying mechanisms, we

sought to investigate how A β might be linked to personality in a cohort of healthy normal older individuals using the amyloid PET imaging radiopharmaceutical [¹¹C] Pittsburgh compound B (PiB) to detect brain A β . Furthermore, to explore whether personality may affect A β by influencing health behaviors, we examined relationships between personality and health factors related to risks for cardiovascular disease.

Methods

Participants

Participants were 129 normal older individuals who underwent [¹¹C] PiB PET and personality assessments within 1 year. All were enrolled in the Berkeley Aging Cohort Study (BACS), an ongoing observational cohort study of normal cognitive aging, and underwent a basic demographic and medical interview, lifestyle questionnaires, and a detailed neuropsychological test battery that has been previously described (Harrison, Maass, Baker, & Jagust, 2018). Vascular risk factors included hypertension, diabetes, hyperlipidemia (elevated serum cholesterol or triglycerides), transient ischemic attack (TIA) or stroke and were established through the medical questionnaire (present/absent by medical history, regardless of treatment status). In addition, body mass index (BMI) was measured, and physical activity was assessed using a validated questionnaire that consists of an index of physical activity (Siscovick et al., 1997) recording exercise (as total Kcal, miles walked and hours seated) over the prior 2 weeks. Subjects were genotyped for the apolipoprotein E (APOE) allele, and underwent magnetic resonance imaging (MRI) and [¹¹C] PiB PET. Inclusion criteria were: 1) baseline Mini-Mental State Examination score \geq 25; 2) no neurological, psychiatric, or major medical illness; 3) no medications affecting cognition; 4) normal performance on neuropsychological testing (1.5 SD within age and education adjusted means). The study was approved by the Institutional Review Boards of the University of California, Berkeley and Lawrence Berkeley National Laboratory (LBNL) and written informed consent was obtained from each participant.

In the present study, composite scores were calculated to measure 3 cognitive domains: episodic memory, working memory, and processing speed. Episodic memory tests included the California Verbal Learning Test (CVLT) recall total, Visual Reproduction (VR) I recall total, VR II recall total, VR recognition total, Logical Memory story A plus B1, and Visual Paired Associates total score. Working memory tests were Digit Span total score and Listening Span total recall. Processing speed tests were Trail Making Test B minus A, Stroop color correct in 1 minute, and Digit Symbol total. The z-scores for the individual tests were calculated using the mean and standard deviation, and the composite z-score of each cognitive domain was calculated by the sum of the z-scores of the component cognitive tests divided by square root of the number of the included cognitive tests.

Personality assessment

To investigate FFM personality traits, we used a self-reported questionnaire. The Big Five Inventory (BFI), yielding dimension scores for agreeableness, conscientiousness, extraversion, neuroticism, and openness, is composed of 44-items and consists of short phrases with relatively accessible vocabulary (Benet-Martinez & John, 1998; Digman, 1990;

John, Donahue, & Kentle, 1991; John, Naumann, & Soto, 2008). Each trait is measured with 8–10 questions: 9 items measured agreeableness and conscientiousness individually, 8 items measured extraversion and neuroticism individually, and 10 items measured openness. Questions were scored by a five-point Likert scale ranging from “strongly disagree” (1) to “strongly agree” (5). The scores of each personality dimension were summed to provide an overall measure of each of the big 5 traits. The summed scores of each personality trait ranged from 8–40 for extraversion and neuroticism, 9–45 for agreeableness and conscientiousness, and 10–50 for openness depending on the number of items.

Amyloid deposition in the brain

PET scanning with [¹¹C] PiB is a technique employing a radiotracer to visualize *in vivo* brain A β plaques in the brain, a hallmark pathology and biomarker for AD. PiB-PET measurements are continuous quantitative values measured as distribution volume ratios (DVR), but are also frequently dichotomized as PiB(+) or PiB(–) to describe participant samples for clinical purposes. All participants underwent [¹¹C] PiB PET imaging at LBNL on a Siemens Biograph 6 Truepoint PET/CT scanner and MRI scanning on a Siemens 1.5T Avanto system. A detailed description of these methods has been published previously (Mormino et al., 2011; Villeneuve et al., 2015). All PiB PET data were preprocessed using Statistical Parametric Mapping (SPM) version 12 software (<http://www.fil.ion.ucl.ac.uk/spm/>), summed, and realigned. PiB data were collected as 90 min dynamic data and DVRs were generated with Logan graphical analysis on PiB frames corresponding to 35 to 90 minutes post-injection using a cerebellar grey matter reference region (Logan et al., 1996; Price et al., 2005). Global cortical PiB DVR values were calculated using MRI-defined FreeSurfer-derived gray matter regions of interest (ROIs) as previously described (Mormino et al., 2011). To obtain the global PiB index, mean DVR values from FreeSurfer 5.3-defined frontal, parietal, temporal, and anterior/posterior cingulate cortices were computed. A global cortical PiB DVR threshold of 1.065 was used to dichotomize participants as PiB negative (PiB(–)) or positive (PiB(+)) (Villeneuve et al., 2015).

Statistical analysis

Statistical analyses were performed using SPSS version 20.0 (IBM, Armonk, NY, USA) with a value for statistical significance of $p < 0.05$. We examined the intraclass correlation coefficient (ICC) and Cronbach’s alpha to evaluate the reliability of the personality measures in a subgroup of participants who had two or more BFI assessments. To determine the association between personality and amyloid, we performed bivariate correlation analyses between each personality dimension and PiB DVR. We then fit a series of linear regression models for PiB DVR with each of the five personality scores as the independent variable, which were adjusted for relevant covariates such as age, sex, education, and the vascular risk factors of BMI and hypertension. The first model was adjusted by age, and sex, the second model was adjusted by age, sex, and education, and the third model was adjusted by age, sex, education, BMI, and hypertension in all participants. Lastly, to explore whether personality affects amyloid accumulation by influencing lifestyle or behavioral choices, we examined the relationships between vascular risk factors, BMI, physical activity, and

personality using either Spearman's ρ or Kendall's τ because the data were not normally distributed.

Results

Sixty-one of the 125 subjects had BFI assessed twice, 60 had 3 measurements, and 4 had 4 measurements using the BFI questionnaire. The average interval between tests was 14.2(\pm 4.7) months. The intraclass correlation coefficient (ICC) for all BFI dimensions showed good reliability, with a value of 0.824 for Conscientiousness (95% confidence interval [CI] 0.759–0.873) and the lowest value 0.747 for Agreeableness (95% CI 0.658–0.815). Cronbach's alpha also indicated good test-retest reliability (0.904 for Conscientiousness). These results suggest that the personality measurements are stable at the ages we investigated. Table 1 shows the characteristics of the participants and correlations among variables. Seventy participants (54.3%) were female, the average age was 72.1 years, and 36 participants (27.9%) were APOE ϵ 4 carriers. Based on the DVR threshold of 1.065, 53 participants (41.1%) were PiB(+).

Association between personality and amyloid

Initial bivariate correlations showed that PiB DVR was negatively correlated with only conscientiousness ($r = -0.19$, $p = 0.03$) while there were no correlations between PiB DVR and other personality dimensions (table 1). Results of the multiple regression analyses with each personality trait as a predictor, PiB DVR as a dependent variable, along with covariates are shown in table 2. Conscientiousness was consistently negatively associated with PiB DVR. Neither the parameter estimates, model fits, nor significance levels were substantially affected by adding education, BMI, and hypertension to the models as covariates. Power estimates for the conscientiousness models were 0.94 in model 1 ($f^2 = 0.132$, $R^2 = 0.117$, $\alpha = 0.05$, $n = 129$, predictors = 3), 0.95 in model 2 ($f^2 = 0.148$, $R^2 = 0.129$, $\alpha = 0.05$, $n = 129$, predictors = 4), and 0.92 in model 3 ($f^2 = 0.153$, $R^2 = 0.133$, $\alpha = 0.05$, $n = 129$, predictors = 6).

Correlation between personality and BMI, vascular risk factors, physical activity

To test whether conscientiousness affects amyloid accumulation through lifestyle, we examined vascular factors, BMI, and physical activity in our data. As a whole, the prevalence of vascular and metabolic disorders was low, particularly diabetes, TIA, and stroke. When we compared vascular risk factors, BMI and physical activity variables between PiB(–) and PiB(+) group, there were no differences. There were also no significant correlations between conscientiousness and any of these variables (table 1). These results suggest no convincing association between personality and variables that are related to indices of cardiovascular health in this study.

Discussion

Our results provide evidence supporting associations between personality and A β accumulation. In particular, we found that higher conscientiousness was associated with

lower A β deposition. This association was unaffected by adjustment for age, gender, education, and a number of lifestyle and health variables.

We did not confirm an association between A β and neuroticism as reported in other studies (Schultz et al., 2019; Tautvydaite et al., 2017). This may be attributed to lower scores for neuroticism in our participants, resulting in a floor effect. Previous studies have reported associations between conscientiousness and higher A β in CSF (indicative of lower brain A β) in a group of normal and cognitively impaired individuals (Tautvydaite et al., 2017), while another study using PET imaging of A β in cognitively normal older people found no relationship with conscientiousness (Schultz et al., 2019). Our study is the only one of which we are aware to report an association between conscientiousness and PET measures of brain A β .

We conjectured that conscientiousness plays a role in amyloid pathology by affecting an individual's health behaviors, physical activity, cognitive and social activities, or response to stress over the lifetime (Hampson, 2012; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). This has been supported by numerous observational studies that examined associations between personality and health behaviors. In terms of physical activity, higher conscientiousness has been associated with more time spent in physical activity, physical inactivity was related to a steeper decline in conscientiousness, and lower neuroticism and higher conscientiousness were associated with more physical activity, less inactivity, and sedentary behavior (Allen, Magee, et al., 2017; Stephan et al., 2018; Sutin et al., 2016). Studies using A β PET in cognitively normal older adults have shown that higher levels of self-reported physical activity are associated with lower levels of A β (Brown et al, 2013; Head et al., 2012, Okonkwo et al, 2014; Liang et al. 2010), consistent with the hypothesis that exercise modulates A β production and clearance through direct and indirect pathways (Brown et al, 2019).

In addition to physical activity, unhealthy behaviors are negatively correlated to conscientiousness and positively correlated to neuroticism (Allen, Walter, & McDermott, 2017). Conscientiousness has been associated with higher fruit and vegetable intake (Sutin et al., 2016), lower smoking (Hakulinen, Hintsanen, et al., 2015; Sutin et al., 2016), lower alcohol consumption (Hakulinen, Elovainio, Batty, et al., 2015), and a lower risk for obesity (Gerlach, Herpertz, & Loeber, 2015; Jokela et al., 2013; Sutin, Ferrucci, Zonderman, & Terracciano, 2011; Sutin & Terracciano, 2016, 2017) and depression (Hakulinen, Elovainio, Pulkki-Raback, et al., 2015). Based on associations between A β and a variety of cardiovascular risk factors such as hypertension, hypercholesterolemia, and physical activity (Gottesman et al., 2017; Rodrigue et al., 2013; Shah et al., 2012), it is reasonable to conjecture that lifelong personality traits might affect the risk of A β deposition through these processes. Although it is difficult to infer causality, the reliability and stability of personality traits that we found are consistent with prior reports, and suggest that personality could be a stable factor that might drive brain A β over a lifetime.

Nevertheless, when we examined relationships between personality or A β and lifestyle factors, we were unable to isolate the lifestyle or health factors that may be responsible for the association between conscientiousness and amyloid deposition. These negative findings

could reflect the characteristics of our cohort, in which individuals generally have healthy lifestyles and few chronic medical conditions, or they could reflect stronger associations with health factors in midlife, which we were unable to measure. Despite these negative results, we still consider lifestyle modeling as the most reasonable explanation for the association between personality and A β .

We could not effectively test other potential mechanisms of these associations. For example, evidence suggests that personality may affect stress responses resulting in decreased stress, positive affect, and fulfillment of positive expectations (Besser & Shackelford, 2007). Moreover, given that high cortisol levels, known as a mediator of chronic stress response, may exacerbate the effect of amyloid on cognitive decline in preclinical AD (Pietrzak et al., 2017), better stress coping might drive less A β accumulation. Personality may also be related to the use of neural resources during cognitive computations, producing cognitive reserve seen in neural networks. Given that greater neural activity is associated with more A β release (Jagust & Mormino, 2011; Landau et al., 2012), higher conscientiousness may result in more efficient neural activation associated with less A β release and deposition. These ideas are conjectural and obviously require empirical support.

The present study has several limitations. First, this was not a population-based study but is biased by the volunteer nature of the subjects which limits the generalizability of our results. Second, this study employed a cross-sectional data analysis, which cannot determine causality. For example, it is possible that brain A β drives the personality findings, or that a third unmeasured variable drives both A β and personality. A longer-term study would improve our understanding of these issues. Third, there was a relatively small sample size. However, based on our results, the sample should be adequately powered to detect the relationships we investigated. Nevertheless, studies with a larger sample size would be better positioned to determine causal mechanisms.

In conclusion, our study suggests that conscientiousness is negatively associated with A β accumulation in cognitively normal aging. This suggests that conscientiousness may have a protective role against the development of amyloid pathology. Even though the mechanism is undetermined, this finding expands our understanding of the impact of personality on A β as a key factor of AD.

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Table 1.

Characteristics of 129 participants including descriptive statistics

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	
1 Sex, female	70 (54.3)	1																						
2 APOE ε4 carriers	36 (27.9)	-0.02	1																					
3 Age, year	72.1 ± 15.0	.10	-0.04	1																				
4 Education, year	16.9 ± 1.9	.01	-0.14	.18	1																			
5 PIB DVR	1.1 ± 0.2	.18	.37*	.20*	-.09	1																		
6 Agreeableness	35.8 ± 4.3	.16	-0.10	-0.06	-0.09	-0.08	1																	
7 Conscientiousness	35.1 ± 5.8	-0.04	-0.11	-0.07	.09	-.19*	.32	1																
8 Extraversion	27.5 ± 6.1	.18	.04	-0.11	-0.14	.05	.14	.02	1															
9 Neuroticism	18.0 ± 4.8	.03	.10	-0.03	-0.17	.03	-.39*	-.37*	-.15	1														
10 Openness	38.0 ± 6.2	-0.04	.03	-0.15	.13	-0.11	.05	.09	.49*	-.12	1													
11 MMSE	29.0 ± 1.1	.02	-0.01	-0.04	.02	.05	.18	-0.05	-.99	-0.16	-0.09	1												
12 GDS	3.7 ± 3.3	.04	.07	-0.04	-0.12	.05	-0.08	-.31*	-.11	.55*	-0.02	-0.07	1											
13 Processing speed	-0.0 ± 0.9	.16	-0.01	-0.46*	-0.02	-0.12	.03	.12	.03	.06	.02	.08	.15	1										
14 Working memory	-0.4 ± 1.6	.01	.03	-.35*	.96	-0.14	-0.02	.11	.09	.04	.26*	.22*	.07	.19*	1									
15 Episodic memory	-0.1 ± 1.9	-0.02	.08	-.36*	.09	-0.02	-0.05	.05	.03	-.09	.27*	.19*	-.01	.28*	.38*	1								
16 BMI	25.8 ± 4.2	-.14*	.09	.01	-0.15	-0.06	.04	-0.06	.11	.07	-0.06	.08	-0.12	-0.17	.05	-0.08	1							
17 Total Kcal	6271.7 ± 5578.1	-0.03	-0.05	-0.02	.05	.01	.19*	.11	.05	-0.14	.01	-0.01	-.20*	.05	-0.04	-0.12	.01	1						
18 Walking Miles	7.4 ± 8.9	.09	-0.02	.01	-0.02	-0.04	.20*	-.01	.21*	-.16	.13	-.18*	.01	.08	.08	-.14	.38*	1						

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	
19 Hours Seated	15.8 ± 3.7	-.09	.05	-.06	.07	-.07	.03	-.10	-.09	.03	.01	.07	.05	-.01	-.09	.02	-.05	-.21	-.16	1				
20 Hypertension	46 (35.7)	-.16	.08	.10	-.07	.09	.14	.01	-.04	-.15	-.08	.09	-.14	-.22*	-.17	-.15	.35*	-.15	-.21	.19	1			
21 Diabetes	9 (7.0)	-.05	-.10	.08	.03	-.08	.15	.02	.04	-.13	-.06	.98	-.14	-.01	-.04	-.08	.13	-.02	.05	-.14	.28*	1		
22 Hyperlipidemia	47 (36.4)	.15	.03	.27*	-.09	.14	.06	-.01	-.02	-.06	-.11	-.02	.03	-.02	-.17	-.04	.08	-.03	-.12	.17	.29*	.04	1	
23 Stroke/TIA	16 (12.2)	.01	.01	.26*	.08	.09	.07	-.04	-.15	.01	-.18	.03	.06	-.01	-.12	-.01	.06	.08	-.17	-.01	.11	-.07	-.01	1

Notes: APOE, apolipoprotein E; PiB, Pittsburgh compound B; DVR, distribution volume ratio; MMSE, mini-mental state examination; GDS, geriatric depression scale; TIA, transient ischemic attack; BMI, body mass index; Total Kcal, total calories expended by physical activities in last 2 weeks; Walking miles, incidental walking done in 1 week; Hours Seated; hours seated or lying down in 24 hours. Values in the third column represent the number (%) or mean ± SD. Values from the forth to the last columns represent correlation coefficients.

* $p < .05$ uncorrected for multiple comparisons. Big 5 inventory consists of 6–10. 13–15 mean composite z-scores. 20–23 as vascular risk factors are based on medical diagnosis.

Table 2.

Association between personality and amyloid

Model 1						
amyloid DVR ~ each personality trait + age + sex						
BFI dimension	β(SE)	95% CI	t	p	R²	f²
Agreeableness	-0.11 (0.09)	[-0.28,0.06]	-1.27	0.207	0.091	0.100
Conscientiousness	-0.19 (0.08)	[-0.36,-0.03]	-2.32	0.022 *	0.117	0.132
Extraversion	0.04 (0.09)	[-0.12,0.22]	0.49	0.624	0.082	0.100
Neuroticism	0.04 (0.09)	[-0.13,0.21]	0.48	0.633	0.081	0.088
Openness	-0.08 (0.09)	[-0.25,0.09]	-0.89	0.375	0.085	0.093
Model 2						
amyloid DVR ~ each personality trait + age + sex + education						
BFI dimension	β(SE)	95% CI	t	p	R²	f²
Agreeableness	-0.13 (0.09)	[-0.30,0.04]	-1.51	0.134	0.112	0.125
Conscientiousness	-0.19 (0.08)	[-0.35,-0.02]	-2.19	0.030 *	0.129	0.148
Extraversion	0.04 (0.09)	[-0.13,0.21]	0.45	0.656	0.097	0.107
Neuroticism	0.02 (0.09)	[-0.15,0.19]	0.27	0.789	0.097	0.107
Openness	-0.06 (0.09)	[-0.23,0.11]	-0.65	0.519	0.098	0.109
Model 3						
amyloid DVR ~ each personality trait + age + sex + education + BMI + HTN						
BFI dimension	β(SE)	95% CI	t	p	R²	f²
Agreeableness	-0.14 (0.09)	[-0.31,0.04]	-1.53	0.129	0.091	0.101
Conscientiousness	-0.19 (0.09)	[-0.35,-0.02]	-2.18	0.031 *	0.133	0.153
Extraversion	0.05 (0.09)	[-0.13,0.22]	0.53	0.600	0.082	0.089
Neuroticism	0.03 (0.09)	[-0.14,0.21]	0.34	0.732	0.081	0.088
Openness	-0.06 (0.09)	[-0.23,0.11]	-0.68	0.497	0.085	0.093

Note. BFI, Big Five Inventory; BMI, body mass index; HTN, hypertension

Standardized regression coefficients (β), t, p, R², and f² values correspond to multivariate regression models adjusted by covariates.

* p<0.05

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