
Personality Correlates of Midlife Cardiometabolic Risk: The Explanatory Role of Higher-Order Factors of the Five-Factor Model

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Abstract

Varying associations are reported between Five-Factor Model (FFM) personality traits and cardiovascular disease risk. Here, we further examine dispositional correlates of cardiometabolic risk within a hierarchical model of personality that proposes higher-order traits of Stability (shared variance of Agreeableness, Conscientiousness, inverse Neuroticism) and Plasticity (Extraversion, Openness), and we test hypothesized mediation via biological and behavioral factors. In an observational study of 856 community volunteers aged 30–54 years (46% male, 86% Caucasian), latent variable FFM traits (using multiple-informant reports) and aggregated cardiometabolic risk (indicators: insulin resistance, dyslipidemia, blood pressure, adiposity) were estimated using confirmatory factor analysis (CFA). The cardiometabolic factor was regressed on each personality factor or higher-order trait. Cross-sectional indirect effects via systemic inflammation, cardiac autonomic control, and physical activity were tested. CFA models confirmed the Stability “meta-trait,” but not Plasticity. Lower Stability was associated with heightened cardiometabolic risk. This association was accounted for by inflammation, autonomic function, and physical activity. Among FFM traits, only Openness was associated with risk over and above Stability, and, unlike Stability, this relationship was unexplained by the intervening variables. A Stability meta-trait covaries with midlife cardiometabolic risk, and this association is accounted for by three candidate biological and behavioral factors.

Prominent dimensions of personality predict many consequential life outcomes, including educational and work attainments, marital stability, major mental disorders, behaviors conducive to health or illness, disease-specific morbidity and mortality, and longevity (Bogg & Roberts, 2004; Hampson, 2012; Kotov, Gamez, Schmidt, & Watson, 2010; Ozer & Benet-Martinez, 2006; Poropat, 2009; Rothmann & Coetzer, 2003; Smith, Glazer, Ruiz, & Gallo, 2004; Suls & Bunde, 2005). Among health-related correlates of personality, much recent research has focused on premorbid indicators of disease risk, such as the metabolic syndrome (Mommersteeg & Pouwer, 2012). This construct reflects a clustering of metabolic abnormalities—insulin resistance, dyslipidemia, central adiposity, and elevated blood pressure—that increase risk for type 2 diabetes and incident cardiovascular disease, as well as for late-life cognitive impairment, accelerated brain atrophy, and mortality (Agarwal

et al., 2012; Dearborn et al., 2014; Feng et al., 2013; Gami et al., 2007; Rouch et al., 2014; Song et al., 2014; Suzuki et al., 2014). The metabolic syndrome may occur with heightened frequency in persons of an angry, distressed, or hostile disposition (for a review, see Mommersteeg & Pouwer, 2012), and with respect to the Five-Factor Model of personality (FFM), the metabolic syndrome has been found to be related to high Neuroticism and low levels of Agreeableness, Conscientiousness, Extraversion, and Openness to Experience (Phillips et al., 2010; Ross, Martin, Chen, & Miller, 2011; Sutin, Costa, et al., 2010; van Reedt Dorland, Giltay, Van Veen, Zitman, & Penninx, 2012).

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Associations between FFM traits and the metabolic syndrome, however, tend to be modest and often do not reach statistical significance (Mommersteeg & Pouwer, 2012). For instance, while generally trending in the expected direction, select studies have not replicated associations of the metabolic syndrome with Neuroticism (Ross et al., 2011; van Reedt Dortland et al., 2012), Conscientiousness (van Reedt Dortland et al., 2012), and/or Agreeableness (Ross et al., 2011). With Extraversion, nonsignificant trends suggest both increased and decreased risk for metabolic syndrome (Sutin, Costa, et al., 2010; van Reedt Dortland et al., 2012), with differential trends occurring after adjusting for the effects of other FFM traits (van Reedt Dortland et al., 2012). These nonsignificant associations may, in part, result from overadjusting for covariates that may act as mediators of the association between personality and the metabolic syndrome, such as substance use, diet, and physical activity (Mommersteeg & Pouwer, 2012). Moreover, various measurement challenges associated with both personality and the metabolic syndrome may attenuate observed effects. One important limitation is that most investigations rely exclusively on self-rated personality, which, like all mono-method personality assessment approaches, is subject to certain reporting biases (Biesanz & West, 2004; Galione & Oltmanns, 2014; Vazire & Carlson, 2010). In this regard, more robust associations might be observed if trait measurements were based instead on combined ratings from various informants, including both self- and other-reports (Connelly & Ones, 2010).

Similarly, the metabolic syndrome occasions some analytic difficulties as a metric of aggregated health risk. As a binary construct, the metabolic syndrome is said to be present when a certain number or constellation of indicator variables (high fasting glucose, low high-density lipoprotein [HDL] cholesterol, hypertriglyceridemia, elevated blood pressure, or high waist circumference) exceed criterion values variously defined by prominent health organizations (e.g., American Heart Association, National Cholesterol Education Program, International Diabetes Federation, and other expert bodies; Alberti et al., 2009). In many cohorts, particularly those of midlife or younger, prevalence of the fully established syndrome may be low, even though many individuals may exhibit subthreshold elevations on multiple indicators. In addition, reliance on threshold values of each component requires dichotomizing their distributions, with consequent loss of information. Indeed, a quantitative index of cardiometabolic risk constructed from continuous distributions of the same components better predicts incident cardiovascular disease than the traditional categorically defined metabolic syndrome (Agarwal et al., 2012). Thus, examining a continuous measure of risk may provide better insight into how personality relates to cardiometabolic abnormalities, particularly in a nonclinical population.

As noted above, several of the FFM traits have been associated with the metabolic syndrome (Ross et al., 2011; Sutin, Costa, et al., 2010; van Reedt Dortland et al., 2012). These study outcomes are also frequently interpreted as independent findings on an assumption of trait orthogonality; however, the FFM trait

dimensions are correlated. Many traits represented on common instruments like the NEO Personality Inventory and other FFM scales covary moderately (Costa & McCrae, 1992; Digman, 1997). Indeed, exploratory and confirmatory factor analyses support a hierarchical model of personality, in which two higher-order traits represent, respectively, the covariance of (a) Neuroticism (inverse), Agreeableness, and Conscientiousness; and (b) Extraversion and Openness (DeYoung, 2006; DeYoung, Peterson, & Higgins, 2002; DeYoung, Quilty, & Peterson, 2007; Digman, 1997). First labeled α and β meta-trait by Digman (1997), DeYoung (2002) later retermed these latent variables *Stability* and *Plasticity* to denote key attributes of the shared variance among their constituent traits. High Stability is said to reflect an individual's ability to retain equipoise across multiple domains, and so avoid disruptions in mood (low Neuroticism), social relationships (Agreeableness), and motivated behavior (Conscientiousness). In contrast, Plasticity reflects an individual's capacity to explore and adapt flexibly in both behavior (Extraversion) and cognition (Openness) to new or changing circumstances. Hence, the similar associations with cardiometabolic risk seen across several FFM traits may conceivably stem from the common variance captured by uninvestigated higher-order meta-trait.

Like the hierarchical structure of personality, recent confirmatory factor analyses support continuously distributed components of the metabolic syndrome to exhibit substantial common variance that can be modeled as a single second-order factor underlying four first-order factors (blood pressure, insulin resistance, weight/adiposity, and dyslipidemia), consistent with the hypothesis of a unitary cardiometabolic syndrome (Shen et al., 2003). This structure has been replicated in multiple study samples (including that of the present article) and is robust to variation in age, sex, ethnicity, and history of cardiovascular disease (Marsland, McCaffery, Muldoon, & Manuck, 2010; McCaffery, Marsland, Strohacker, Muldoon, & Manuck, 2012; McCaffery, Shen, Muldoon, & Manuck, 2007; Shen et al., 2003).

In line with transactional and interactional stress models of personality and health (for reviews, see Smith, 2006, and Smith & MacKenzie, 2006), preliminary evidence suggests that the Stability meta-trait is associated with broad biological processes that could indirectly influence cardiometabolic risk. Specifically, several of the trait components of Stability (e.g., Neuroticism/negative affectivity, anger/hostility, Conscientiousness) have been independently linked to systemic inflammation and cardiac automatic control (low heart rate variability [HRV]; Bleil, Gianaros, Jennings, Flory, & Manuck, 2008; Chapman et al., 2009; Chapman et al., 2011; Demaree & Everhart, 2004; Huang et al., 2013; Luchetti, Barkley, Stephan, Terracciano, & Sutin, 2014; Marsland, Prather, Petersen, Cohen, & Manuck, 2008; Sloan et al., 1994; Sutin, Costa, et al., 2010; Turiano, Mroczek, Moynihan, & Chapman, 2013; Watkins, Grossman, Krishnan, & Sherwood, 1998). In turn, we have previously found systemic inflammation and HRV likewise associated with cardiometabolic risk (Marsland et al., 2010; McCaffery et al., 2012). Similar associations with the metabolic syndrome and its

components have been reported by others (Han et al., 2002; Hemingway et al., 2005; Lee & Pratley, 2005; Rana, Nieuwendorp, Jukema, & Kastelein, 2007; Soares-Miranda et al., 2012). Taken together, this suggests two potential biological pathways linking dispositional traits to metabolic abnormalities: inflammation and autonomic control.

Consistent with health behavior models linking personality traits to health (Smith, 2006; Smith & MacKenzie, 2006), the Stability trait may manifest in health practices that indirectly influence cardiometabolic risk. For instance, components of the Stability meta-trait—Neuroticism (positively), Agreeableness (negatively), and Conscientiousness (negatively)—have each been associated with health behaviors such as substance use, imprudent diet, and physical inactivity (Bogg & Roberts, 2004; Bogg & Vo, 2014; Bunde & Suls, 2006; Courneya & Hellsten, 1998; Malouff, Thorsteinsson, Rooke, & Schutte, 2007; Malouff, Thorsteinsson, & Schutte, 2006; Terracciano & Costa, 2004). Moreover, the latter of these, sedentariness, is a well-established correlate and predictor of the metabolic syndrome (Rennie, McCarthy, Yazdgerdi, Marmot, & Brunner, 2003), which suggests that physical inactivity could contribute to associations between Stability and cardiometabolic risk.

The purpose of this study was to further investigate relations of personality to cardiometabolic risk, as seen in a nonpatient sample of midlife community volunteers. We initially examined the full suite of FFM traits as univariate and multivariate predictors of cardiometabolic risk. We hypothesized that Agreeableness and Conscientiousness would be negatively associated with risk, whereas Neuroticism would be positively related. Following these preliminary analyses, we next accounted for covariation among the FFM traits by estimating higher-order latent factors of Stability and Plasticity, in accord with literature on hierarchical structure of personality (DeYoung, 2006; DeYoung et al., 2002; Digman, 1997). Based on past literature, we hypothesized that Stability would predict lower cardiometabolic risk, whereas Plasticity's role in our investigation was exploratory. Trait ratings were obtained from multiple informants, and our index of cardiometabolic risk was likewise estimated by structural modeling from multiple indicators. In addition, consistent with hypothesized mediational pathways, we expected that the relation between Stability and lower cardiometabolic risk would be accounted for by biological and behavioral factors, including autonomic control and inflammatory markers, or by level of physical activity.

METHOD

Participants

Data for the present study were derived from the University of Pittsburgh Adult Health and Behavior (AHAB) project, a registry of behavioral and biological measurements on non-Hispanic Caucasian and African American individuals (30–54 years old) recruited in 2001–2005 via mass-mail solicitation from communities of southwestern Pennsylvania in the United States

(principally Allegheny County; Manuck, Phillips, Gianaros, Flory, & Muldoon, 2010; Marsland et al., 2010). Exclusion criteria included history of atherosclerotic cardiovascular, chronic kidney, or liver disease; past-year cancer treatment; neurologic disorders; and psychotic illness. Other exclusions included pregnancy and using insulin, nitrates, glucocorticoid, antiarrhythmic, psychotropic, or prescription weight-loss medications. Informed consent was obtained in accordance with approved protocol guidelines of the University of Pittsburgh Institutional Review Board.

The requisite personality data were available for 1,164 AHAB participants. Additional exclusion criteria for the current analyses included use of antihypertensives, oral hypoglycemics, and cholesterol-lowering medications. Of the resulting 948 participants, individuals were further excluded from the current analyses for use of immunosuppressants, cold medication, or antibiotics at the time of testing; interleukin-6 (IL-6) levels exceeding upper limits of detection for the high-sensitivity assay used here (>10 pg/ml); or C-reactive protein (CRP) elevation (>10 mg/L), indicative of acute illness (e.g., common respiratory infection). This resulted in a final sample of 856 participants, whose demographic characteristics are summarized in Table 1.¹

Measures

Personality Assessments. Each participant completed the 240-item Revised NEO Personality Inventory (NEO PI-R), which includes five subscales assessing the FFM personality domains: Neuroticism, Agreeableness, Openness to Experience, Extraversion, and Conscientiousness (Costa & McCrae, 1992). Up to two informants also rated the participant using the 60-item abbreviated form (i.e., the NEO Five-Factor Inventory [NEO-FFI]), with the majority of participants (88%) having ratings from two informants. Informants were chosen by the participant, and they included spouses/partners (30%), parents (9%), siblings (12%), other close relatives (12%), close friends (31%), or other (6%). To be consistent across self- and informant report, the subset of 60 items from the NEO PI-R that overlapped with the NEO-FFI was used to create the five self-reported FFM traits. Thus, most participants had three subscale scores (i.e., ratings by the participant himself or herself and two other informants) for each 12-item personality trait.

Cardiometabolic and Inflammatory Assessments. The components of cardiometabolic risk were assessed in the morning after a 12-hour overnight fast, as detailed by Manuck and colleagues (2010). Blood pressure was measured by sphygmomanometry as the mean of two consecutive readings obtained in a seated position. At this visit, a nurse completed a medical history interview, determined body mass index (BMI; kg/m²), and drew a 40 ml blood sample. Determination of fasting serum lipids, glucose, and insulin was performed by the Heinz Nutrition Laboratory, University of Pittsburgh Graduate School of Public

Table 1 Descriptive Statistics of Demographic, Personality, and Biomedical Characteristics

	Mean (SD) or %		
Demographics			
Age	44.36 (6.86)		
Sex (male)	46.0		
Race (Caucasian)	86.0		
Education (y)	15.87 (2.84)		
Current smokers	15.9		
Personality			
(self, informant x & y)	Self	Inf _x	Inf _y
Agreeableness	34.01 (5.76)	33.22 (7.77)	33.97 (7.10)
Conscientiousness	33.48 (6.35)	33.22 (7.77)	36.35 (8.20)
Neuroticism	16.55 (7.92)	17.64 (9.01)	16.35 (8.33)
Extraversion	28.70 (6.85)	29.62 (7.53)	31.14 (7.44)
Openness to Experience	28.28 (6.95)	26.78 (6.61)	27.31 (5.88)
Cardiometabolic risk indicators			
Insulin (μ U/ml)	12.41 (6.29)		
Glucose (mg/dl)	94.87 (13.90)		
BMI (kg/m^2)	26.83 (5.42)		
Waist circumference (in.)	35.42 (6.04)		
HDL cholesterol (mg/dl)	54.27 (14.45)		
Triglycerides (mg/dl)	117.22 (80.35)		
Systolic BP (mm Hg)	115.52 (13.35)		
Diastolic BP (mm Hg)	77.87 (9.36)		
Mediators			
IL-6 (pg/mL)	1.74 (1.68)		
CRP (mg/L)	1.56 (1.80)		
Paced RMSSD*	3.15 (0.65)		
Unpaced RMSSD*	3.21 (0.57)		
Paced HRV*	6.11 (1.31)		
Unpaced HRV*	5.72 (1.13)		
Physical activity (kcal/week)	2486.39 (1836.46)		

Note. BMI = body mass index; HDL = high-density lipoprotein; BP = blood pressure; IL = interleukin; CRP = C-reactive protein; RMSSD = root mean square of successive differences; HRV = heart rate variability.

*Variable is natural log transformed.

Health, as described previously (Muldoon, Nazzaro, Sutton-Tyrrell, & Manuck, 2000). IL-6 was determined using a high-sensitivity quantitative sandwich enzyme immunoassay kit (R & D Systems, Minneapolis, MN). C-reactive protein (CRP) was measured with the BNII nephelometer from Dade Behring (Newark, DE) using a particle-enhanced immunonephelometric assay (Marsland et al., 2010).

Resting Cardiac Vagal Activity. After a 10-minute rest period, heart rate was recorded continuously using a two-lead electrocardiogram (ECG) during two successive 5-minute periods of unpaced and paced breathing. During the paced breathing segment, participants breathed 11 breaths per minute in response to two auditory tones (inhale, exhale). Respiration was monitored by a thoracic strain-gauge. ECG signals were digitized at a sampling rate of 1000 Hz (LabView acquisition software, National Instruments Corporation, Austin, TX). An interbeat-interval time series was derived from the ECG, corrected for arti-

facts in the R-wave detection, and the band-limited variance within the high frequency (HF-HRV; 0.12–0.40 Hz) was extracted using PhysioScripts (Christie & Gianaros, 2013). The square root of the mean of the squares of successive normal-to-normal interval differences (RMSSD) was calculated, and the unpaced and paced HF-HRV and RMSSD distributions normalized by natural-log transformations.

Physical Activity. Participants completed the Paffenbarger Physical Activity Questionnaire, which estimated kilocalorie expenditure (kcal) over a 7-day period (e.g., blocks walked, stairs climbed, leisure time activity; Paffenbarger, Wing, & Hyde, 1978).

Analytic Plan

Overview. Study hypotheses were tested using structural equation modeling (SEM) estimated in Mplus Version 7.2 (Muthén & Muthén, 1998–2012). Missing data were handled using full information maximum likelihood. Due to the sensitivity of the chi-square test to negligible sources of ill fit in large samples using real-world data, we followed convention and relied on multiple alternative fit indices as a reasonable but conservative approach to evaluate model fit (Browne, Cudeck, & Bollen, 1993; Hu & Bentler, 1999). Appropriate fit corresponds with the root mean square error of approximation (RMSEA) and its 90% confidence interval (CI) with values less than .05, the comparative fit index (CFI) close to .95 or greater, and the standardized root mean residual (SRMR) of less than .08.

We first tested measurement models of the personality and metabolic assessments separately. Then, using the resulting personality and cardiometabolic risk latent variables, the first-order FFM personality factors and corresponding higher-order latent factors were examined as predictors of cardiometabolic risk. Finally, we tested whether inflammation, autonomic control, and physical activity account for associations of personality with cardiometabolic risk, as would be predicted from mediational models of personality and health outcomes (Smith, 2006). Analyses controlled for age in years, sex (0 = male; 1 = female), and race (0 = Caucasian; 1 = African American).

PRELIMINARY RESULTS

Latent Factor Model Fitting

Personality Models. First, five correlated personality latent factors were modeled representing each of the FFM traits: Neuroticism, Agreeableness, Openness to Experience, Conscientiousness, and Extraversion. We used the multi-informant modeling approach described by DeYoung (2006). Specifically, each personality latent variable had three indicators: a self-report and two informant reports. Informant effects were accounted for by correlating the residual variances of the five personality subscales. The resulting model evinced appropriate fit, $\chi^2 = 96.78$,

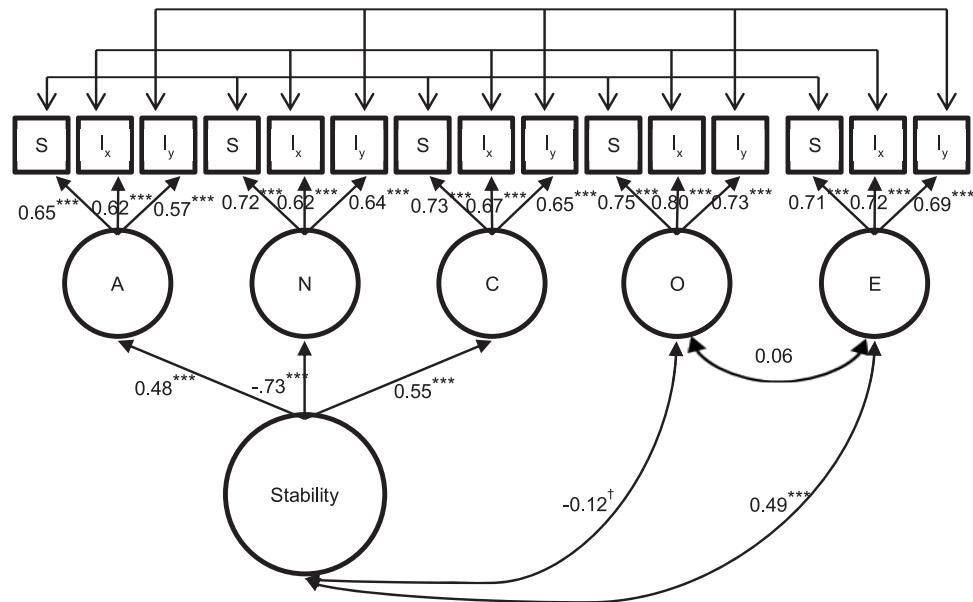


Figure 1 A higher-order stability factor underlying Agreeableness, Neuroticism, and Conscientiousness. Standardized path coefficients are reported. Residual arrows for latent factors are omitted to simplify the figure. The first letter of each personality trait is used as its abbreviation. Each personality trait is a latent factor based on responses from self-report (i.e., S) and informant report (i.e., I_x and I_y). Model fit: $\chi^2 = 138.10$, $df = 54$, $p < .001$, CFI = .98, RMSEA = .04, 90% CI [.03, .05], SRMR = .04. *** $p < .001$. ** $p < .01$. * $p < .05$. † $p < .10$.

$df = 50$, $p < .001$; RMSEA = .03, 90% CI [.02, .04]; SRMR = .03; CFI = .99.

Next, we tested a higher-order factor model, with factors based on Stability (Neuroticism, Conscientiousness, Agreeableness) and Plasticity (Extraversion, Openness), to explain the common variances among the five personality factors. A Plasticity factor was not supported because the Openness and Extraversion latent variables did not covary significantly ($r = .06$, $p = .17$). Therefore, these variables were examined as trait-level latent factors in subsequent models. A Stability factor was supported given that all were significantly correlated ($r_s = .25\text{--}.43$, all $p < .001$), and the resulting model (depicted in Figure 1 with factors for Openness, Extraversion, and higher-order Stability) had appropriate fit to the data, $\chi^2 = 138.10$, $df = 54$, $p < 0.01$; RMSEA = .04, 90% CI [.03, .05]; SRMR = .04; CFI = .98. The fit appeared comparable to the correlated FFM model fit, as the 90% RMSEA CIs were overlapping, and SRMR and CFI were only slightly higher in the Stability model despite the four degrees of freedom gained by constraining the latent correlations of Extraversion and Openness with Agreeableness, Neuroticism, and Conscientiousness to 0.0, and limiting the Extraversion and Openness associations to each other and the latent factor.

Cardiometabolic Risk Model. Consistent with previous AHAB investigations (Marsland et al., 2010; McCaffery et al., 2012), a second-order factor model representing cardiometabolic risk was estimated with four first-order factors having two indicators each (see Figure 2): insulin resistance (i.e., natural log-transformed fasting insulin and glucose), adiposity (i.e.,

BMI, waist circumference), dyslipidemia (i.e., fasting HDL and natural log-transformed triglycerides), and blood pressure (i.e., systolic and diastolic). The resulting model exhibited appropriate fit, $\chi^2 = 34.35$, $df = 20$, $p = .02$; RMSEA = .03, 90% CI [.01, .05]; SRMR = .02, CFI = .99.

RESULTS

Structural Analysis of Personality, Biological, Behavioral, and Cardiometabolic Factors

Associations Between FFM Personality Traits and Cardiometabolic Risk. The latent cardiometabolic factor (depicted in Figure 2) was regressed on the individual correlated FFM latent factors. First, in five separate univariate models (see Table 2), the Agreeableness, Conscientiousness, and Openness traits were related inversely and significantly to risk, whereas a positive association with Neuroticism was marginally significant ($p = .056$). Extraversion was unrelated to risk ($\beta = -0.01$, $p = .74$). When simultaneously controlling for each FFM trait in a multivariate model (Table 2), only Conscientiousness and Openness emerged as independent predictors. Together, the personality traits explained 7% of the variance in cardiometabolic risk (over and above the effect of the covariates).

Associations Between the Higher-Order Stability Factor and Cardiometabolic Risk. We next regressed cardiometabolic risk on the Stability factor alone and then controlling for Openness, given the marginal correlation between Openness

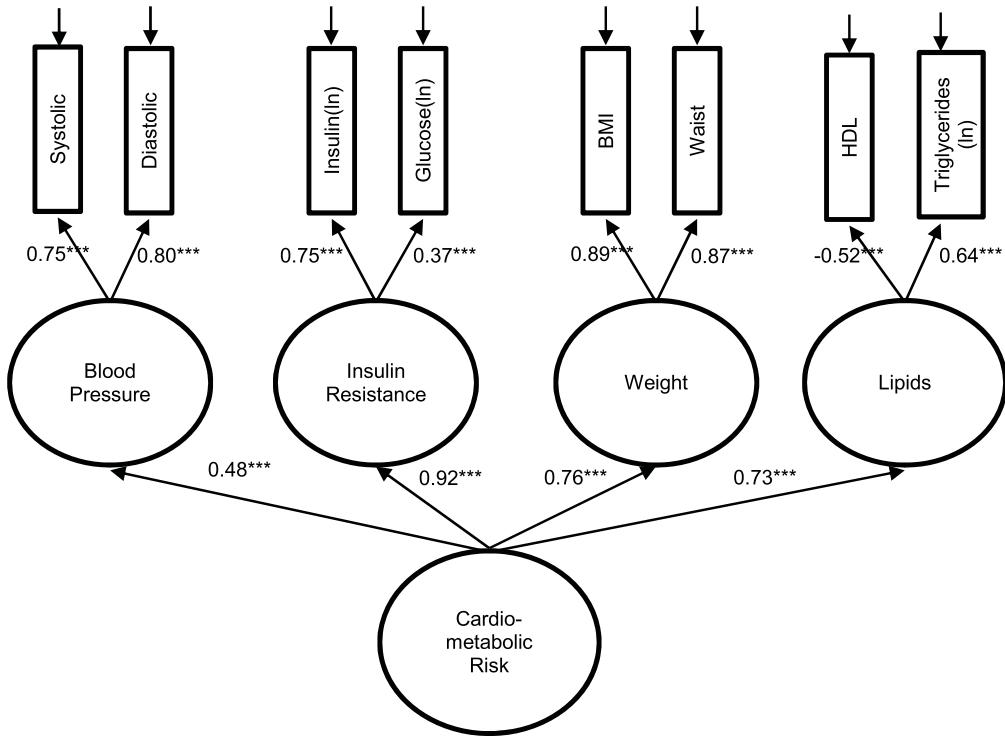


Figure 2 The factor structure of cardiometabolic risk. Standardized path coefficients are reported. Residual arrows for latent factors are omitted to simplify the figure. BMI = body mass index; HDL = high-density lipoprotein. Model fit: $\chi^2 = 34.35$, $df = 20$, $p = .02$; RMSEA = .03, 90% CI [.01, .05]; SRMR = .02, CFI = .99. *** $p < .001$. ** $p < .01$. * $p < .05$. † $p < .10$.

Table 2 The Associations Between the FFM Personality Traits With Cardiometabolic Risk

	Associations With Cardiometabolic Risk	
	Unadjusted Effects ^a	Adjusted Effects (Unique Effects) ^b
Agreeableness	$\beta = -0.11$, $p = .02$, $R^2 = .01$	$\beta = -0.08$, $p = .12$
Neuroticism	$\beta = 0.09$, $p = .056$, $R^2 = .01$	$\beta = 0.01$, $p = .93$
Conscientiousness	$\beta = -0.15$, $p = .001$, $R^2 = .02$	$\beta = -0.19$, $p < .001$
Extraversion	$\beta = -0.01$, $p = .74$, $R^2 = 0$	$\beta = 0.04$, $p = .49$
Openness	$\beta = -0.13$, $p = .004$, $R^2 = .02$	$\beta = -0.18$, $p < .001$

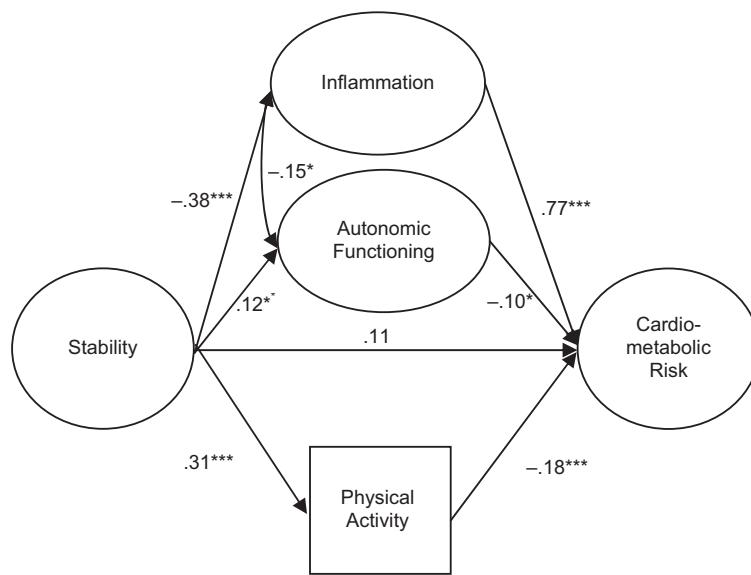
Note. $N = 856$. Standardized regression coefficients are reported. Significant path coefficients are **boldfaced**. FFM = Five-Factor Model.

^aEach personality factor was examined as a predictor of the metabolic syndrome without controlling for the effects of the other four personality traits. R^2 is the total variance explained by each personality trait net of the effect of covariates. ^bThe cardiometabolic risk latent factor was regressed on all five personality factors simultaneously. Each path coefficient is the association with the respective personality trait controlling for the effects of the other four personality traits. R^2 is the total variance explained by the combination of the personality traits net of the effect of covariates.

and Stability. When examined alone, greater Stability was associated with lower cardiometabolic risk ($\beta = -0.14$, $p = .01$). When controlling for Openness, the Stability factor remained a significant predictor ($\beta = -0.19$, $p = .001$). Openness also remained a predictor of cardiometabolic risk independent of Stability ($\beta = -0.16$, $p < .001$). Together, Stability, Openness, and Extraversion accounted for 8% of the variance in the cardiometabolic risk (over and above the effect of covariates).

To examine whether residual variance in specific traits composing the Stability factor is associated with cardiometabolic

risk above and beyond their common variances (i.e., Stability), we estimated an alternative hierarchical model (i.e., a bifactor or general-specific model). Specifically, the nine self- and informant ratings of Neuroticism, Conscientiousness, and Agreeableness were loaded on a Stability general factor. Orthogonal specific factors were estimated for Neuroticism, Conscientiousness, and Agreeableness by loading the corresponding informant and self-report scores on each factor. We then simultaneously regressed cardiometabolic risk on the general Stability and three specific trait factors. The Stability general



Cross-Sectional Indirect Effect [95% CI]	
Inflammation	-0.29 [-0.42, -0.18]
Autonomic functioning	-0.01 [-0.03, -0.0001]
Physical activity	-0.06 [-0.09, -0.03]

Figure 3 Pathways accounting for the effect of the Stability factor on cardiometabolic risk. Standardized path coefficients are reported. Model controls for age, race, and sex. Latent variables are represented by circles and observed variables as squares. The model was simplified by not depicting indicators of each latent variable. Model fit: $\chi^2 = 783.68$, $df = 273$, $p < .001$; CFI = .96; RMSEA = .047, 90% CI [.04, .05]; SRMR = .05. *** $p < .001$. ** $p < .01$. * $p < .05$. $^t p < .10$.

factor was again a significant predictor of cardiometabolic risk, and there were no additional trait-specific contributions by Neuroticism ($p = .11$), Conscientiousness ($p = .11$), or Agreeableness ($p = .69$). This supported focusing on Stability in the following analyses.

Accounting for Effects of the Stability Factor on Cardiometabolic Risk. The model depicted in Figure 3 was estimated to examine the extent to which proposed intermediate processes accounted for the relation of Stability with cardiometabolic risk, including systemic inflammation (i.e., latent factor with two indicators and factor loadings fixed to 1 for model identification purposes: C-reactive protein and interleukin-6), autonomic functioning (i.e., latent factor with four indicators: paced and unpaced HRV and paced and unpaced RMSSD), and physical activity (observed variable). Specifically, we tested the association of the Stability factor with each of the three intermediate processes, and, in turn, the association between the intermediate processes and cardiometabolic risk factor. By examining the pathways together, their intercorrelatedness was accounted for and the independent indirect effect of each pathway was established. The indirect

effect was estimated using the Model Indirect command in Mplus. The corresponding bias-corrected CIs were estimated using the Asymmetric Confidence Interval method implemented in the Proclinal program, which accounts for the non-normal distribution of the mediated effect (MacKinnon, Fritz, Williams, & Lockwood, 2007). The same approach was then used to examine whether the three factors account for the association between Openness (as opposed to Stability) and cardiometabolic risk. Specifically, the associations between Openness alone and the indirect processes, and, in turn, cardiometabolic risk were tested (not depicted).

The model examining the indirect pathways of the Stability effect adequately fit the data (Figure 3). The association between the Stability factor and cardiometabolic risk was accounted for by the indirect pathways. In our sample, all of the intervening variables were significantly correlated with each other ($r_s = .09\text{--}.23$, $p < .05$). The Stability factor was associated with systemic inflammation, low HRV (cardiac autonomic control), and a lower level of physical activity, which, in turn, were each associated with the cardiometabolic factor (see Figure 3 for effects). This resulted in three significant cross-sectional indirect effects. In sum, systemic inflammation, HRV, and a lower level

of physical activity each independently and indirectly explained the association between lower Stability and heightened cardiometabolic risk.

The cross-sectional indirect effects model examining Openness as the independent variable (as opposed to Stability) also adequately fit the data, $\chi^2 = 1425.46$, $df = 426$, $p < .001$; RMSEA = .05, 90% CI [.049, .06]; SRMR = .06, CFI = .93. The same variables, however, did not account for the association between Openness and cardiometabolic risk because Openness was not significantly associated with systemic inflammation ($\beta = -0.08$, $p = .11$), HRV ($\beta = 0.06$, $p = .11$), or physical activity ($\beta = 0.05$, $p = .24$).

DISCUSSION

We investigated whether a hierarchical model of FFM personality traits would be associated with midlife cardiometabolic risk and whether any such associations might be accounted for by candidate behavioral and biological processes. In line with much of the existing literature on self-reported ratings of FFM traits, multi-informant ratings of lower Conscientiousness, Agreeableness, and Openness and (marginally) higher Neuroticism showed heightened cardiometabolic risk. Thus, FFM traits provide important insight into cardiometabolic risk.

The present study adds to the existing literature by providing preliminary evidence that Conscientiousness, Agreeableness, and Neuroticism have shared effects on cardiometabolic risk that may be parsimoniously explained by an underlying latent factor, Stability. Specifically, the traits were significantly correlated, and when considered simultaneously as predictors, only Conscientiousness remained a significant predictor—highlighting the explanatory role of their shared variance. Moreover, when the shared variance of these traits was modeled as Stability, higher Stability was associated with lower risk. Variation in trait-level Neuroticism, Agreeableness, and Conscientiousness did not predict risk over and above Stability. In line with this finding, the Stability factor together with Extraversion and Openness explained a similar amount of variance in Stability (8%) as the FFM traits (7%). Taken together, the association of these three personality traits with cardiometabolic risk may be accounted for by the Stability meta-trait, which is thought to reflect individual differences in self-regulatory capacities common to emotion regulation, social management, and impulse control (DeYoung et al., 2002).

The association of the Stability trait with hierarchically modeled cardiometabolic risk could be explained through correlated variation in three intermediate factors: cardiac autonomic control (HRV), inflammation, and physical activity. Regarding the first of these, our findings agree with cumulative evidence showing HRV indicators of autonomic control associated with the metabolic syndrome (Stuckey, Tulppo, Kiviniemi, & Petrella, 2014). At a mechanistic level, it is plausible that these relations are bidirectional (Tentolouris, Argyrakopoulou, & Katsilambros, 2008), although current prospective evidence suggests compo-

nents of the metabolic syndrome do not predict changes in HRV indices (Soares-Miranda et al., 2012). In contrast, altered autonomic control, as reflected in suppressed HRV, has been linked prospectively to cardiometabolic outcomes, such as hypertension (Liao et al., 1997) and type 2 diabetes (e.g., Carnethon, Jacobs, Sidney, & Liu, 2003; Carnethon et al., 2006). Thus, autonomic control may also contribute to the acquisition of pre-clinical cardiometabolic abnormalities.

A second, independent biological indirect effect supported here was low-grade systemic inflammation, indexed by the inflammatory markers IL-6 and CRP. The association of personality traits with inflammation has been supported (e.g., Marsland et al., 2008; Sutin, Terracciano, et al., 2010; Turiano et al., 2013), and much previous work ties inflammatory markers to both individual components and clinical definitions of the metabolic syndrome (Han et al., 2002; Lee & Pratley, 2005; Marques-Vidal et al., 2002; Piché et al., 2005; Wannamethee et al., 2005). The latter associations have prompted speculation that systemic inflammation may play a primary etiologic role in the co-expression of insulin resistance, central adiposity, elevated blood pressure, and dyslipidemia (Marsland et al., 2010; Reilly & Rader, 2003). Nonetheless, obesity itself is considered a proinflammatory state, with adipocytes releasing proinflammatory cytokines, particularly IL-6, and, in turn, stimulating the peripheral production of CRP by hepatocytes (Berg & Scherer, 2005; Mohamed-Ali et al., 1997). Therefore, the inflammatory process linking Stability with cardiometabolic risk likely reflects some bidirectional association between inflammatory markers and the adiposity component of the latent metabolic factor.

The association between Stability and cardiometabolic risk was also accounted for by physical inactivity, a well-established correlate and predictor of the metabolic syndrome (Rennie et al., 2003). Exercise-related motivation and engagement in exercise have been related previously to two components of the Stability meta-trait, Neuroticism (negatively) and Conscientiousness (positively; e.g., Courneya & Hellsten, 1998). In our sample, physical activity was also significantly correlated with Neuroticism ($r = -.18$, $p < .001$), Conscientiousness ($r = .19$, $p < .001$), and Stability ($r = .15$, $p = .003$), but not with Agreeableness ($r = -.04$, $p = .40$).

Although, as noted in the introduction, hierarchical modeling of FFM traits often shows Extraversion and Openness to cohere as a second higher-order latent factor, termed *Plasticity*, they did not do so here. Instead, Extraversion and Openness were uncorrelated and had differential patterns of relations with both the Stability and latent cardiometabolic factors. In prior research estimating a Plasticity factor, the correlation of Extraversion and Openness has differed widely across samples and informant sources (e.g., $rs = .08\text{--}.34$; DeYoung, 2006; DeYoung et al., 2002). Thus, identification of this factor may be more sensitive than Stability to differing sample characteristics or measurement instruments.

As a result, in our multi-informant sample, it appeared that Openness may offer unique explanatory power in cardiometabolic risk not accounted for by a higher-order Plasticity trait.

Specifically, while only marginally related to Stability, greater Openness was associated independently with lower cardiometabolic risk. Unlike Stability, the latter association was not explained by inflammation, autonomic control, or physical activity. Elsewhere, Openness has been shown to covary inversely with inflammatory markers, although meta-analysis suggests this finding may be limited to CRP (e.g., Chapman et al., 2011; Luchetti et al., 2014) and may be moderated by race (Jonassaint et al., 2010). It is likely that the association of Openness with cardiometabolic risk is accounted for by other processes. In this regard, greater Openness has been linked to protective and health-promoting dietary habits (e.g., Lunn, Nowson, Worsley, & Torres, 2014; Möttus et al., 2013; Tiaanen et al., 2013) and health-related information seeking (e.g., Internet searches) in the general population (Bogg & Vo, 2014; Flynn, Smith, & Freese, 2006). These studies suggest that people higher in Openness may have a heightened awareness of factors affecting their physical well-being (i.e., greater health literacy), which warrants further study.

LIMITATIONS

Our findings are qualified by certain study limitations. The analyses are cross-sectional and thus preclude causal inferences regarding personality differences on health risk. Some correlated third variable might contribute to personality traits along the Stability spectrum or to Openness and, independently, to cardiometabolic risk, which was partly addressed by controlling for associated demographic factors such as age, sex, and race. Also, the present study cannot speak to the temporal ordering of associations. As a result, the present study cannot determine whether the hypothetical mediators are mediating or confounding processes, or a separate set of correlated outcomes impacted by personality and metabolic syndrome. Further longitudinal research, such as a prospective multiwave design with repeated personality and cardiometabolic risk measures, is needed to elucidate how personality (and any possible changes in personality) and mediating processes relate to the accelerating cardiometabolic risk that accompanies midlife aging (Graham & Lachman, 2012). Finally, a strength of our study was its reliance on a well-validated measure of personality completed by multiple informants. Yet, failure to detect a Plasticity factor may reflect attributes of this particular instrument as well as different sources of error that informant report adds. By using a latent variable approach, the present investigation attempted to remove error variances like reporting bias. Nonetheless, the current findings need to be replicated using other multi-informant measurements of personality.

CONCLUSION

In sum, our study provides new evidence that several FFM traits, particularly those contributing to the higher-order factor of Stability, are associated with aggregated cardiometabolic disease risk. In addition, this relationship was accounted for by a combi-

nation of biological (inflammatory and autonomic) pathways and physical activity. These explanatory factors nominate targets for future mechanistic mediation in longitudinal research, provide a basis for theory development linking personality to health, and may offer modifiable targets for intervention and prevention.

Declaration of Conflicting Interests

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Notes

1. The analyzed sample did not significantly differ from the unanalyzed participants with respect to age, sex, race, or self-reported personality traits. Compared to participants in the analyses, averaged informant scores for excluded participants tended to be slightly higher on self-reported Extraversion (Cohen's $d = .18$, $p = .004$). No other differences in self- or informant-reported FFM traits were detected. It was not possible to compare analyzed and unanalyzed participants with respect to cardiometabolic risk due to excluded subjects' use of medications affecting key indicators (e.g., effects of anti-hypertensives on measured blood pressure). It is to be expected that these subjects therefore also had higher aggregated cardiometabolic risk than did the analyzed sample as a whole.

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