



Published in final edited form as:

J Pers. 2016 December ; 84(6): 765–776. doi:10.1111/jopy.12216.

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

Sarah S. Dermody^a, Aidan G.C. Wright^a, JeeWon Cheong^b, Karissa G. Miller^a, Matthew F. Muldoon^c, Janine D. Flory^d, Peter J. Gianaros^a, Anna L. Marsland^a, and Stephen B. Manuck^a

^aDepartment of Psychology, University of Pittsburgh

^bDepartment of Health Behavior, University of Alabama

^cHeart and Vascular Institute, University of Pittsburgh School of Medicine

^dDepartment of Psychiatry, Mount Sinai School of Medicine

Abstract

Objective—Varying associations are reported between Five Factor Model (FFM) personality traits and cardiovascular disease risk within a hierarchical model of personality that posits higher-order risk. Here, we further examine dispositional correlates of cardiometabolic risk: Stability (shared variance of Agreeableness, Conscientiousness, inverse Neuroticism) and Plasticity (Extraversion, Openness), and test hypothesized mediation via biological and behavioral factors.

Method—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.

Results—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.

Conclusions—A Stability meta-trait covaries with midlife cardiometabolic risk, and this association is accounted for by three candidate biological and behavioral factors.

Corresponding Author: Sarah S. Dermody, M.S., Department of Psychology, University of Pittsburgh, 4311 Sennott Square, 210 South Bouquet Street, Pittsburgh, PA 15260, USA, ssdermody@gmail.com, Phone: 412 624 4302, Fax: 412 624 4428.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Keywords

Five Factor Model Personality Traits; Stability; Plasticity; Cardiometabolic Risk; Metabolic Syndrome

Introduction

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, 2004a; 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 pre-morbid 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 review see Mommersteeg & Pouwer, 2012), and with respect to the Five Factor Model of personality (FFM), the metabolic syndrome has been found 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 Jr, et al., 2010; van Reedt Dortland, Giltay, Van Veen, Zitman, & Penninx, 2012).

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, non-significant trends both suggest increased and decreased risk for metabolic syndrome (Sutin, Costa Jr, 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 non-significant associations may, in part, result from over-adjusting 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, are 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 non-clinical population.

As noted above, several of the FFM traits have been associated with the metabolic syndrome previously (Ross et al., 2011; Sutin, Costa Jr, 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-traits by Digman (1997), DeYoung (2002) later re-termed 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-traits.

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 systematic inflammation and cardiac autonomic 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 Jr, 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, Nieuwdorp, 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, 2004b; 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 (Huang & Liu, 2014; 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 non-patient 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, USA (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; or 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 1164 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; IL-6 levels exceeding upper limits of detection for the high-sensitivity assay used here (>10 pg/ml); or 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 5 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 included spouses/partners (30%), parents (9%), siblings (12%), other close relatives (12%), close friends (31%), or other (6%). To be

¹The 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 antihypertensives 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.

consistent across self- and informant-report, the subset of 60-items from the NEO-PI-R that overlapped with the NEO-FFI were used to create the 5 self-reported FFM traits. Thus, most participants had 3 subscale scores (i.e., ratings by participant him/her-self 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/m^2) 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 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 2-lead electrocardiogram (ECG) during two successive 5-minute periods of unpaced and paced breathing. During the paced breathing segment, participants breathed 11 breaths/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 Texas). An interbeat-interval time series was derived from the ECG, corrected for artifacts 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 $< .05$, 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 1st-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: self- 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 ($\chi^2 = 96.78$, $df=50$, $p<.001$; 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–.43, all p 's $<.001$) and the resulting model (depicted in Figure 1 with factors for Openness, Extraversion, and higher-order Stability) had appropriate fit to the data ($\chi^2=138.10$, $df=54$, $p<.001$; 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 4 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., SBP, DBP). 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, while 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 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 comprising the Stability factor are 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 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 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 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 Prodcin 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 if 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–.23; p 's < .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 with 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 if 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 components 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 et al., 2003; Carnethon et al., 2006). Thus, autonomic control may also contribute to the acquisition of preclinical 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 (Huang & Liu 2014). 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., $r = .08 - .34$ [DeYoung et al., 2002; DeYoung 2006]). 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., 2011b; 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., Möttus et al., 2013, Tiainen et al., 2013, Lunn et al., 2014) and health-related information-seeking (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 (greater health literacy), which warrants further study.

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 if 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 multi-wave 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.

In sum, our study provides new evidence that several FFM traits, particularly those contributing to the higher-order factor of Stability, associate with aggregated

cardiometabolic disease risk. In addition, this relationship was accounted for by a combination 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.

Acknowledgments

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: Preparation of this manuscript was supported by a grant awarded to Dr. Stephen Manuck (PO1 HL040962) from the National Institutes of Health.

References

- Agarwal S, Jacobs DR, Vaidya D, Sibley C, Jorgensen NW, Rotter JI, et al. Metabolic syndrome derived from principal component analysis and incident cardiovascular events: The multi ethnic study of atherosclerosis (MESA) and health, aging, and body composition (Health ABC). *Cardiology research and practice*. 2012
- Alberti K, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the Metabolic Syndrome A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*. 2009; 120(16):1640–1645. [PubMed: 19805654]
- Berg AH, Scherer PE. Adipose tissue, inflammation, and cardiovascular disease. *Circulation research*. 2005; 96(9):939–949. [PubMed: 15890981]
- Biesanz JC, West SG. Towards Understanding Assessments of the Big Five: Multitrait-Multimethod Analyses of Convergent and Discriminant Validity Across Measurement Occasion and Type of Observer. *Journal of Personality*. 2004; 72(4):845–876. [PubMed: 15210019]
- Bleil ME, Gianaros PJ, Jennings JR, Flory JD, Manuck SB. Trait negative affect: toward an integrated model of understanding psychological risk for impairment in cardiac autonomic function. *Psychosomatic Medicine*. 2008; 70(3):328–337. [PubMed: 18378862]
- Bogg T, Roberts BW. Conscientiousness and health-related behaviors: a meta-analysis of the leading behavioral contributors to mortality. *Psychological bulletin*. 2004a; 130(6):887. [PubMed: 15535742]
- Bogg T, Roberts BW. Conscientiousness and health-related behaviors: a meta-analysis of the leading behavioral contributors to mortality. *Psychol Bull*. 2004b; 130(6):887–919. [PubMed: 15535742]
- Bogg T, Vo PT. Openness, neuroticism, conscientiousness, and family health and aging concerns interact in the prediction of health-related Internet searches in a representative US sample. *Frontiers in psychology*. 2014; 5
- Browne MW, Cudeck R, Bollen KA. Alternative ways of assessing model fit. Sage Focus Editions. 1993; 154:136–136.
- Bunde J, Suls J. A quantitative analysis of the relationship between the Cook-Medley Hostility Scale and traditional coronary artery disease risk factors. *Health Psychology*. 2006; 25(4):493. [PubMed: 16846324]
- Carnethon MR, Jacobs DR, Sidney S, Liu K. Influence of Autonomic nervous system dysfunction on the development of type 2 diabetes the cardia study. *Diabetes care*. 2003; 26(11):3035–3041. [PubMed: 14578236]
- Carnethon MR, Prineas RJ, Temprosa M, Zhang ZM, Uwaifo G, Molitch ME. The association among autonomic nervous system function, incident diabetes, and intervention arm in the Diabetes Prevention Program. *Diabetes Care*. 2006; 29(4):914–919. [PubMed: 16567837]

- Chapman BP, Khan A, Harper M, Stockman D, Fiscella K, Walton J, et al. Gender, race/ethnicity, personality, and interleukin-6 in urban primary care patients. *Brain, behavior, and immunity*. 2009; 23(5):636–642.
- Chapman BP, van Wijngaarden E, Seplaki CL, Talbot N, Duberstein P, Moynihan J. Openness and conscientiousness predict 34-SSweek patterns of Interleukin-6 in older persons. *Brain, behavior, and immunity*. 2011; 25(4):667–673.
- Christie IC, Gianaros PJ. PhysioScripts: An extensible, open source platform for the processing of physiological data. *Behavior research methods*. 2013; 45(1):125–131. [PubMed: 22956357]
- Connelly BS, Ones DS. An other perspective on personality: meta-analytic integration of observers' accuracy and predictive validity. *Psychological Bulletin*. 2010; 136(6):1092. [PubMed: 21038940]
- Costa, PT.; McCrae, RR. Revised neo personality inventory (neo pi-r) and neo five-factor inventory (neo-ffi). Vol. 101. *Psychological Assessment Resources*; Odessa, FL: 1992.
- Courneya KS, Hellsten LAM. Personality correlates of exercise behavior, motives, barriers and preferences: An application of the five-factor model. *Personality and Individual Differences*. 1998; 24(5):625–633.
- Dearborn JL, Knopman D, Sharrett AR, Schneider A, Jack JC, Coker LH, et al. The Metabolic Syndrome and Cognitive Decline in the Atherosclerosis Risk in Communities Study (ARIC). *Dementia and geriatric cognitive disorders*. 2014; 38(5–6):337–346. [PubMed: 25171458]
- Demaree HA, Everhart DE. Healthy high-hostiles: Reduced parasympathetic activity and decreased sympathovagal flexibility during negative emotional processing. *Personality and Individual Differences*. 2004; 36(2):457–469.
- DeYoung CG. Higher-order factors of the Big Five in a multi-informant sample. *Journal of personality and social psychology*. 2006; 91(6):1138. [PubMed: 17144770]
- DeYoung CG, Peterson JB, Higgins DM. Higher-order factors of the Big Five predict conformity: Are there neuroses of health? *Personality and Individual Differences*. 2002; 33(4):533–552.
- DeYoung CG, Quilty LC, Peterson JB. Between facets and domains: 10 aspects of the Big Five. *Journal of personality and social psychology*. 2007; 93(5):880. [PubMed: 17983306]
- Digman JM. Higher-order factors of the Big Five. *Journal of personality and social psychology*. 1997; 73(6):1246. [PubMed: 9418278]
- Feng L, Chong MS, Lim WS, Lee TS, Collinson SL, Yap P, et al. Metabolic syndrome and amnesic mild cognitive impairment: singapore longitudinal ageing study-2 findings. *Journal of Alzheimer's Disease*. 2013; 34(3):649–657.
- Flynn KE, Smith MA, Freese J. When do older adults turn to the internet for health information? Findings from the Wisconsin Longitudinal Study. *Journal of General Internal Medicine*. 2006; 21(12):1295–1301. [PubMed: 16995892]
- Galione, J.; Oltmanns, T. *Personality traits. Multimethod Clinical Assessment*; Guilford; New York: 2014.
- Gami AS, Witt BJ, Howard DE, Erwin PJ, Gami LA, Somers VK, et al. Metabolic Syndrome and Risk of Incident Cardiovascular Events and Death: A Systematic Review and Meta-Analysis of Longitudinal Studies. *Journal of the American College of Cardiology*. 2007; 49(4):403–414. [PubMed: 17258085]
- Graham EK, Lachman ME. Personality stability is associated with better cognitive performance in adulthood: Are the stable more able? *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*. 2012; 67(5):545–554.
- Hampson SE. Personality processes: Mechanisms by which personality traits “get outside the skin”. *Annual review of psychology*. 2012; 63:315.
- Han TS, Sattar N, Williams K, Gonzalez-Villalpando C, Lean ME, Haffner SM. Prospective study of C-reactive protein in relation to the development of diabetes and metabolic syndrome in the Mexico City Diabetes Study. *Diabetes care*. 2002; 25(11):2016–2021. [PubMed: 12401749]
- Hemingway H, Shipley M, Brunner E, Britton A, Malik M, Marmot M. Does autonomic function link social position to coronary risk? The Whitehall II study. *Circulation*. 2005; 111(23):3071–3077. [PubMed: 15939818]

- Hu, Lt; Bentler, PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*. 1999; 6(1):1–55.
- Huang WL, Chang LR, Kuo TB, Lin YH, Chen YZ, Yang CC. Gender differences in personality and heart-rate variability. *Psychiatry research*. 2013; 209(3):652–657. [PubMed: 23499230]
- Huang Y, Liu X. Leisure-time physical activity and the risk of metabolic syndrome: meta-analysis. *European journal of medical research*. 2014; 19(1):22. [PubMed: 24758610]
- Jonassaint CR, Boyle SH, Kuhn CM, Siegler IC, Copeland WE, Williams R. Personality and inflammation: the protective effect of openness to experience. *Ethnicity & disease*. 2010; 20(1):11. [PubMed: 20178176]
- Kotov R, Gamez W, Schmidt F, Watson D. Linking “big” personality traits to anxiety, depressive, and substance use disorders: a meta-analysis. *Psychological bulletin*. 2010; 136(5):768. [PubMed: 20804236]
- Lee YH, Pratley RE. The evolving role of inflammation in obesity and the metabolic syndrome. *Current diabetes reports*. 2005; 5(1):70–75. [PubMed: 15663921]
- Liao D, Cai J, Rosamond WD, Barnes RW, Hutchinson RG, Whitsel EA, et al. Cardiac Autonomic Function and Incident Coronary Heart Disease: A Population-based Case-Cohort Study The ARIC Study. *American Journal of Epidemiology*. 1997; 145(8):696–706. [PubMed: 9125996]
- Luchetti M, Barkley JM, Stephan Y, Terracciano A, Sutin AR. Five-factor model personality traits and inflammatory markers: New data and a meta-analysis. *Psychoneuroendocrinology*. 2014; 50:181–193. [PubMed: 25233337]
- Lunn TE, Nowson CA, Worsley A, Torres SJ. Does personality affect dietary intake? *Nutrition*. 2014; 30(4):403–409. [PubMed: 24607300]
- MacKinnon DP, Fritz MS, Williams J, Lockwood CM. Distribution of the product confidence limits for the indirect effect: Program PRODCLIN. *Behavior Research Methods*. 2007; 39(3):384–389. [PubMed: 17958149]
- Malouff JM, Thorsteinsson EB, Rooke SE, Schutte NS. Alcohol involvement and the five-factor model of personality: A meta-analysis. *Journal of Drug Education*. 2007; 37(3):277–294. [PubMed: 18047183]
- Malouff JM, Thorsteinsson EB, Schutte NS. The five-factor model of personality and smoking: A meta-analysis. *Journal of Drug Education*. 2006; 36(1):47–58. [PubMed: 16981639]
- Manuck SB, Phillips JE, Gianaros PJ, Flory JD, Muldoon MF. Subjective socioeconomic status and presence of the metabolic syndrome in midlife community volunteers. *Psychosomatic medicine*. 2010; 72(1):35–45. [PubMed: 19933505]
- Marques-Vidal P, Mazoyer E, Bongard V, Gourdy P, Ruidavets JB, Drouet L, et al. Prevalence of insulin resistance syndrome in southwestern France and its relationship with inflammatory and hemostatic markers. *Diabetes Care*. 2002; 25(8):1371–1377. [PubMed: 12145237]
- Marsland AL, McCaffery JM, Muldoon MF, Manuck SB. Systemic inflammation and the metabolic syndrome among middle-aged community volunteers. *Metabolism*. 2010; 59(12):1801–1808. [PubMed: 20619428]
- Marsland AL, Prather AA, Petersen KL, Cohen S, Manuck SB. Antagonistic characteristics are positively associated with inflammatory markers independently of trait negative emotionality. *Brain, behavior, and immunity*. 2008; 22(5):753–761.
- McCaffery JM, Marsland AL, Strohacker K, Muldoon MF, Manuck SB. Factor structure underlying components of allostatic load. *PloS one*. 2012; 7(10):e47246. [PubMed: 23112812]
- McCaffery JM, Shen BJ, Muldoon MF, Manuck SB. Ambulatory blood pressure and the metabolic syndrome in normotensive and untreated hypertensive men. *Metabolic syndrome and related disorders*. 2007; 5(1):34–44. [PubMed: 18370812]
- Mohamed-Ali V, Goodrick S, Rawesh A, Katz D, Miles J, Yudkin J, et al. Subcutaneous Adipose Tissue Releases Interleukin-6, But Not Tumor Necrosis Factor- α , in Vivo 1. *The Journal of Clinical Endocrinology & Metabolism*. 1997; 82(12):4196–4200. [PubMed: 9398739]
- Mommersteeg P, POUWER F. Personality as a risk factor for the metabolic syndrome: A systematic review. *Journal of psychosomatic research*. 2012; 73(5):326–333. [PubMed: 23062804]

- Möttus R, McNeill G, Jia X, Craig LC, Starr JM, Deary IJ. The associations between personality, diet and body mass index in older people. *Health Psychology*. 2013; 32(4):353. [PubMed: 21928903]
- Muldoon MF, Nazzaro P, Sutton-Tyrrell K, Manuck SB. White-coat hypertension and carotid artery atherosclerosis: a matching study. *Archives of internal medicine*. 2000; 160(10):1507–1512. [PubMed: 10826466]
- Muthén, LK.; Muthén, BO. *Mplus User's Guid*. 7. Los Angeles, CA: Muthén & Muthén; 1998–2012.
- Ozer D, Benet-Martinez V. Personality and the prediction of consequential outcomes. *Annu Rev Psychol*. 2006; 57:401–421. [PubMed: 16318601]
- Paffenbarger RS, Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. *American Journal of epidemiology*. 1978; 108(3):161–175. [PubMed: 707484]
- Phillips AC, Batty GD, Weiss A, Deary I, Gale CR, Thomas GN, et al. Neuroticism, cognitive ability, and the metabolic syndrome: The Vietnam Experience Study. *Journal of psychosomatic research*. 2010; 69(2):193–201. [PubMed: 20624519]
- Piché M-È, Lemieux S, Weisnagel SJ, Corneau L, Nadeau A, Bergeron J. Relation of high-sensitivity C-reactive protein, interleukin-6, tumor necrosis factor-alpha, and fibrinogen to abdominal adipose tissue, blood pressure, and cholesterol and triglyceride levels in healthy postmenopausal women. *The American journal of cardiology*. 2005; 96(1):92–97. [PubMed: 15979442]
- Poropat AE. A meta-analysis of the five-factor model of personality and academic performance. *Psychological bulletin*. 2009; 135(2):322. [PubMed: 19254083]
- Rana J, Nieuwdorp M, Jukema J, Kastelein J. Cardiovascular metabolic syndrome—an interplay of, obesity, inflammation, diabetes and coronary heart disease. *Diabetes, Obesity and Metabolism*. 2007; 9(3):218–232.
- Reilly MP, Rader DJ. The metabolic syndrome more than the sum of its parts? *Circulation*. 2003; 108(13):1546–1551. [PubMed: 14517150]
- Rennie KL, McCarthy N, Yazdgerdi S, Marmot M, Brunner E. Association of the metabolic syndrome with both vigorous and moderate physical activity. *International journal of epidemiology*. 2003; 32(4):600–606. [PubMed: 12913036]
- Ross K, Martin T, Chen E, Miller GE. Social encounters in daily life and 2-year changes in metabolic risk factors in young women. *Development and psychopathology*. 2011; 23(03):897–906. [PubMed: 21756440]
- Rothmann S, Coetzer E. The big five personality dimensions and job performance. *SA Journal of Industrial Psychology*. 2003; 29(1):68–74.
- Rouch I, Trombert B, Kossowsky MP, Laurent B, Celle S, Ntougou Assoumou G, et al. Metabolic Syndrome is Associated with Poor Memory and Executive Performance in Elderly Community Residents: The PROOF Study. *The American Journal of Geriatric Psychiatry*. 2014
- Shen BJ, Todaro JF, Niaura R, McCaffery JM, Zhang J, Spiro A III, et al. Are metabolic risk factors one unified syndrome? Modeling the structure of the metabolic syndrome X. *American journal of epidemiology*. 2003; 157(8):701–711. [PubMed: 12697574]
- Sloan RP, Shapiro PA, Bigger JT Jr, Bagiella E, Steinman RC, Gorman JM. Cardiac autonomic control and hostility in healthy subjects. *The American journal of cardiology*. 1994; 74(3):298–300. [PubMed: 8037145]
- Smith TW. Personality as risk and resilience in physical health. *Current directions in psychological science*. 2006; 15(5):227–231.
- Smith TW, Glazer K, Ruiz JM, Gallo LC. Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion, and health. *Journal of personality*. 2004; 72(6):1217–1270. [PubMed: 15509282]
- Smith TW, MacKenzie J. Personality and risk of physical illness. *Annu Rev Clin Psychol*. 2006; 2:435–467. [PubMed: 17716078]
- Soares-Miranda L, Sandercock G, Vale S, Santos R, Abreu S, Moreira C, et al. Metabolic syndrome, physical activity and cardiac autonomic function. *Diabetes/metabolism research and reviews*. 2012; 28(4):363–369. [PubMed: 22238216]
- Song S-W, Chung J-H, Rho JS, Lee Y-A, Lim H-K, Kang S-G, et al. Regional cortical thickness and subcortical volume changes in patients with metabolic syndrome. *Brain imaging and behavior*. 2014:1–9. [PubMed: 24078043]

- Stuckey MI, Tulppo MP, Kiviniemi AM, Petrella RJ. Heart rate variability and the metabolic syndrome: a systematic review of the literature. *Diabetes/metabolism research and reviews*. 2014
- Suls J, Bunde J. Anger, anxiety, and depression as risk factors for cardiovascular disease: the problems and implications of overlapping affective dispositions. *Psychological bulletin*. 2005; 131(2):260. [PubMed: 15740422]
- Sutin AR, Costa PT Jr, Uda M, Ferrucci L, Schlessinger D, Terracciano A. Personality and metabolic syndrome. *Age*. 2010; 32(4):513–519. [PubMed: 20567927]
- Sutin AR, Terracciano A, Deiana B, Naitza S, Ferrucci L, Uda M, et al. High neuroticism and low conscientiousness are associated with interleukin-6. *Psychological medicine*. 2010; 40(09):1485–1493. [PubMed: 19995479]
- Suzuki T, Voeks J, Zakai NA, Jenny NS, Brown TM, Safford MM, et al. Metabolic Syndrome, C-reactive Protein, and Mortality in US Blacks and Whites: The Reasons for Geographic and Racial Differences in Stroke (REGARDS) Study. *Diabetes Care*. 2014:DC_132059.
- Tentolouris N, Argyrakopoulou G, Katsilambros N. Perturbed autonomic nervous system function in metabolic syndrome. *Neuromolecular medicine*. 2008; 10(3):169–178. [PubMed: 18224460]
- Terracciano A, Costa PT. Smoking and the Five-Factor Model of personality. *Addiction*. 2004; 99(4):472–481. [PubMed: 15049747]
- Tiainen AMK, Männistö S, Lahti M, Blomstedt PA, Lahti J, Perälä MM, et al. Personality and Dietary Intake—Findings in the Helsinki Birth Cohort Study. *PloS one*. 2013; 8(7):e68284. [PubMed: 23874573]
- Turiano NA, Mroczek DK, Moynihan J, Chapman BP. Big 5 personality traits and interleukin-6: Evidence for “healthy Neuroticism” in a US population sample. *Brain, behavior, and immunity*. 2013; 28:83–89.
- van Reedt Dortland AK, Giltay EJ, Van Veen T, Zitman FG, Penninx BW. Personality traits and childhood trauma as correlates of metabolic risk factors: the Netherlands Study of Depression and Anxiety (NESDA). *Progress in Neuro-Psychopharmacology and Biological Psychiatry*. 2012; 36(1):85–91. [PubMed: 22001949]
- Vazire S, Carlson EN. Self-Knowledge of Personality: Do People Know Themselves? *Social and personality psychology compass*. 2010; 4(8):605–620.
- Wannamethee SG, Lowe G, Shaper AG, Rumley A, Lennon L, Whincup PH. The metabolic syndrome and insulin resistance: relationship to haemostatic and inflammatory markers in older non-diabetic men. *Atherosclerosis*. 2005; 181(1):101–108. [PubMed: 15939060]
- Watkins LL, Grossman P, Krishnan R, Sherwood A. Anxiety and vagal control of heart rate. *Psychosomatic medicine*. 1998; 60(4):498–502. [PubMed: 9710297]

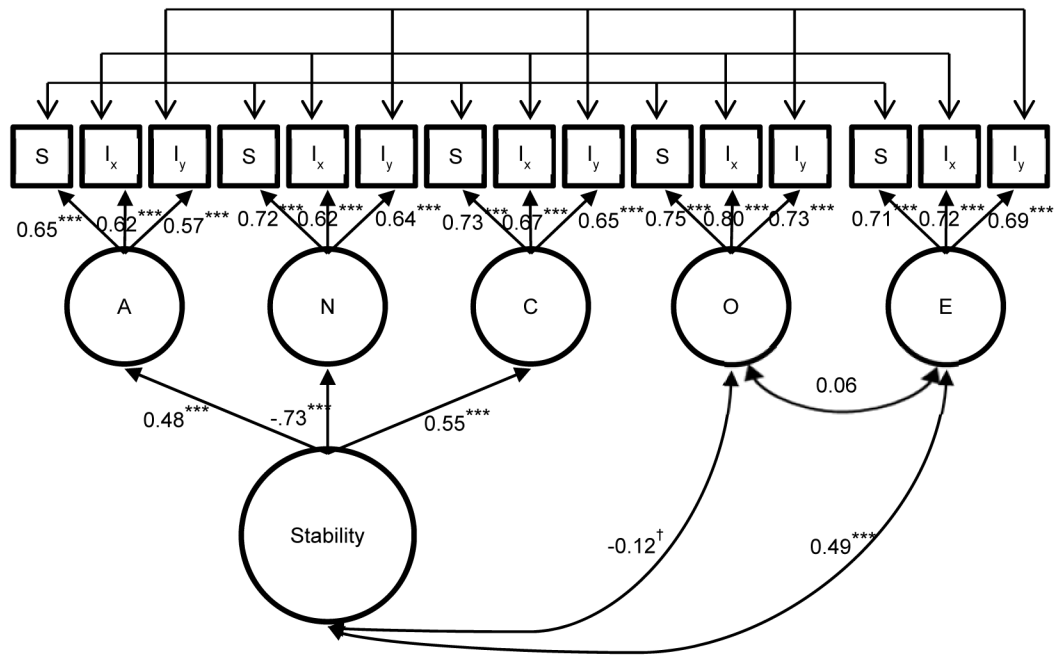


Figure 1.

A Higher-order Stability Factor Underlying Agreeableness, Neuroticism, and Conscientiousness.

Note. Standardized path coefficients are reported. Residual arrows for latent factors are omitted to simplify the figure. The first letter of each personality trait are used as abbreviations. 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

*** $p < .001$, ** $p < .01$, * $p < .05$, [†] $p < .10$

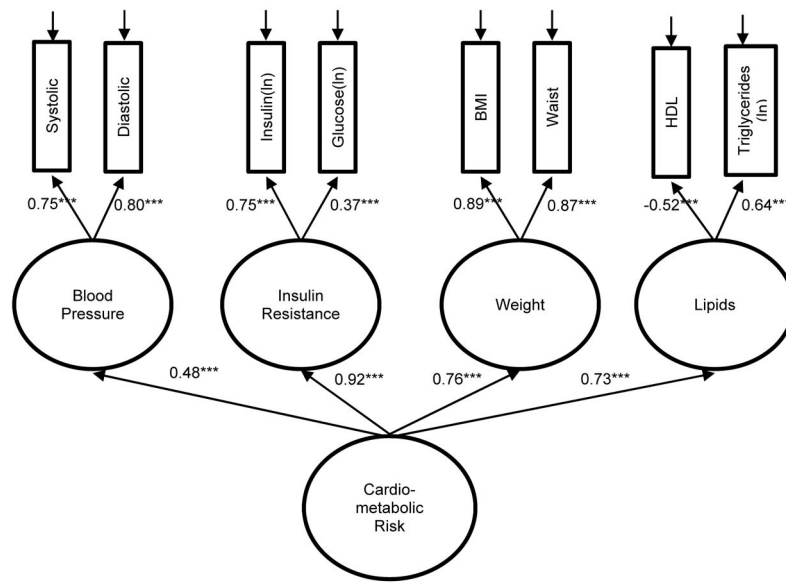
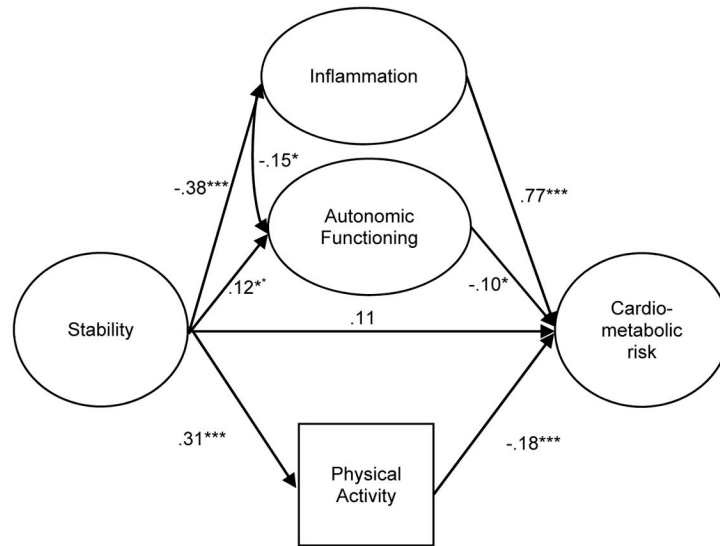


Figure 2.
 The Factor Structure of Cardiometabolic Risk.
 Note. Standardized path coefficients are reported. Residual arrows for latent factors are omitted to simplify the figure.
 Model fit: $\chi^2=34.35$, $df=20$, $p=.02$; RMSEA=.03, 90% CI: .01-.05; SRMR=.02, CFI = .99
 *** $p < .001$, ** $p < .01$, * $p < .05$, † $p < .10$



	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.

Note. 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

*** $p < .001$, ** $p < .01$, * $p < .05$, † $p < .10$

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 (μU/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 (mmHg)	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)		

* Variable is natural log transformed.

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	$\beta = -0.11, p = .02, R^2 = .01$	$\beta = -0.08, p = .12$	} $R^2 = .07$
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 **bolded**.

^aEach personality factor was examined as a predictor of the metabolic syndrome without controlling for the effects of the other 4 personality traits. R^2 is 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 4 personality traits. R^2 is total variance explained by the combination of the personality traits net of the effect of covariates

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript