Published in final edited form as:
Psychosomatics. 2010 ; 51(4): 289–296. doi:10.1176/appi.psy.51.4.289.

Personality Disorders and Traits as Predictors of Incident Cardiovascular Disease: Findings From the 23-Year Follow-Up of The Baltimore ECA Study

Hochang Benjamin Lee, M.D., **O. Joseph Bienvenu, M.D., Ph.D.**, **Seong-Jin Cho, M.D., Ph.D.**, **Christine M. Ramsey**, **B.S. Karen Bandeen-Roche, Ph.D.**, **William W. Eaton, M.D.**, and **Gerald Nestadt, M.D., M.P.H.**

Dept. of Psychiatry & Behavioral Sciences, School of Medicine; the Dept. of Mental Health, Dept. of Statistics, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD; and the Dept. of Psychiatry, Gachon University of Medicine and Science, Incheon, South Korea

Abstract

Background—Over the past several decades, the relationship between personality traits and heart disease has interested clinicians and researchers alike.

Objective—The authors investigated personality disorders (PDs) and PD dimensional traits as prospective risk factors for incident cardiovascular disease (CVD) in the Baltimore Epidemiologic Catchment Area (ECA) follow-up study.

Method—In 1981, 244 community residents were examined for DSM–III PDs, and PD dimensional traits and were followed for incident CVD by 2004.

Results—Logistic-regression models with or without adjustment for potential confounders revealed that Cluster B PD and PD dimensional traits at baseline were consistently associated with increased risk of incident CVD by 2004. Post-hoc analysis also revealed that Cluster B PD and traits also predict CVD mortality.

Conclusion—Cluster B PDs and dimensional traits may be independent risk factors for incident CVD in the community.

Over the past several decades, the relationship between personality traits and heart disease has interested clinicians and researchers alike. Early studies reported an association between Type A personality traits or behavioral patterns: these are characterized by competitiveness, anger, and hostility, and incident cardiovascular disease (CVD);[1–3] but later reports, including two systematic reviews, found inconsistent associations.[4,5] More recently, high scores on negative affectivity and social inhibition personality dimensions, that is, type D, or "distressed" personality traits, have been linked to cardiac disease and mortality.[6]

Send correspondence and reprint requests to Hochang Benjamin Lee, M.D., Assistant Professor of Psychiatry and Behavioral Sciences, The Johns Hopkins University School of Medicine, 4th Floor, 5300 Alpha Commons Drive, Baltimore, MD 21224. Hochang@jhmi.edu.

The Diagnostic and Statistical Manual of Mental Disorders (DSM) categorizes personality disorders (PDs) into three clusters: Cluster A: “odd and eccentric,” Cluster B: “dramatic, irrational or erratic,” and Cluster C: “anxious or fearful.”[7,8] Individuals diagnosed with PDs tend to be found in the extreme of personality dimensions such as neuroticism, introversion, antagonism, and negligence.[9–11] Until recently, few studies have examined the relationship between personality disorders and CVD.[12,13] Using data from 8,580 British adults between ages 16 and 74 years, Moran et al.[12] found that participants with DSM–IV PDs were more likely to report experiencing a stroke (age- and sex-adjusted odds ratios [ORs]: 2.1; 95% confidence interval [CI]: 1.2–3.8) or ischemic heart disease (OR: 1.5 [95% CI]: 1.1–2.1). Similarly, using data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), Pietrzak et al.[13] found that having a DSM–IV PD increased the odds of having coronary heart disease (CHD), after adjusting for putative risk factors (OR: 1.26; CI: 1.01–1.58). However, the cross-sectional design of both studies precluded examination of the temporal or potentially causal relationship between personality disorders and CVD.

Few studies have examined longitudinal outcomes of individuals with PDs,[14] and even fewer studies have specifically examined long-term general health outcomes.[15] The goal of the current study was to examine the longitudinal relationship between DSM–III PDs in 1981 and later incidence of CVD in a 23-year follow-up of community residents who participated in the Hopkins Epidemiology of Personality Disorders Study (HEPS) and the Baltimore Epidemiologic Catchment Area (ECA) Follow-up Study.[16,17] As a secondary aim, we examined whether a specific cluster (A, B, or C) of PD diagnoses or PD dimensional traits was longitudinally associated with incident CVD after adjusting for other putative risk factors. As a post-hoc analysis, we also examined PD diagnoses and dimensional traits as risk factors for mortality due to CHD.

METHOD

Sample

Details of the HEPS and the Baltimore ECA studies have been provided previously.[17] Briefly, a two-stage population survey was conducted at the Johns Hopkins Medical Institutions in 1981 as part of the ECA program of the National Institute of Mental Health (NIMH). After a probability sampling of households in the area, 3,481 residents between the ages of 18 and 64 years were interviewed by specially-trained, non-clinician interviewers at Wave 1 of the Baltimore ECA study. During the second stage, a stratified sample (N=810) was examined by psychiatrists in a “clinical reappraisal.” Of 810 original subjects in 1981, 369 respondents died during the 23-year follow-up period; 115 were lost to follow-up (their whereabouts could not be determined), and 77 declined to participate in Wave 4. Psychiatrist-diagnosed PD in 1981 was not correlated with attrition in the cohort. Overall, 76% of the 326 available participants, or 249 individuals, were re-interviewed during Wave 4. Five participants who had incomplete CVD information at Wave 4 were excluded from the analysis, and the final sample for the current study comprised 244 participants.

Diagnostic Assessment of DSM–III Personality Disorders in 1981

A board-certified or board-eligible psychiatrist examined each participant by use of a semi-structured interview. Two methods were utilized to assess individual items of DSM–III PD psychopathology, as described previously.[17] First, in the inventory method, the DSM–III PD dimensional traits of each subject were rated on a scale, ranging from 0 ("absent") to 3 ("likely to result in distress from even minor life stresses"). A dimensional trait score for each of the 11 DSM–III PDs in three clusters was created by summing the score for all of the traits included in each of the personality scores. The second method required that the examiner ask a series of direct questions about particular characteristics. The psychiatrists based their assessments on historical information provided by the subject and behavior observed during the examination, which took 2 to 3 hours. Diagnoses of specific PDs were generated by algorithms based on DSM–III criteria. A dimensional score for each PD was calculated for each of the 11 DSM–III PDs by summing each constituent item of the specific disorder. The interrater agreement among the four psychiatrists in terms of the total number of DSM–III PD criteria assigned was high (intraclass correlation coefficient: 0.88).[17]

Cardiovascular Health Outcome in 2004

Questions regarding cardiovascular health were administered during each wave of the Baltimore ECA study. In Wave 1 (1981), there was a single question: "Have you ever had heart trouble?" Respondents answering positively to this question were excluded from the analyses, which concentrated on the individuals at risk for new onset of CVD. In Wave 4 (2004), after the respondent was asked whether he or she had ever had "heart trouble," those who responded positively were then asked, "What kind of heart trouble have you had?" The interviewer then read a list of five specific conditions: rheumatic fever, rheumatic heart disease, angina pectoris, congestive heart failure (CHF), and myocardial infarction (MI). During Wave 4, each participant was also asked about past history of angioplasty and coronary artery bypass graft (CABG) surgery. Incident CVD was defined as self-report of "heart attack," CHF, or having angioplasty or CABG surgery by Wave 4. Those who reported "heart trouble" at Wave 1 (N=12) were excluded from analyses, because we were interested in incident CVD between Waves 1 and 4.

For post-hoc analysis, mortality data for all participants (N=810) was obtained via the National Death Index and death certificates. Death from coronary heart disease (CHD) was designated when a death certificate (coded according to the 9th Revision of the International Classification of Diseases) indicated "Ischemic Heart Disease" (Diagnosis Codes: 410 – 414) as an underlying cause of death.

Covariates

In all four waves of the Baltimore ECA study, information was collected about participants' sociodemographic characteristics; history of common general-medical illnesses, including hypertension and diabetes mellitus; use/misuse of tobacco, alcohol, and other substances; and history of other psychiatric disorders, including major depressive disorder, panic disorder, and phobia. Psychiatric diagnoses were made with the Diagnostic Interview Schedule (DIS). The DIS is a highly-structured survey instrument designed to produce diagnoses of specific mental disorders according to the criteria of the DSM–III. A computer

algorithm combined responses into diagnostic categories. Studies of the reliability and validity of the DIS show that it has good reliability and acceptable validity.[18]

Statistical Analysis

For the a priori analyses on the relationship between Cluster B personality disorders and incident CVD, a series of comparisons of sociodemographic, general-medical, and mental health variables between surviving participants, with (N=32) or without (N=212) a DSM–III PD, were conducted with the Mann-Whitney *U* test for continuous variables and chi-square or Fisher's exact tests for categorical variables, as appropriate. We used logistic regression to calculate unadjusted and adjusted ORs with 95% CIs. Diagnoses of PDs and PD dimensional trait scores were the main predictor variables, and incident CVD was the main outcome of interest. We adjusted for factors of age; race; gender; socioeconomic status, as measured by years of education; social support, as measured by marital status at Wave 1; and other health-related variables, such as current cigarette smoking (≥1 pack per day), history of diabetes, and hypertension. Variables were retained in the model if they were significant at the level of <0.10 or if their removal altered the ORs associated with PD or PD dimensional traits by more than 20%. In post-hoc analyses examining the relationship between PDs and CHD mortality, the same analytic procedures were followed, using the entire original cohort (N=810), with CHD mortality as the outcome of interest.

RESULTS

Table 1 shows the distribution of DSM–III PDs and PD dimensional trait scores for the 244 surviving participants. Overall, 32 of 244 subjects were diagnosed with one or more DSM–III PDs. Among the three clusters, Cluster B personality disorders (histrionic personality [N=19]; antisocial personality [N=4]; narcissistic personality [N=0]; and borderline personality [N=4]) were the most common and were diagnosed in 24 participants. Cluster A (paranoid, schizoid, and schizotypal [N=2]) and Cluster C PDs (avoidant, dependent, and compulsive [N=9]) were diagnosed less commonly; three respondents were diagnosed with both Cluster B and Cluster C PDs.

Table 2 shows demographic and clinical information by diagnosis of DSM–III PDs. No baseline differences were detected between the two groups in terms of demographic or cardiovascular variables. As expected, a higher percentage of participants with a diagnosis of DSM–III PDs also had a lifetime history of DSM–III major depressive disorder, panic disorder, or substance abuse/dependence at Wave 1. A higher percentage of subjects with a DSM–III PD (21.8%; 7 of 32) developed incident CVD, as compared with those who had no PD diagnosis (13.9%; 29 of 212), but the difference was not statistically significant.

Table 3 shows the unadjusted ORs for sociodemographic and health- and mental health-related variables as risk factors for incident CVD. Among demographic variables, older age was associated with incident CVD. Among health-related variables, smoking one-or-more packs of cigarettes per day (within the past year) and having diabetes at Wave 1 were associated with incident CVD by Wave 4. DSM–III Axis I diagnoses (including major depressive disorder) at Wave 1 were not associated with incident CVD by Wave 4. Having a

diagnosis of any DSM–III PD was not associated with incident CVD by Wave 4 (OR: 1.74; 95% CI: 0.69 – 4.38).

However, when analyzed separately, DSM–III Cluster B personality disorder (PD) diagnosis at Wave 1 was significantly associated with incident CVD (OR: 2.67; 95% CI: 1.02–6.99). In fact, all of the 7 subjects with comorbid DSM–III PD and incident CVD had a diagnosis of Cluster B personality disorder, and a significantly higher proportion of subjects with cluster B PD than with no PD (29.2% versus 13.4%; p=0.026) had developed incident CVD by Wave 4. The numbers of baseline PD-affected participants were too small to assess Cluster A (N=2) or C (N=9) PDs as risk factors for incident CVD.

The total DSM–III PD dimensional-trait score was not quite statistically significantly associated with incident CVD by Wave 4 (OR: 1.04; 95% CI: 1.00–1.07; p=0.058). However, the Cluster B PD dimensional-trait score was itself associated with a higher odds ratio for incident CVD by Wave 4 (OR: 1.05 for a single-point higher Cluster B score, 95% CI: 1.01–1.10), whereas Cluster A and C dimensional-trait scores were not.

Table 4 shows the adjusted models. Model 1 is adjusted for pertinent demographic variables (age, race, and education), and Model 2 is adjusted for health-related variables (diabetes and smoking more than 1 pack per day). Model 3 is adjusted for age, diabetes, and smoking, which were significantly associated with incident CVD in the univariate analyses (Table 3). We did not find an association between a diagnosis of any DSM–III PD and incident CVD, but the total PD dimensional-trait score was predictive of incident CVD in the adjusted models. More specifically, Cluster B PDs and PD dimensional-trait scores, but not Cluster A and C PD dimensional-trait scores, consistently increased the odds of incident CVD by Wave 4 in the adjusted models. The addition of any DSM–III Axis I psychiatric disorder, such as major depression, phobia, panic disorder, substance abuse/dependence, and alcohol abuse/dependence at Wave 1, did not influence the models significantly.

Table 5 shows the results of the post-hoc analyses on PD diagnoses and dimensional traits as risk factors for CHD mortality. Of 810 original participants in 1981 (Cluster A PD: N=3; Cluster B PD: N=66; Cluster C PD: N=30), 41 participants, or 5.1%, died of CHD during the subsequent 23 years. Having a diagnosis of any DSM–III PD was longitudinally associated with CHD mortality in the final adjusted model (OR: 3.18; CI: 1.12–8.15), but this association was primarily driven by the strength of the association between Cluster B PDs and CHD mortality (OR: 6.13; 95% CI: 2.14–18.78; p=0.001). Having a Cluster A or C PD diagnosis was not associated with CHD mortality (OR: 0.59; 95% CI: 0.07–4.75; p=0.641). Similarly, Cluster B PD dimensional trait scores increased the odds of CHD mortality in the final adjusted model (OR: 1.09; 95% CI: 1.03–1.16, p=0.008), whereas Cluster A and C PD dimensional-trait scores did not.

DISCUSSION

The results of this study support previous reports of a cross-sectional association between personality disorders (PDs) and cardiovascular disease (CVD) and extend this area of research by focusing on the longitudinal association between (Cluster B) PDs and incident

CVD among community-dwelling persons. The association between Cluster B PD dimensional-traits at Wave 1 and incident CVD by Wave 4 suggests a dose-dependent relationship between Cluster B personality traits and incident CVD. These findings are also supported by the post-hoc analyses that confirmed the longitudinal relationship between Cluster B PDs and CHD mortality.

The reason for this longitudinal relationship may be multifactorial. Behavioral aspects of Cluster B PDs could interfere with proper management of chronic medical conditions that are known cardiovascular risk factors; these include hypertension, hypercholesterolemia, and diabetes. Compliance is a crucial determinant of the treatment success of any medical condition. Stresses related to symptoms and management of chronic medical conditions include loss of well-being, financial strains, change in self-image, loss of autonomy, and complicated interactions with healthcare providers. Poor coping skills related to Cluster B personality traits could hinder compliance with complex regimens necessary for proper management of chronic medical conditions.

An emotionally stressful event, such as an event arousing anger, could possibly trigger an acute coronary syndrome or MI and could have implications on the relationship between Cluster B PD (e.g., borderline personality disorder) traits and CVD. According to DSM–IV, frequent and uncontrollable anger is a hallmark of borderline personality disorder.[8] Both the Determinants of Myocardial Infarction Onset Study and the Stockholm Heart Epidemiology study have confirmed that acute episodes of anger could trigger MI.[19,20] Frequent and unregulated episodes of anger in borderline PD patients, especially those with cardiovascular risk factors, could render them vulnerable to incident CVD.

Another possible explanation for the increased risk for CVD in persons with Cluster B PDs is comorbidity with other psychiatric disorders. Recent longitudinal studies have shown that Cluster B PDs are associated with a higher risk of developing depressive and anxiety disorders.[21–23] Numerous studies have established that depressive symptoms or episodes increase risk of incident cardiovascular disease in community-dwellers.[24] Anxiety disorders also appear to increase risk for coronary heart disease risk, independently of major depressive disorder.[25,26] Also, high levels of substance abuse among those with Cluster B personality disorders, particularly antisocial personality disorder, could lead to a multitude of medical problems, including cardiovascular disease. In our analyses, baseline diagnoses of mood, anxiety, and substance-use disorders did not appear to explain the longitudinal relationship between cluster B PDs and incident CVD. However, we cannot rule out the possibility that later-onset Axis I conditions could mediate the relationship between Cluster B PDs and CVD to some extent.

Among Cluster B PD dimensional traits, borderline traits (OR: 1.23; CI: 1.01–1.51) were significantly associated with incident CVD. This finding is consistent with the previous report by Moran and colleagues[12] that, among DSM–IV PDs, borderline PD was the strongest correlate of ischemic heart disease (OR: 7.2; 95% CI: 2.1–24.3). Importantly, a recent report found that, among 51 young women with Type 1 diabetes, borderline personality traits were associated with irregular insulin use, binge eating, and poor glycemic control.[27] These poor health-related choices could lead to exacerbation of chronic medical

conditions such diabetes and subsequent cardiovascular complications. Also, a 6-year follow up study of patients with remitted or unremitted borderline personality disorder found that remitted patients were more likely to make healthy lifestyle choices.[28] Remitted patients were less likely to smoke cigarettes, drink alcohol every day, and become obese, and more likely to exercise than unremitted patients. These findings suggest that identification and treatment of vulnerable Cluster B PD traits could lead to improved long-term health outcomes.

Physiological pathways linking borderline PD and incident CVD are plausible, as well. Various physiological mechanisms (e.g., cortisol response and inflammatory markers) in relation to negative affectivity and stress-proneness have been proposed to explain the association between Type D personality and CVD.[29,30] Persons with PDs, especially borderline PD, are similarly vulnerable to significant stress in social, occupational, or other important areas of functioning.[8] Although the results have been mixed, borderline PD, with or without posttraumatic stress syndrome, has been associated with dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis.[27,31,32] HPA-axis dysregulation has a negative impact on established cardiovascular-disease risk factors,[33] and it is associated with early atherosclerosis.[34] A recent study suggests that HPA-axis dysregulation is a mediating factor between depression and increased risk of cardiovascular death.[35] In our results suggesting a relationship between borderline PD and CVD, HPA-axis dysregulation may have a similar mediating role.

This study has several limitations. The most important limitation is the small cell sizes in analyses of Clusters A and C PDs, as risk factors for incident CVD or CHD mortality. Although we could examine the relationship between Cluster B PDs and incident CVD, we could not conclusively analyze the relationship between Clusters A or C PDs and incident CVD. Certainly, a cohort study of a large, population-based sample (e.g., the NESARC study) could further clarify the relationship between personality disorder and incident CVD. Another limitation is the fact that we relied on subjects' self-reports of incident CVD, and subjects with PDs might be less reliable in their report about their health status.

Nevertheless, strengths of this study include the longitudinal, community-based cohort design and assessments by experienced clinicians. Also, the longitudinal nature of the cohort analysis establishes the temporal relationship between Cluster B personality disorder/traits and incident CVD. Future studies should examine behavioral and pathophysiological mechanisms underlying the association between Cluster B personality disorders and incident CVD.

Acknowledgments

This research was supported by grants MH68793 (Lee), K23 MH64543 (Bienvenu), MH50616 (Nestadt), and MH47447 (Eaton) from the National Institute of Mental Health.

References

1. Friedman M, Rosenman RH. Association of specific overt behavior pattern with blood and cardiovascular findings; blood cholesterol level, blood clotting time, incidence of arcus senilis, and clinical coronary artery disease. JAMA. 1959; 169:1286–1296.

2. Rosenman RH, Brand RJ, Wurm M, et al. Coronary heart disease in western collaborative group study: final follow-up experience of 81/2 years. JAMA. 1975; 233:872–877. [PubMed: 1173896]
3. Dimsdale JE. A perspective on Type A behavior and coronary disease. N Engl J Med. 1988; 318:110–112. [PubMed: 3336390]
4. Kuper H, Marmot M, Hemingway H. Systematic review of prospective cohort studies of psychosocial factors in the etiology and prognosis of coronary heart disease. Semin Vasc Med. 2002; 2:267–314. [PubMed: 16222620]
5. Myrtek M. Meta-analyses of prospective studies on coronary heart disease, Type A personality, and hostility. Int J Cardiol. 2001; 79:245–251. [PubMed: 11461748]
6. Denollet J, Sys SU, Brutsaert DL, et al. Personality as independent predictor of long-term mortality in patients with coronary heart disease. Lancet. 1996; 347:417–421. [PubMed: 8618481]
7. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 3. Washington, DC: American Psychiatric Association; 1980.
8. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4. Washington, DC: American Psychiatric Publishing; 1994.
9. Mulder RT, Joyce PR. Temperament and the structure of personality disorder symptoms. Psychol Med. 1997; 27:99–106. [PubMed: 9122314]
10. Deary IJ, Peter A, Gibson G, et al. Personality traits and personality disorders. Br J Psychol. 1998; 89(Pt 4):647–661. [PubMed: 9854807]
11. Saulsman LM, Page AC. The five-factor model and personality disorder empirical literature: a meta-analytic review. Clin Psychol Rev. 2004; 23:1055–1085. [PubMed: 14729423]
12. Moran P, Stewart R, Coid JW, et al. Personality disorder and cardiovascular disease: results from a national household survey. J Clin Psychiatry. 2007; 68:69–74. [PubMed: 17284132]
13. Pietrzak RH, Wagner JA, Petry NM. DSM–IV personality disorders and coronary heart disease in older adults: results from The National Epidemiologic Survey on Alcohol and Related Conditions. J Gerontol B Psychol Sci Soc Sci. 2007; 62:295–299.
14. Goldstein RB, Dawson DA, Grant BF, et al. Antisocial behavioral syndromes and past-year physical health among adults in the United States: results from The National Epidemiologic Survey on Alcohol and Related Conditions. J Clin Psychiatry. 2008; 69:368–380. [PubMed: 18348594]
15. Skodol AE. Longitudinal course and outcome of personality disorders. Psychiatr Clin North Am. 2008; 31:495–503. viii. [PubMed: 18638648]
16. Eaton WW, Shao H, Zandi P, et al. Population-based study of first onset and chronicity in major depressive disorder. Arch Gen Psychiatry. 2008; 65:513–520. [PubMed: 18458203]
17. Samuels JF, Nestadt G, McHugh PR, et al. DSM–III personality disorders in the community. Am J Psychiatry. 1994; 151:1055–1062. [PubMed: 8010364]
18. Regier DA, Myers JK, Locke BZ, et al. The NIMH Epidemiologic Catchment Area Program: historical context, major objectives, and study population characteristics. Arch Gen Psychiatry. 1984; 41:934–941. [PubMed: 6089692]
19. Mittleman MA, Maclure M, Muller JE, et al. Educational attainment, anger, and the risk of triggering myocardial infarction onset: the determinants of myocardial infarction onset study investigators. Arch Intern Med. 1997; 157:769–775. [PubMed: 9125009]
20. Moller J, Hallqvist J, Ahlbom A, et al. Do episodes of anger trigger myocardial infarction? a case-crossover analysis in the Stockholm HEart Epidemiology Program (SHEEP). Psychosom Med. 1999; 61:842–849. [PubMed: 10593637]
21. Gunderson JG, Stout RL, Skodol AE, et al. New episodes and new onsets of major depression in borderline and other personality disorders. J Affect Disord. 2008; 111:40– 45. [PubMed: 18358539]
22. Johnson JG, Cohen P, Kasen S, et al. Personality disorders evident by early adulthood and risk for anxiety disorders during middle adulthood. J Anxiety Disord. 2006; 20:408– 426. [PubMed: 16054332]
23. Johnson JG, Williams JB, Gorman JM, et al. Personality disorders predict onset of Axis I disorders and impaired functioning among homosexual men with and at risk of HIV infection. Arch Gen Psychiatry. 1996; 53:350–357. [PubMed: 8634013]

24. Rugulies R. Depression as a predictor for coronary heart disease. a review and meta-analysis. Am J Prev Med. 2002; 23:51–61. [PubMed: 12093424]
25. Kubzansky LD, Kawachi I, Sparrow D, et al. Anxiety and coronary heart disease: a synthesis of epidemiological, psychological, and experimental evidence. Ann Behav Med. 1998; 20:47–58. [PubMed: 9989308]
26. Barger SD, Sydeman SJ. Does generalized anxiety disorder predict coronary heart disease risk factors independently of major depressive disorder? J Affect Disord. 2005; 88:87–91. [PubMed: 16009431]
27. Lieb K, Rexhausen JE, Bohus M, et al. Increased diurnal salivary cortisol in women with borderline personality disorder. J Psychiatr Res. 2004; 38:559–565. [PubMed: 15458851]
28. Frankenburg FR, Zanarini MC. The association between borderline personality disorder and chronic medical illnesses, poor health-related lifestyle choices, and costly forms of healthcare utilization. J Clin Psychiatry. 2004; 65:1660–1665. [PubMed: 15641871]
29. Pedersen SS, Denollet J. Type D personality, cardiac events, and impaired quality of life: a review. Eur J Cardiovasc Prev Rehabil. 2003; 10:241–248. [PubMed: 14555878]
30. Sher L. Type D personality, the heart, stress, and cortisol. QJM. 2005; 98:323–329. [PubMed: 15820973]
31. Carrasco JL, Diaz-Marsa M, Lopez-Ibor JJ, et al. Hypothalamic-pituitary-adrenal axis response in borderline personality disorder without post-traumatic features. Br J Psychiatry. 2007; 190:357–358. [PubMed: 17401044]
32. Rinne T, de Kloet ER, van den Brink W, et al. Hyperresponsiveness of hypothalamic-pituitary-adrenal axis to combined dexa-methasone/corticotropin-releasing hormone challenge in female borderline personality disorder subjects with a history of sustained childhood abuse. Biol Psychiatry. 2002; 52:1102–1112. [PubMed: 12460693]
33. Rosmond R, Bjorntorp P. Hypothalamic-pituitary-adrenal axis activity as a predictor of cardiovascular disease, type 2 diabetes, and stroke. J Intern Med. 2000; 247:188–197. [PubMed: 10692081]
34. Troxler RG, Sprague EA, Thompson AJ, et al. The association of elevated plasma cortisol and early atherosclerosis as demonstrated by coronary angiography. Atherosclerosis. 1977; 26:151–162. [PubMed: 836352]
35. Jokinen J, Nordström P. HPA-axis hyperactivity and cardiovascular mortality in mood-disorder inpatients. J Affect Disord. 2009; 116:88–92. [PubMed: 19054568]

TABLE 1

Distribution of Personality Disorders and Dimensional Trait Scores in the Sample (N = 244)

Personality Disorders		Dimensional Trait Scores			
	N (%)	Mean	SD	Median	Range
Cluster A	2 (0.88)	0.67	2.35	0	(0–17)
Paranoid	0 (0)	0.34	1.29	0	(0–11)
Schizoid	2 (0.88)	0.14	0.56	0	(0–5)
Schizotypal	0 (0)	0.20	0.95	0	(0–8)
Cluster B	24 (10.0)	5.22	7.48	2	(0–44)
Antisocial	4 (1.7)	1.64	3.81	0	(0–30)
Borderline	4 (1.7)	0.54	1.51	0	(0–10)
Histrionic	19 (10.9)	2.90	4.07	1	(0–17)
Narcissistic	0 (0)	0.15	0.64	0	(0–6)
Cluster C	9 (3.7)	2.29	2.96	1	(0–16)
Compulsive	8 (3.3)	1.50	1.91	1	(0–12)
Dependent	1 (0.4)	0.25	0.68	0	(0–4)
Avoidant	0 (0)	0.39	1.15	0	(0–8)
Passive-Aggressive	1 (0.4)	0.14	0.64	0	(0–6)
Any (Cluster A, B or C)	32 (13.3)	8.01	1.29	4	(0–47)

SD: standard deviation.

TABLE 2

Characteristics at Wave 1 and Incident CVD by Wave 4 by PD Diagnosis

Variables	PD (N = 32) Mean (SD)	No PD (N = 212) Mean (SD)	p	Total (N = 244) N (%)
Sociodemographics at Wave 1				
Age	33.3 (10.7)	34.8 (12.1)	0.445	34.4 (11.9)
Education	13.1 (2.6)	12.25 (3.0)	0.142	12.4 (3.0)
	N (%)	N (%)	p-value	N (%)
Gender			0.839	
Female	23 (71.9)	146 (68.9)		169 (69.3)
Male	9 (28.1)	66 (31.1)		75 (30.7)
Marital Status			0.997	
married	16 (50.0)	100 (49.3)		116 (49.2)
widowed	4 (12.5)	27 (13.3)		31 (12.9)
separated/divorced	8 (25.0)	48 (23.6)		56 (24.2)
never married	4 (12.5)	28 (13.8)		32 (13.3)
Race			0.739	
White	21 (65.6)	126 (59.4)		147 (60.2)
Black	10 (31.2)	81 (38.2)		91 (37.3)
Other	1 (3.1)	5 (2.4)		6 (2.4)
Health Variables at Wave 1				
“Heart Trouble”	1 (3.2)	11 (5.2)	1.000	12 (5.0)
Stroke	0 (0.0)	3 (1.4)	1.000	3 (1.2)
Hypertension	4 (12.9)	24 (23.2)	0.248	53 (21.9)
Diabetes	0 (0.0)	10 (4.7)	0.369	11 (4.1)
Smoking 1+ pack/day	9 (29.0)	63 (29.9)	0.579	72 (29.8)
DSM III Diagnosis at Wave 1				
Alcohol abuse or dependece	7 (22.6)	33 (15.6)	0.311	40 (16.5)
Drug abuse or dependence	9 (29.0)	19 (19.9)	**0.003**	28 (11.7)
Major Depressive Disorder	9 (29.0)	27 (12.8)	**0.028**	36 (14.9)
Generalized Anxiety Diosrder	2 (8.3)	11 (5.0)	0.372	13 (5.3)
Panic Disorder	5 (16.1)	9 (4.3)	**0.022**	14 (5.8)
Social Phobia	0 (0)	6 (2.7)	0.413	6 (2.5)
Incident CVD by Wave IV	7 (21.8)	29 (13.9)	0.242	36 (15.4)
Myocardial Infarction	4 (12.5)	17 (8.1)	0.495	22 (9.1)
Congestive Heart Failure	2 (6.3)	11 (5.1)	0.783	13 (5.2)
Angioplasty	3 (9.4)	17 (8.1)	0.833	20 (8.2)
CABG surgery	0 (0)	5 (2.3)	0.629	5 (2.0)

PD: personality disorder; CVD: cardiovascular disease; CABG: coronary artery bypass graft; SD: standard deviation.

p <0.05 bolded.

TABLE 3

Results of Univariate Logistic Regression: Correlates of Incident CVD by Wave 4

Variables	OR (95% Confidence Interval)	p
Demographic Variables		
Age (in years)	**1.04 (1.01, 1.07)**	**0.008**
Gender (female)	0.67 (0.32, 1.40)	0.290
Race (white)	0.46 (0.21, 1.03)	0.059
Marital status (married)	1.04 (0.51, 2.10)	0.923
Education (grade)	0.89 (0.80, 1.00)	0.054
Cardiovascular Risk Factors		
Smoking 1+ pack at wave 1	**2.43 (1.18, 5.00)**	**0.016**
Diabetes at wave 1	**6.55 (1.79, 23.91)**	**0.004**
Hypertension at Wave 1	1.31 (0.58, 3.10)	0.513
Axis 1 Diagnosis at Wave 1		
Alcohol abuse/dependence	0.98 (0.38, 2.55)	0.981
Illcit drug abuse/dependece	0.94 (0.31, 2.90)	0.918
Major Depression	1.43 (0.58, 3.56)	0.440
Generalized Anxiety Disorder	1.65 (0.44, 6.21)	0.463
Social Phobia	1.17 (0.13, 10.33)	0.887
Panic Disorder	0.96 (0.21, 4.46)	0.954
Axis 2 Diagnosis at Wave 2		
Any PD Diagnosis	1.74 (0.69, 4.38)	0.242
Cluster B PD Diagnosis	**2.67 (1.02, 6.99)**	**0.046**
Any PD Traits	1.04 (1.00, 1.07)	0.058
Cluster A Traits	0.98 (0.84, 1.16)	0.844
Cluster B Traits	**1.05 (1.01, 1.10)**	**0.016**
Cluster C Traits	1.08 (0.97, 1.20)	0.180

PD: Personality Disorder; CVD: Cardiovascular Disease

$p<0.05$ is bolded

TABLE 4

Personality Disorder/Traits and Incident CVD: Adjusted Odds Ratios with 95% CI

Predictor	Model 1[a]	Model 2[b]	Model 3[c]
Any Personality Disorder	1.99 (0.76, 5.19)	2.17 (0.83, 5.67)	2.30 (0.86, 2.14)
Cluster B Personality Disorder	**3.15 (1.15, 8.70)**	**3.26 (1.20, 8.87)**	**4.14 (1.41, 12.14)**
Any PD Dimensional Traits	**1.04 (1.00, 1.08)**	**1.04 (1.00, 1.08)**	**1.05 (1.01, 1.10)**
Cluster A Dimesional Traits	1.00 (0.85, 1.17)	1.01 (0.86, 1.02)	1.03 (0.87, 1.22)
Cluster B Dimensional Traits	**1.05 (1.01, 1.10)**	**1.06 (1.01, 1.11)**	**1.07 (1.02, 1.12)**
Antisocial PD traits	**1.09 (1.01, 1.18)**	1.07 (0.99, 1.16)	1.09 (1.01, 1.19)
Borderline PD traits	1.20 (0.99, 1.46)	**1.21 (1.00, 1.51)**	**1.23 (1.01, 1.51)**
Histrionic PD traits	1.08 (0.98, 1.19)	1.10 (1.00, 1.20)	1.08 (0.99, 1.20)
Narcissistic PD traits	1.20 (0.73, 1.99)	1.37 (0.75, 2.47)	1.81 (0.94, 3.51)
Cluster C Dimensional Traits	1.06 (0.94, 1.20)	1.11 (0.98, 1.14)	1.10 (0.97, 1.24)

CI: confidence interval.

[a] adjusted for demographic factors (age, gender, race, education, and marital status)

[b] adjusted for health factors (body-mass index, hypertension, diabtes and smoking 1+ pack/day)

[c] adjusted for pertinent demographic and health factors (age, diabetes and smoking 1+ pack/day)

p<0.05 is bolded.

TABLE 5

Personality Disorder/Traits and Coronary Heart Disease Mortality: Adjusted Odds Ratios with 95% CI

Predictor	Model 1[a]	Model 2[b]	Model 3[c]
Any Personality Disorder	**3.22 (1.25, 8.31)**	1.84 (0.77, 4.39)	**3.18 (1.22, 8.15)**
Cluster A or C Personality Disorder	0.61 (0.07, 4.87)	0.62 (0.08, 4.71)	0.59 (0.07, 4.75)
Cluster B Personality Disorder	**6.85 (2.28, 20.47)**	2.51 (0.98, 6.44)	**6.13 (2.14, 18.78)**
Any PD Dimensional Traits	1.04 (0.98, 1.09)	0.99 (0.93, 1.04)	1.05 (0.99, 1.11)
Cluster A Dimesional Traits	0.91 (0.69, 1.20)	0.89 (0.68, 1.16)	0.93 (0.70,1.23)
Cluster B Dimensional Traits	**1.07 (1.01, 1.13)**	1.01 (0.95, 1.07)	**1.09 (1.03, 1.16)**
Antisocial PD traits	1.07 (0.98, 1.16)	1.00 (0.91, 1.09)	1.08 (0.99, 1.18)
Borderline PD traits	1.20 (0.93, 1.56)	0.96 (0.73, 1.26)	1.16 (0.89, 1.51)
Histrionic PD traits	**1.17 (1.06, 1.29)**	1.05 (0.95, 1.15)	**1.15 (1.04, 1.27)**
Narcissistic PD traits	1.11 (0.68, 1.78)	0.99 (0.70, 2.11)	1.13 (0.65, 1.94)
Cluster C Dimensional Traits	0.99 (0.84, 1.17)	0.95 (0.81, 1.12)	0.99 (0.83, 1.17)

CI: confidence interval.

[a] adjusted for demographic factors (age, gender, race, education, and marital status)

[b] adjusted for health factors (body-mass index, hypertension, diabtes and smoking 1+ pack/day)

[c] adjusted for pertinent demographic and health factors (age, diabetes, hypertension and smoking 1+ pack/day)

p<0.05 is bolded.