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Published in:
Journal of Psychosomatic Research

Publication date:
2007

Document Version
Publisher's PDF, also known as Version of record

Link to publication in Tilburg University Research Portal

Citation for published version (APA):
Short communication

Type-D personality is a stable taxonomy in post-MI patients over an 18-month period

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Received 1 December 2006; received in revised form 4 June 2007; accepted 5 June 2007

Abstract

Objective: Type-D personality comprises a risk factor for adverse prognosis in patients with cardiovascular disease (CVD). However, concerns that type-D personality may not be a stable personality taxonomy and that progression of CVD may contribute to the manifestation of type-D personality have been voiced. The present study examined the stability of type-D personality in patients with acute myocardial infarction (MI) and evaluated the influence of demographic and clinical risk factors and mood status on the stability of type-D personality during the course of 18 months. Methods: Patients hospitalized for acute MI (N=475) were assessed on demographic and clinical variables, type-D personality, depression, and anxiety at three time points, using both self-report measures and diagnostic interviews. Longitudinal hierarchical latent class regression models were used to examine the stability of type-D personality and the influence of potential confounders. Results: Type-D personality was a stable construct. Multivariate analysis showed that demographic and clinical characteristics, time (P=.11), and intraindividual variability in depressive (P=.19) and anxiety (P=.18) symptoms over time did not affect type-D status. The mean levels of depressive (P=.05) and anxiety (P<.0001) symptoms within a subject over time were significantly related to type-D status. Conclusion: Type-D personality is a stable taxonomy over an 18-month period in post-MI patients. Type-D classification was not confounded by variability in mood status and by disease severity. These findings support the importance of including personality variables in cardiovascular research and the need for intervention trials targeting this personality taxonomy in order to enhance secondary prevention in CVD patients.

Keywords: Anxiety; Depression; Heart disease; Myocardial infarction; Type-D personality

Introduction

Accumulating evidence indicates that the distressed personality, type-D, comprises a risk factor for adverse prognosis [1–4], impaired health status [5–7], and increased emotional distress in patients with cardiovascular disease (CVD) [8–10] (see Pedersen and Denollet [11,12] for a more extensive review). The type-D construct was delineated according to existing personality theory and the notion that the interaction of specific traits may have deleterious effects on health [13]. type-D personality combines two familiar constructs in personality research: negative affectivity (NA) and social inhibition (SI) (e.g., Denollet et al. [1,2]), which are both viewed as global traits and reflect consistencies in the general affective level and behavior of individuals. Good test–retest reliability of type-D status was shown over a 3-month period [14], and a recent prospective study of percutaneous coronary intervention (PCI) patients suggested that type-D personality may exert a stable effect on health status over a 6-month period [15].

However, no prior study has tested the long-term stability of the type-D construct, and concerns that an ischemic event...
or even progression of CVD may contribute to the manifestation of type-D personality have been voiced. Therefore, the current study examined the stability of type-D personality over the course of 18 months in patients with acute myocardial infarction (MI), with type-D personality being assessed at three time points. It was further tested whether demographic and clinical risk factors and variability in symptoms of depression and anxiety influenced the long-term stability of type-D status.

**Methods**

**Patient population**

Patients hospitalized for acute MI (N=475, of which 371 were male) in four teaching hospitals south of The Netherlands were included. Additional information on the patient population has been reported previously [16].

The study was approved by the medical ethics committees of the participating hospitals. The study was conducted in accordance with the Helsinki Declaration, and all patients provided written informed consent.

**Measures**

**Demographic and clinical characteristics**

Demographic variables included gender and age. Clinical variables were obtained from patients’ medical records and included comorbidity (defined as arthritis, renal insufficiency, and chronic obstructive pulmonary disease), cardiac history (defined as MI, angina, PCI, or coronary artery bypass graft (CABG) surgery prior to MI), multivessel disease, diabetes mellitus, PCI versus conservative treatment, anterior MI location, participation in cardiac rehabilitation, smoking status (self-report), body mass index (BMI), hypertension, hypercholesterolemia, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol levels, LDL/HDL ratio, and medication [(β-blockers, angiotensin-converting enzyme (ACE) inhibitors, Ca antagonists, anticoagulants, statins, diuretics, A2 antagonists, vasodilators, aspirin, and psychotropics].

**Type-D personality**

The 14-item type-D Scale (DS14), which assesses type-D personality [14], was administered during hospitalization and at 12 and 18 months post-MI. Items are answered on a 5-point Likert scale from 0 to 4. The scale consists of seven-item subscales: NA and SI. NA and SI correlate highly with the neuroticism and extraversion scales of the NEO Five-Factor Inventory [17] in healthy subjects \( r = .68/-.59\) and in patients with cardiac conditions \( r = .68/-.65\), respectively [14,18]. Only patients scoring high on both subscales according to a standardized cutoff of ≥10 are categorized as type-D personality [14]. The DS14 is a valid and reliable scale, with Cronbach’s \( \alpha \) of .88/.86 and a test–retest reliability of \( r = .72/.82\) for the NA and SI subscales over a 3-month period [14].

**Self-reported symptoms of depression and anxiety**

On the same survey occasions, symptoms of depression and anxiety were assessed using two self-report questionnaires. The Beck Depression Inventory (BDI) assessed the presence and severity of depressive symptoms [19], while the State–Trait Anxiety Inventory (STAI) measured the level of general state and trait anxiety [20]. In the current study, we only included the state scale of the STAI.

**Clinical diagnosis of depressive and anxiety disorders**

The Composite International Diagnostic Interview [21] was used to assess the lifetime diagnoses of (major) depressive and anxiety disorders (consisting of panic disorder, social phobia, and/or generalized anxiety disorder) based on the criteria listed in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* [22]. A trained psychologist conducted the interview 2 months post-MI.

**Statistical analysis**

**Confounders**

* t tests and cross-tabulations (SPSS 14.0; SPSS Inc., Chicago, IL, USA) were used to evaluate demographic, medical, and psychological variables as possible confounders for type-D status during hospitalization and at 12 and 18 months post-MI. Given that we were interested in examining the temporal stability of type-D personality within patients, we opted to preprocess time-varying BDI and STAI scores by centering them across measurement occasions per subject and per variable. This procedure provided two variables: one representing the average level of BDI or STAI scores within a patient over time (grand mean), and the other representing intraindividual variability in depressive or anxiety symptoms over time, in which interindividual differences in the general level are eliminated. Variables that were significant at \( P < .01 \) on univariate analysis were evaluated in a final analysis by entering them simultaneously into a multivariate logistic regression model for all three measurement occasions. Significant confounders \( (P < .5) \) were added as predictors of latent class (LC) regression models for type-D personality.

**Hierarchical LC regression**

Six longitudinal hierarchical LC regression models (Latent GOLD) [23] were fitted. Forty-two percent of patients included at baseline had complete data. A further 21% had only one missing measurement. Missing measurements mostly reflected the number of patients who had not yet had their 1-year and/or 1.5-year follow-up. Missing values, therefore, were missing at random. Latent GOLD allows for the inclusion of all patients on all measurement occasions irrespective of loss to follow-up when missing...
values are missing at random. Type-D personality was considered a binomial count variable.

First, confirmation was sought for the assumption that regression models would consist of two classes due to the dichotomous nature of type-D personality. To this end, the relative fit of a one-class regression model and a two-class regression model was judged. In the best-fitting model at this stage, it was tested whether the intercept was significantly different for the classes in the model and whether time affected the classes differently.

Subsequently, the effects of confounding variables on type-D stability were examined. In a final step, it was tested whether the effects of the confounding variables were similar for both classes. Likelihood ratio tests determined whether a model was a significant improvement compared to the former one that contained fewer classes or fewer restrictions (in a likelihood ratio test, −2LL of nested models is subtracted). This difference is indicated in the table by Δχ². When the increase in Δχ² is not significant (P>.05) with respect to gained degrees of freedom (df), the more restrictive model fits the observed data best. In addition, the Bayesian Information Criterion (BIC) [24], indicating the goodness of fit of a model (the lower, the better), was also reported. For all tests described above, Wald statistic was used to evaluate the statistical significance of individual independent variables.

### Results

#### Patient characteristics

Table 1 shows the demographic and clinical characteristics of the 475 MI patients. The prevalence of type-D personality was 18.3% during hospitalization for MI (mean NA=7.3±6.2; mean SI=9.1±6.5), 22.2% at 12 months post-MI (mean NA=7.1±6.6; mean SI=8.7±6.3), and 23.2% at 18 months post-MI (mean NA=7.1±6.3; mean SI=8.7±6.5). Seventy patients (17%) had a diagnosis of lifetime depressive disorder, and 29 patients (7%) had lifetime anxiety disorder. On average, depressive symptoms scores were 7.1±6.3, 6.9±6.3, and 7.1±6.8, respectively, while state anxiety scores were 39.3±11.5, 36.3±12.5, and 36.1±11.6 for the three measurement occasions.

#### Confounders

None of the demographic and disease-related characteristics were significantly related to type-D status (Table 1). Smoking status, psychotropic medication, depressive and anxiety symptoms on the three measurement occasions, and lifetime diagnoses of depressive and anxiety disorders were significant univariate confounders for type-D status. In multivariate analyses, only depressive and anxiety symptoms (both mean levels and intraindividual variability) remained significant (P<.05).

### Table 1

Baseline characteristics of post-MI patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>All patients (N=475)</th>
<th>Type-D (n=87)</th>
<th>Non-type-D (n=374)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age in years [mean (S.D.)]</td>
<td>60 (11.6)</td>
<td>58 (10.9)</td>
<td>60 (11.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Female gender</td>
<td>104 (22)</td>
<td>21 (24)</td>
<td>76 (20)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Medical variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>181 (38)</td>
<td>45 (52)</td>
<td>131 (35)</td>
<td>.004</td>
</tr>
<tr>
<td>BMI in kg/m² [mean (S.D.)]</td>
<td>27 (4.0)</td>
<td>27 (4.0)</td>
<td>27 (4.1)</td>
<td>NS</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>99 (21)</td>
<td>20 (23)</td>
<td>77 (21)</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>69 (15)</td>
<td>7 (8)</td>
<td>59 (16)</td>
<td>.062</td>
</tr>
<tr>
<td>Cardiac history</td>
<td>89 (17)</td>
<td>14 (16)</td>
<td>61 (17)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>130 (30)</td>
<td>19 (23)</td>
<td>106 (31)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>56 (13)</td>
<td>10 (12)</td>
<td>44 (13)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Laboratory results [mean (S.D.)]</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol (mmol/l)</td>
<td>1.2 (0.6)</td>
<td>1.2 (0.5)</td>
<td>1.2 (0.6)</td>
<td>NS</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/l)</td>
<td>3.1 (1.0)</td>
<td>3.1 (1.0)</td>
<td>3.1 (1.0)</td>
<td>NS</td>
</tr>
<tr>
<td>LDL/HDL ratio (mmol/l)</td>
<td>4.6 (1.2)</td>
<td>4.6 (1.1)</td>
<td>4.6 (1.2)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Cardiac function [mean (S.D.)]</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>140 (29)</td>
<td>136 (24.6)</td>
<td>141 (29.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>82 (17)</td>
<td>80 (13.8)</td>
<td>83 (17.1)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Disease severity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>157 (40)</td>
<td>25 (34)</td>
<td>128 (41)</td>
<td>NS</td>
</tr>
<tr>
<td>PCI</td>
<td>284 (61)</td>
<td>51 (59)</td>
<td>225 (62)</td>
<td>NS</td>
</tr>
<tr>
<td>Anterior MI location</td>
<td>171 (40)</td>
<td>29 (37)</td>
<td>136 (41)</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac rehabilitation</td>
<td>281 (67)</td>
<td>54 (68)</td>
<td>223 (68)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Medication use</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-Blockers</td>
<td>398 (86)</td>
<td>77 (90)</td>
<td>311 (85)</td>
<td>NS</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>174 (38)</td>
<td>31 (36)</td>
<td>137 (38)</td>
<td>NS</td>
</tr>
<tr>
<td>Ca antagonists</td>
<td>83 (18)</td>
<td>15 (17)</td>
<td>63 (17)</td>
<td>NS</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>384 (83)</td>
<td>74 (86)</td>
<td>302 (83)</td>
<td>NS</td>
</tr>
<tr>
<td>Statins</td>
<td>421 (91)</td>
<td>81 (94)</td>
<td>330 (90)</td>
<td>NS</td>
</tr>
<tr>
<td>Diuretics</td>
<td>89 (19)</td>
<td>17 (20)</td>
<td>68 (19)</td>
<td>NS</td>
</tr>
<tr>
<td>A₂-antagonists</td>
<td>44 (10)</td>
<td>7 (8)</td>
<td>36 (10)</td>
<td>NS</td>
</tr>
<tr>
<td>Vasodilators</td>
<td>148 (32)</td>
<td>31 (36)</td>
<td>116 (32)</td>
<td>NS</td>
</tr>
<tr>
<td>Aspirin</td>
<td>379 (82)</td>
<td>70 (81)</td>
<td>300 (82)</td>
<td>NS</td>
</tr>
<tr>
<td>Antiarrhythmics</td>
<td>33 (7)</td>
<td>5 (6)</td>
<td>28 (8)</td>
<td>NS</td>
</tr>
<tr>
<td>Psychotropes</td>
<td>61 (13)</td>
<td>20 (24)</td>
<td>36 (10)</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

*Values are expressed as the number (percentage) of patients, unless otherwise indicated. Only P values <.10 are reported.

Given that 14 patients did not have complete data on the baseline assessment of the DS14, baseline characteristics stratified by type-D personality are shown only for 461 patients; nevertheless, due to the nature of the statistical analyses used to model the stability of type-D personality, it is possible to include all 475 patients since they had DS14 assessment on follow-up.

Arthritis, renal insufficiency, and chronic obstructive pulmonary disease.

Myocardial infarction, percutaneous coronary intervention, or CABG surgery prior to the index myocardial infarction.

Selective serotonin reuptake inhibitors and benzodiazepines.

### Type-D personality

LC regression showed that a two-class solution provided a better fit to the data than a one-class solution (Table 2,
Models 1–3. In this two-class model, classification error was small (7%), and the model explained 67% of the variance. Class 1, comprising 78% of the patients, was most common, with a minimal chance of having a type-D personality (~6%). Class 2 comprised 22% of the patients who had a high risk of having a type-D personality (between 64% and 91% for the three measurement occasions). In this univariate model, there was a main time effect that affected classes differently (the influence on Class 2 was much larger than the influence on Class 1).

In a subsequent multivariate analysis, depression and anxiety means and variability scores over time were added as additional predictors. Grand mean depression and anxiety scores significantly predicted type-D status, with a greater chance of having a type-D personality when mean depression or anxiety scores were higher (Table 2, Models 4–6), whereas intraindividual variability in depression and anxiety as predictors. The grand means of depressive and anxiety symptoms within a patient over this time period were significantly related to type-D status, with a greater chance of having a type-D personality when mean depression or anxiety scores were higher. This is not surprising, since type-D patients are more inclined to experience emotional and interpersonal difficulties such as depression and anxiety [11,25]. Moreover, personality has long been thought to predispose to mood and anxiety [26,27].

The type-D personality construct was designed to reflect a chronic psychological condition, as opposed to more acute emotional states such as depression and anxiety. Personality refers to a complex organization of traits that reflect consistencies in the general affective level and behavior of individuals over time [28]. A sound personality construct needs to have high predictive value, good construct validity, and stability over time [29]. Previous studies have already reported on the predictive value [1–4], construct validity [14,18,30], and 3-month stability [14] of the type-D construct. In concordance with these previous results, the current study provides further evidence for type-D personality being conceptualized as a stable personality construct rather than an epiphenomenon of an underlying cardiac disease or a reflection of mood status.

The prevalence of type-D personality in the current study increased slightly (3.9%) over the first year post-MI. The same kind of pattern was observed in a previous study of acute MI patients in which depression prevalence was 6.3% higher at 1 year post-MI [31].

The present findings should be interpreted with caution. First, since not all patients had had an echocardiography, we could only adjust for multivessel disease and not for left ventricular ejection fraction as a measure of disease severity. Second, we did not assess type-D personality at 6 months post-MI. Since the main difference in the probability of having a type-D personality was found between hospitalization and 12 months post-MI, and not between 12 and 18 months, it would be interesting to include a 6-month assessment in future studies. Third, we had no information on the overall response rate of the study. However, we were
able to look into a subsample of 63 patients who met the inclusion criteria; 46 of these patients gave informed consent, leaving a response rate of 73%. Finally, type-D personality was analyzed as a dichotomous variable, although a recent study by Emons et al. [32], using item response theory analysis, showed that all items of the Type D scale had the highest measurement precision around a cutoff of ≥10, and items were most informative at the higher end of the scale.

In conclusion, type-D personality was a stable taxonomy over an 18-month period in post-MI patients, and type-D classification was not affected by changes in mood status. These findings support the importance of including personality variables in cardiovascular research and the need for intervention trials targeting type-D personality in order to enhance secondary prevention in this subset of CVD patients. Future studies should replicate these findings in healthy and other CVD populations, including patients with chronic heart failure, peripheral arterial disease, arrhythmias, and hypertension.

Acknowledgments

This research was supported by The Netherlands Organization for Scientific Research (NWO) with a VENI grant (451-05-001) to Dr. S.S. Pedersen and a VICI grant (453-04-004) to Prof. Dr. J. Denollet. We thank Prof. Dr. J.K. Vermunt (Tilburg University) for his support with statistical procedures.

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