Type D Personality Is Associated with the Development of Stress Cardiomyopathy Following Emotional Triggers

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Abstract

Background Stress cardiomyopathy (SCM) can be triggered by emotional events. Recently, type D personality has been established as an independent predictor of acute cardiac adverse events.

Purpose We sought to examine whether type D personality can be identified in SCM patients.

Methods A case–control study with 37 SCM patients, 37 myocardial infarction (AMI) patients, who both experienced emotional triggering, and 37 SCM patients without emotional triggers was performed. The DS14 and Interview for Recent Life Events were administered.

Results Twenty-eight (76 %) SCM emotional trigger patients were categorized as type D compared with 13 (43 %) SCM patients without emotional trigger and 12 (32 %) AMI patients (p<0.001). SCM patients with emotional triggers had higher scores on the social inhibition subscale than the other patient groups.

Conclusions The present study highlights the possible link between type D, with a specific key role for social inhibition component, and increased biological reactivity to acute emotional stress.

Keywords Stress cardiomyopathy · Case–control Studies · Personality traits · Emotional triggering · Type D

Introduction

During the last few years, there has been increasing awareness of specific transient cardiomyopathy, referred to as takotsubo or stress cardiomyopathy (SCM) [1–3], that has a clinical presentation indistinguishable from an acute myocardial infarction (AMI). The main features of stress cardiomyopathy are transient apical left ventricular dysfunction that mimics myocardial infarction, but in the absence of significant coronary artery disease. Several variants, including mid-ventricle and basal inverted patterns, have also been reported [4]. An excessive catecholamine-induced sympathetic stimulation of the heart, causing an acute stunning of the myocardium, has been suggested as a pivotal mechanism in stress cardiomyopathy [2]. This hypothesis is further supported by the observation that stress cardiomyopathy patients with pheochromocytoma develop transient reduction in left ventricular systolic function in conjunction with a rise in plasma catecholamine levels [2, 5]. Despite evidence that the myocardial stunning of stress cardiomyopathy may be sympathetically mediated, the precise pathogenesis of this disorder remains incompletely understood.

Frequently, stress cardiomyopathy may be triggered by acute emotionally stressful events in the hours preceding symptom onset. Emotional triggers have been observed in 20–33 % of patients in retrospective studies [6, 7] and 36–47 % in prospective clinical cohort studies [8, 9]. Since most people are subjected to repeated emotional and physiologic...
acute stressors throughout their lives, the fact that only a relatively small number develop stress cardiomyopathy suggests that there are likely to be risk factors that increase individual susceptibility. These risk factors may influence a person’s physiologic response to acute stress and increase their vulnerability to the pathophysiologic mechanisms that result in myocardial stunning. Studies support the role of hormonal processes [10], endothelial dysfunction [6], and genetic factors [11] as susceptibility factors for stress cardiomyopathy. In terms of mental health, stress cardiomyopathy patients appear to have a high prevalence of mixed depression and anxiety, ranging between 21 and 40 % [12–14]. Studies that have distinguished anxiety and depression have shown rates of 30–56 % for anxiety [15, 16] and 36–48 % for depression [15, 16]. Additionally, a recent study showed high trait anxiety in 60 % of stress cardiomyopathy subjects [17]. Although some studies have focused on pre-morbid psychiatric symptoms or disorders, no research to date has specifically investigated the personality traits of stress cardiomyopathy patients. Investigation of the characteristic patterns of thoughts, feelings, and behaviors of stress cardiomyopathy patients would promote understanding of the personality traits that may make some people susceptible to the deleterious cardiovascular effects of sympathetic stimulation following an acute emotional stressor.

Findings from behavioral genetic studies support the notion that stable personality traits may play a role in stress-related autonomic reactions. Specific personality traits are associated with genotypic variations in hypothalamic–pituitary–adrenocortical (HPA) afferents and seem to regulate the HPA response to psychological and physiological stressors [18, 19]. One personality trait that may be particularly relevant is type D personality [20].

Type D personality is thought to be a relatively stable personality trait [21] characterized by high negative affectivity (NA—tendency to experience negative emotions such as dysphoria, anxiety, irritability, and negative view of self across time/situations) and social inhibition (SI—tendency to inhibit the expression of emotions/behaviors in social interactions to avoid disapproval by others or feeling inhibited, tense, and insecure when with others). Several studies have demonstrated that type D is an independent predictor of major adverse cardiac events in patients with coronary heart disease [22–25]. The prevalence estimates of type D personality are between 24 and 31 % in the general population [26, 27] and ranges between 15 % [28] and 53 % [29], respectively, in coronary artery disease (CAD) and hypertensive patients. Recent cross-cultural analyses of coronary heart disease patients have shown that the prevalence of type D personality ranges between 24 and 37 % [30]. Although these studies have confirmed a significant association between type D personality and mortality and nonfatal myocardial infarction, heterogeneity between studies was large and the prognostic effects of type D have decreased considerably in recent compared with older studies [31–34].

Recent studies [31, 35, 36] have been critical of the conceptualization of type D as a personality construct based on the practice of dichotomizing the negative affectivity and social inhibition scales and defining type D as being in the upper division of both scales. It has been argued that type D is better represented as a dimensional than a categorical construct and that the categorization of variables is not an appropriate representation of the synergistic combination of the two dimensions that supposedly underlies the concept [37]. Our primary analyses were therefore based on continuously distributed scores on the two components and their interactions, though we also present results based on the categorization of patients into high and low negative affectivity and social inhibition groups.

There has been no previous research on type D personality in stress cardiomyopathy patients. However, a recent laboratory study [38] involving healthy women showed that type D individuals displayed reduced cardiac relative to vascular responses to acute laboratory stress, consistent with other studies indicating that type D personality is associated with modified cardiovascular responses to acute psychological stress [39, 40]. It is possible, therefore, that type D personality may make some individuals more susceptible to stress cardiomyopathy reactions following acute emotional stressors.

Accordingly, the aim of this controlled investigation was to test whether emotion-triggered stress cardiomyopathy patients differ in type D personality from those without emotion-triggered stress cardiomyopathy. In order to discover whether any differences were due to emotional triggering per se, we also included a group of acute myocardial infarction patients who experienced emotional triggering matched for socio-demographic variables. We hypothesized that type D would be more common in emotionally triggered stress cardiomyopathy than in the other two groups of patients.

**Method**

In this cross-sectional study, 37 stress cardiomyopathy patients who experienced emotional triggering (4 males, 33 females; mean age, 66 years, SD=12.8), 37 stress cardiomyopathy patients who did not experience emotional triggering (4 males, 33 females; mean age, 66 years, SD=11.1), and 37 acute myocardial infarction patients who also experienced emotional triggers (4 males, 33 females; mean age, 66 years, SD=10.1) were enrolled. Both patients with stress cardiomyopathy and those with acute myocardial infarction were newly diagnosed and had not previously been treated at the time of study. The stress cardiomyopathy patients were admitted consecutively to our hospital-based specialized cardiovascular clinics over a 3 year period. Stress cardiomyopathy was diagnosed according to
the Mayo Clinic diagnostic criteria [41] including: (1) an acute cardiac event typically presenting with substernal chest pain; (2) systolic dysfunction with a marked left ventricular contraction abnormality, extending beyond the geographic territory of a single epicardial coronary artery, assessed with left ventricular angiography, cardiovascular magnetic resonance imaging, or two-dimensional echocardiography; and (3) absence of obstructive atherosclerotic coronary artery stenosis (i.e., ≤50% luminal narrowing of the epicardial arteries by angiography). The most common presenting cardiovascular symptoms were substantial chest pain (n=46), exertional dyspnea (n=18), and syncope (n=11). Careful history taking identified whether significant stressful events immediately preceded the presentation of stress cardiomyopathy. Preliminary assessment of trigger events was carried out by cardiologists during admission to the emergency unit. Information about trigger events was, where possible, confirmed with the companion (if any) who accompanied the patient to the emergency unit. Thirty-seven of the 75 stress cardiomyopathy patients (49.33%) reported an emotional trigger within the past 48 h, while 38 (50.66%) experienced a physical trigger (of which 71% was acute respiratory infection, 26% post-surgical/fracture, and 3% migraine headache) or else no trigger event. Over the same period of stress cardiomyopathy enrollment, the first consecutive 37 acute myocardial infarction patients who reported acute emotional triggers, and without knowledge of the clinical diagnosis of the patients. The clinical psychologist made sure that all subjects provided complete data in the questionnaire.

Psychological Assessment

All patients were interviewed by two clinical psychologists who were blind to the patients’ group. Interviews involved a thorough assessment of emotional trigger events that occurred within 48 h of symptom onset and were performed within 8 h of initial screening in order to avoid memory distortion effects. Type D personality assessment was performed by two different clinical psychologists 3 months after initial screening and without knowledge of the clinical diagnosis of the patients. The clinical psychologist made sure that all subjects expressed as the mean±SD. Differences among groups were assessed by one-way ANOVA for continuous variables followed by Bonferroni-corrected post hoc tests. Hierarchical logistic regression analyses with z-transformed negative affectivity and social inhibition scores, entering the main effect terms followed by the NA×SI interaction, were used to test differences among groups. These analyses were adjusted for diabetes, history of CAD, systolic blood pressure (SBP), and hypertension since these factors may be particularly relevant to the development of stress cardiomyopathy, as indicated in Table 1.
The final results are presented as adjusted odds ratios (ORs) with 0.95 confidence intervals (CI) and exact p values. Statistical analyses were performed with SPSS, version 18.0 (SPSS Institute Inc., Chicago, IL).

Results

Socio-demographic and clinical characteristics of stress cardiomyopathy and acute myocardial infarction patients are reported in Table 1. Patients with stress cardiomyopathy and acute myocardial infarction were not significantly different with regard to age, sex, marital status, living alone, and ST segment elevation (Table 1). As expected, patients with acute myocardial infarction had significantly higher levels of troponin T, creatine kinase-MB fraction, white cell count, hypertension, and systolic blood pressure compared with stress cardiomyopathy patients. Moreover, acute myocardial infarction patients had a higher prevalence of diabetes and history of coronary artery disease compared with the other groups.

Emotional Trigger Events

The acute emotional events experienced by patients in this study are summarized in Table 2. Loss experiences through death or separation from someone close (son, father/mother, husband, friend, etc.) were the most prevalent emotional triggers in both the stress cardiomyopathy and acute myocardial infarction groups (Table 2). Ten (27.0 %) stress cardiomyopathy patients and nine (24.3 %) acute myocardial infarction patients had severe loss experiences within 12 h of cardiac symptom onset. Other common events were job loss, a serious quarrel, and the unexpected hospitalization of someone close. The overall severity score for emotional triggers (Paykel Stress Index) and their timing did not differ significantly between the two groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>SCM-t (n=37)</th>
<th>SCM-nt (n=38)</th>
<th>AMI (n=37)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD)</td>
<td>66 (12.8)</td>
<td>66 (11.1)</td>
<td>66 (10.1)</td>
<td>0.81</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>4 (10.1)</td>
<td>4 (10.8)</td>
<td>4 (10.8)</td>
<td>0.92</td>
</tr>
<tr>
<td>Marital status, n (%)</td>
<td>0.78</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>22 (59.5)</td>
<td>20 (54.1)</td>
<td>21 (56.8)</td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>13 (35.1)</td>
<td>14 (37.8)</td>
<td>11 (29.7)</td>
<td></td>
</tr>
<tr>
<td>Divorced</td>
<td>2 (5.4)</td>
<td>3 (8.1)</td>
<td>5 (13.5)</td>
<td></td>
</tr>
<tr>
<td>Living alone</td>
<td>6 (16.2)</td>
<td>7 (18.9)</td>
<td>8 (21.6)</td>
<td>0.58</td>
</tr>
<tr>
<td>Cardiac risk factors</td>
<td>0.64</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>5 (13.5)</td>
<td>4 (10.8)</td>
<td>7 (18.9)</td>
<td>0.36</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>4 (10.8)</td>
<td>2 (5.4)</td>
<td>18 (48.6)</td>
<td>0.04 c</td>
</tr>
<tr>
<td>History of CAD, n (%)</td>
<td>0.03 c</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>4 (10.8)</td>
<td>2 (5.4)</td>
<td>10 (27.0)</td>
<td>0.03 c</td>
</tr>
<tr>
<td>BMI, mean (SD)</td>
<td>23.8 (3.3)</td>
<td>23.1 (2.4)</td>
<td>23.6 (3.1)</td>
<td>0.37</td>
</tr>
<tr>
<td>Clinical presentation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest pain, n (%)</td>
<td>32 (86.0)</td>
<td>0 (0.0)</td>
<td>32.0 (86.0)</td>
<td>0.74</td>
</tr>
<tr>
<td>EF, mean (SD)</td>
<td>24.0 (5.6)</td>
<td>26.3 (4.4)</td>
<td>27.0 (4.4)</td>
<td>0.83</td>
</tr>
<tr>
<td>TT, mean (SD)</td>
<td>2.6 (3.7)</td>
<td>0.0 (0.0)</td>
<td>9.4 (4.1)</td>
<td>0.04 ab</td>
</tr>
<tr>
<td>CK-MB, mean (SD)</td>
<td>11.8 (5.9)</td>
<td>0.0 (0.0)</td>
<td>28.5 (6.3)</td>
<td>0.03 ab</td>
</tr>
<tr>
<td>WBCC, mean (SD)</td>
<td>7.3 (4.2)</td>
<td>4.7 (3.1)</td>
<td>12.4 (5.6)</td>
<td>0.02 c</td>
</tr>
<tr>
<td>HR, mean (SD)</td>
<td>90.2 (5.3)</td>
<td>89.4 (3.4)</td>
<td>95.3 (2.3)</td>
<td>0.54</td>
</tr>
<tr>
<td>SBP, mean (SD)</td>
<td>117.2 (2.1)</td>
<td>115.2 (2.3)</td>
<td>124.1 (3.8)</td>
<td>0.02 c</td>
</tr>
</tbody>
</table>

CAD coronary artery disease, SBP systolic blood pressure (in millimeters of mercury), HR heart rate (in beats per minute), WBCC white blood cell count (×10³/μL), EF ejection fraction (in percent), BMI body mass index (in kilograms per square meter), CK-MB Creatine kinase-MB fraction (in nanograms per milliliter), TT troponin T (in nanograms per milliliter), SCM-t stress cardiomyopathy patients with emotion triggers, SCM-nt stress cardiomyopathy patients without emotion triggers, AMI acute myocardial infarction

a p<0.01, SCM-t and AMI vs. SCM-nt
b p<0.01, SCM-nt and AMI vs. SCM-t
c p<0.01, SCM-t and SCM-nt vs. AMI
Type D Personality

In the statistical analysis of the continuous score of two components of Type D (Table 3), the three groups differed on the social inhibition subscale \( (p<0.001) \), but not on the negative affectivity scale. Subsequent tests indicated that social inhibition scores were higher in patients with emotionally triggered stress cardiomyopathy than non-emotionally triggered stress cardiomyopathy or acute myocardial infarction \( (p<0.01) \), while the SCM without emotional triggers and acute myocardial infarction groups did not differ. Additionally, analysis of type D as a categorical construct showed that of the 37 SCM patients with emotional triggers, 28 (76 %) were categorized as type D compared with 13 (43 %) SCM patients without emotional triggers and 12 (32 %) acute myocardial infarction patients. The difference among the three groups was significant \( (\chi^2=15.12; \text{see Table 3 and Fig. 1}) \). Post hoc analyses revealed that for type D classification, the difference was significant between SCM with emotional triggers and both SCM without emotional triggers and acute myocardial infarction, and between SCM without emotional triggers and acute myocardial infarction. For negative affectivity, the differences were significant between SCM with emotional triggers and SCM without emotional triggers in comparison with acute myocardial infarction patients, but did not differ from each other. For social inhibition, the differences were significant between SCM with emotional triggers and SCM without emotional triggers, and between SCM with emotional triggers and acute myocardial infarction, but not between SCM without emotional triggers and acute myocardial infarction.

Results of the hierarchical logistic regressions are summarized in Table 4. The negative affectivity and social inhibition scales were z-transformed for these analyses so that the interaction between negative affectivity and social inhibition gave equal weight to the two components. After adjusting for diabetes, history of CAD, SBP, and hypertension, the NA × SI interaction term distinguished stress cardiomyopathy from acute myocardial infarction.

<table>
<thead>
<tr>
<th>Emotional trigger categories</th>
<th>SCM-t (n=37)</th>
<th>SCM-nt (n=37)</th>
<th>AMI (n=37)</th>
<th>(p) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sudden hospitalization—others</td>
<td>7 (18.9)</td>
<td>6 (16.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis of illness</td>
<td>3 (8.1)</td>
<td>10 (18.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Judicial notification</td>
<td>3 (10.8)</td>
<td>3 (9.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quarrel</td>
<td>6 (16.2)</td>
<td>7 (18.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Job loss</td>
<td>6 (16.2)</td>
<td>7 (18.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eviction from home</td>
<td>4 (10.8)</td>
<td>2 (4.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>29 (78)</td>
<td>17 (44)</td>
<td>12 (32)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 2 Emotional trigger events on the Interview for Recent Life Events in SCM and AMI patients

Table 3 Comparison between SCM, with and without emotional triggers, and AMI patients on Type D and subscales in terms of categorical and continuous variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>SCM-t (n=37)</th>
<th>SCM-nt (n=37)</th>
<th>AMI (n=37)</th>
<th>(p) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type D-NA, mean (SD)</td>
<td>14.3 (2.1)</td>
<td>13.2 (3.4)</td>
<td>14.8 (2.6)</td>
<td>0.44</td>
</tr>
<tr>
<td>Type D-SI, mean (SD)</td>
<td>25.4 (2.4)</td>
<td>13.4 (1.1)</td>
<td>11.1 (3.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Categorical variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type D classification, (n) (%)</td>
<td>28 (76)</td>
<td>16 (43)</td>
<td>12 (32)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Type D-NA ≥10, (n) (%)</td>
<td>29 (78)</td>
<td>27 (74)</td>
<td>14 (38)</td>
<td>0.04</td>
</tr>
<tr>
<td>Type D-SI ≥10, (n) (%)</td>
<td>34 (92)</td>
<td>17 (44)</td>
<td>15 (41)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

\(NA\) negative affectivity, \(SI\) social inhibition, \(SCM-t\) stress cardiomyopathy patients with emotion triggers, \(SCM-nt\) stress cardiomyopathy patients without emotion triggers, \(AMI\) acute myocardial infarction

\(a\ p<0.01, \text{SCM-t vs. AMI and SCM-nt}\)

\(b\ p<0.01, \text{SCM-t vs. AMI}\)

\(c\ p<0.01, \text{SCM-nt vs. AMI}\)
myocardial infarction (OR=1.72, 95% CI=1.21–2.11, \(p=0.03\)), indicating that the interaction between the two components is crucial. Within stress cardiomyopathy, the odds of belonging to the SCM with emotional triggers compared with SCM without emotional triggers group were 1.11 (\(p=0.04\)) for every unit increase in the NA by SI interaction and 1.86 (\(p=0.02\)) in comparison with the acute myocardial infarction group. Scores on the social inhibition component also distinguished SCM with emotional triggers from acute myocardial infarction (OR=1.92, \(p=0.01\)) and SCM with emotional triggers from SCM without emotional triggers (OR=1.53, \(p=0.04\)).

**Discussion**

The results of this study document for the first time a higher prevalence of type D personality in stress cardiomyopathy patients, both with and without emotional triggers, compared with emotion-triggered acute myocardial infarction patients. In particular, the social inhibition component of type D personality seems to represent a trait which differs between stress cardiomyopathy patients who experience emotional triggers and matched stress cardiomyopathy patients without emotional triggers and acute myocardial infarction patients. The prevalence of type D in stress cardiomyopathy patients was not only higher than the comparison group tested in this study but also higher than the levels observed in other studies of acute myocardial infarction patients [28] and in general populations [26, 27].

As noted in the introduction, there have been criticisms of the categorical approach to type D personality. However, it is worth noting the prevalence of type D as conventionally defined in comparison with rates in different cardiac groups. General population studies have reported a prevalence of 22% [45] of type D personality. Within the cardiac patient population, the prevalence rate has ranged from 29% [46] in general cardiac patients to 37% in acute myocardial infarction patients [30]. Our findings show a prevalence rate of type D personality within the acute myocardial infarction group of 32%, similar to that shown elsewhere in the literature. The prevalence of type D in the group of stress cardiomyopathy patients was 59.5%, a greatly elevated level. There are no other data in the literature on stress cardiomyopathy with which to compare our results.

Our research underscores some differences in results between considering type D as a dimensional rather than a categorical construct, as has been suggested by others [31, 35, 36]. Both the analyses of continuous scores on the two dimensions and the categorical analyses show that the differences in the prevalence of type D personality between groups were primarily attributable to social inhibition, with the synergistic interaction between social inhibition and negative affectivity being driven in large part by elevated social inhibition scores in the stress cardiomyopathy patients (SCM) with emotional triggers group. As shown in Table 2, patients who experienced emotionally triggered stress cardiomyopathy had a substantially higher rate of elevated social inhibition (92%) compared with stress cardiomyopathy patients without emotional triggers (44%). The difference between these two

| Table 4 | ORs and 95% CIs between groups |

<table>
<thead>
<tr>
<th></th>
<th>SCM vs. AMI*a</th>
<th>SCM-t vs. SCM-ntb</th>
<th>SCM-t vs. AMI*a</th>
<th>SCM-nt vs. AMI*a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR 95% CI</td>
<td>OR 95% CI</td>
<td>OR 95% CI</td>
<td>OR 95% CI</td>
</tr>
<tr>
<td>Type D-NA (z score)</td>
<td>1.01 0.82–1.15 0.11</td>
<td>1.05 0.54–1.23 0.21</td>
<td>1.00 0.89–1.12 0.38</td>
<td>1.22 0.97–1.87 0.21</td>
</tr>
<tr>
<td>Type D-SI (z score)</td>
<td>1.04 0.92–1.21 0.13</td>
<td>1.53 1.12–2.28 0.04</td>
<td>1.92 1.22–2.45 0.01</td>
<td>0.99 0.72–1.04 0.32</td>
</tr>
<tr>
<td>NA by SI</td>
<td>1.72 1.21–2.11 0.03</td>
<td>1.11 1.09–2.02 0.04</td>
<td>1.86 1.12–2.53 0.02</td>
<td>1.18 0.89–1.68 0.33</td>
</tr>
</tbody>
</table>

*NA negative affectivity, SI social inhibition, SCM-t stress cardiomyopathy patients with emotion triggers, SCM-nt stress cardiomyopathy patients without emotion triggers, AMI acute myocardial infarction

*a AMI is the reference group

*b SCM-nt is the reference group
groups in elevated negative affectivity was much smaller (78 vs. 74%). Denollet [29] defines social inhibition as the “tendency to inhibit expression of emotions/behaviors in social interaction to avoid disapproval of others” and the tendency to “feel inhibited, tense, and insecure when with others” (p. 89). Social inhibition, a construct conceptually related to emotional inhibition and suppression, has been linked to cardiovascular reactivity to acute stress [47]. Findings from theories of emotion and self-regulation indicate that expressive suppression increases sympathetic activation of the cardiovascular system [48, 49]. Indeed, the tendency not to share negative thoughts and feelings, such as those resulting from stressful emotional events, has been shown to be associated with exaggerated sympathetic and cardiovascular responses in response to later events [50, 51]. Additionally, high dispositional negative affectivity may serve to amplify negative emotional responses following stressful events, and this may have biological correlates that stimulate cardiac dysfunction.

Emotional triggering is recognized as a significant phenomenon in a proportion of acute myocardial infarction cases [52]. The acute myocardial infarction comparison group was selected on the basis of reporting emotional triggers, so the prevalence of triggers is not representative of patients with acute myocardial infarction in general. Emotional triggering of acute myocardial infarction has previously been associated with chronic life stress and depression in the post-hospitalization period [53], so the elevated negative affectivity scores of the acute myocardial infarction group were anticipated. However, it is striking that social inhibition scores were not raised in the acute myocardial infarction group, suggesting a specific association of social inhibition with stress cardiomyopathy pathology.

Our findings lead to the hypothesis that there is a vulnerable personality profile for stress cardiomyopathy reactions after emotional triggers. A possible explanation is that type D personality is associated with heightened cardiovascular reactivity to acute stress [39] and that social inhibition is associated with higher cardiovascular reactivity [54], lower cardiovascular recovery and heart rate variability [55], and with overactivity of the sympathetic nervous system [56]. In our stress cardiomyopathy sample, the prevalence of conventional cardiac risk factors was lower than that of acute myocardial infarction patients. This finding confirms previous reports [57] which indicate that the pathophysiology of the syndrome differs from that of coronary heart disease. Current evidence emphasizes the pathophysiological importance of extreme sympathetic nervous system activation in stress cardiomyopathy and suggests that endothelial dysfunction is involved [6]. Thus, type D personality traits could be conceptualized as contributing to individual differences in cardiovascular reactivity to acute emotional stressors through sympathetic outflow to the heart, with cardiac noradrenaline spillover potentially achieving very high values [58].

The emotional trigger data collected in this study show that 27% of stress cardiomyopathy patients experienced the death of someone close between 3 and 12 h of symptom onset. This finding is analogous with data from previous retrospective studies where rates of grief/loss trigger events in stress cardiomyopathy patients varied from 23% [59] to 28% [7].

**Strengths and Limitations**

Emotional triggers were assessed with thorough systematic procedures and established questionnaires rather than impressionistic clinical observations, as in much previous work. The three groups were carefully characterized and well matched socio-demographically. The proportion of male emotion-triggered stress cardiomyopathy patients (10.8%) was similar in our study and others [7, 60], strengthening the possibility of generalizing the results of the present study to the emotion-triggered stress cardiomyopathy population. But since other studies have found different proportions of patients experiencing emotional triggers, the results cannot necessarily be generalized to all stress cardiomyopathy. However, the sample size was small in this cross-sectional observational study and causal conclusions cannot be drawn. A limitation to the study is the timing of the personality assessment. This was performed 3 months after hospitalization to allow the stabilization of the patient’s emotional condition. We do not know whether ratings would have been similar if the assessment had taken place before the clinical episode. However, if the social inhibition and negative affectivity effects were secondary to cardiac disease, one might have expected a worse profile in the acute myocardial infarction group since they suffered from more sustained myocardial damage. This is not the pattern that we observed.

**Conclusions**

In conclusion, the present study shows for the first time a link between type D personality and emotionally triggered stress cardiomyopathy. Our results highlight the possible link between type D personality and increased biological reactivity to acute emotional stress. They are in keeping with findings from experimental studies showing that the cardiovascular effects of acute stress may be amplified by the presence of chronic psychosocial stress [61, 62]. The key role of social inhibition of negative emotions as a characteristic of type D personality in emotionally triggered stress cardiomyopathy patients emphasizes the importance of emotional regulation processes in biological reactions to acute emotional stress. This implies that the risk of a stress
cardiomyopathy event may depend not only on the individual’s cardiovascular vulnerability following stress exposure but also on his or her coping mechanisms. Further investigation may shed light both on these emotional regulatory processes and on the protective role of coping mechanisms in the links between personality and susceptibility to stress cardiomyopathy following acute emotional events.

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References


