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Research report

Type D personality in never-depressed patients and the development of major and minor depression after acute coronary syndrome

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Carlo Marchesi [a,b,*], Paolo Ossola [a,b], Francesca Scagnelli [a,b], Francesca Paglia [a,b], Sonja Aprile [a,b], Alberto Monici [b], Matteo Tonna [b], Giulio Conte [c], Franco Masini [c], Chiara De Panfilis [a,b], Diego Ardissino [c]

[a] Department of Neuroscience, Psychiatry Unit, University of Parma, Parma, Italy
[b] Mental Health Department, AUSL, Parma, Italy
[c] Department of Cardiology, University Hospital, Parma, Italy

ABSTRACT

Background: Type D personality (TDP) has been proposed as a risk factor for the development of depressive symptoms after an acute coronary syndrome (ACS). However, contrasting findings emerged about its predicting power on the onset of depression, since an overlap between TDP and depressive symptoms has been proposed. The present study was aimed to verify whether TDP predicts the development of a depressive disorder in the 6 months after the discharge from hospital.
Methods: Two hundred fifty consecutive patients were recruited, at the Coronary Intensive Care Unit at the University Hospital of Parma, who were both presenting their first ACS and had no history of depression. The presence and the severity of major (MD) and minor (md) depression were evaluated with the Primary Care Evaluation of Mental Disorders (PRIME-MD) and the Hospital Anxiety and Depression Scale (HADS) respectively. Type D Personality was assessed with the DS14, both at baseline and at 1, 2, 4 and 6 month follow ups.
Results: Out of 250 subjects (81.2% males), MD was diagnosed in 12 patients (4.8%) and md in 18 patients (7.2%). At baseline risk factors for a post-ACS depressive disorder were HADS depression scores, whereas TDP, or its subscales, did not showed any effect.
Limitation: The small amount of patients with incidence of depression, due to highly selective inclusion criteria, tempers the reliability of our results.
Conclusion: Our data suggests that TDP does not predict the development of depressive disorders in never-depressed patients at their first ACS, when the baseline depression severity was controlled.

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1. Introduction

An increased association between Coronary Artery Disease (CAD) and depression has been widely documented (Rudisch and Nemeroff, 2003; Härter et al., 2007; Baumeister et al., 2010) and a twofold risk of developing depression in patients with CAD has been estimated (Ormel et al., 2007), with up to two out of 10 patients with Acute Coronary Syndrome (ACS) developing depressive symptoms in the following year (McGee et al., 2006; Thombs et al., 2006). Several risk factors for developing depression after ACS have been identified (Spijkerman et al., 2005; Martens et al., 2008; Kaptein et al., 2006; Doyle et al., 2011a,) including psychopathological history, personality features and stressful events.

Since depression itself has been suggested as a cardiac risk factor (Carney, 1998; Frasure-Smith and Lespérance, 2005, 2010), the development of depression after ACS seemed to worsen cardiac outcome (Frasure-Smith et al., 2000; Parashar et al., 2006; Parker et al., 2008; Baumeister et al., 2011), increasing mortality rate (Lespérance and Frasure-Smith, 1996; Barth et al.; 2004; Nicholson et al., 2006;) and the incidence of new cardiac events (de Jonge et al., 2006; Kaptein et al., 2006;).

Distressed (Type D) Personality, as defined by Denollet and colleagues (Denollet et al.,1996), is characterized by two different dimensions: Negative Affectivity (NA) and Social Inhibition (SI). Individuals displaying high NA experience feelings of dysphoria, anxiety, irritability, have a negative self view and scan the world for signs of impending trouble (Watson and Pennebaker, 1989); while high SI predisposes individuals to inhibit their behaviour and expressions of emotion while engaged in social interactions in order to avoid disapproval and feeling tense and insecure when around others (Asendorpf, 1993; Gest, 1997). Type D personality

* Corresponding author at: Department of Neuroscience, Psychiatry Unit, University of Parma, University Hospital, Braga Building, 43125 Parma, Italy. Tel.: +39 0521 903597; fax: +39 0521 396825.
E-mail address: carlo.marchesi@unipr.it (C. Marchesi).

(TDP) is defined by the presence of both high NA and SI and a specific instrument (DS14) has been developed for its assessment (Denollet, 2005).

Furthermore, type D personality (TDP) is also thought to be a risk for the development (Pedersen et al., 2006; Spindler et al., 2009; Yu et al., 2010; De Fazio et al., 2012), persistence (Martens et al., 2008; Smith et al., 2008; Doyle et al., 2011a, b; Romppel et al., 2012a) and worsening (Romppel et al., 2012a) of depressive symptoms in cardiac patients, and this effect has been particularly attributed to Negative Affectivity (NA). Although some studies suggested that Type D is a stable construct (Denollet, 2005; Martens et al., 2007; Kupper et al., 2011; Romppel et al., 2012b), with conceptual (Denollet et al., 1996, 2010; Denollet and Pedersen, 2008; Denollet and Conraads, 2011) and biological basis (Kupper et al., 2007) and not influenced by mood changes (Denollet et al. 2005; de Jonge et al., 2007; Martens et al., 2007; Yu et al., 2010), others found that TDP was associated with depressive features (Kuijpers et al., 2007; Spindler et al., 2009; Dannemann et al., 2010; Svansdottir et al., 2012), general distress (Bergvik et al., 2010) and a history of depression (Martens et al., 2008; Starrenburg et al., 2013); with the stronger association (Kudielka et al., 2004; Bergvik et al., 2010; Coyne et al., 2011; Coyne and de Voogd, 2012; Howard and Hughes, 2012; Starrenburg et al., 2013; Tully and Penninx, 2012) found for NA.

To elucidate the relationship between TDP and depression, previous studies, using factor analysis, found a partial overlap between NA and depression (Kudielka et al., 2004) or anxiety (Pelle et al., 2009); suggesting that NA might also measure some features of depression and anxiety rather than solely a personality disposition. This suggestion, was supported by a recent study which stressed that "NA comprises state as well as trait facets" (Pelle et al., 2009). These findings underscore the need for assessing depression and anxiety levels when TDP is evaluated as a risk factor for depression in ACS; this implies that the predictive power of TDP should be adjusted for depression and anxiety at baseline.

A support for the hypothesis that TDP is a risk for depression comes from previous prospective studies (Pedersen et al., 2006; Martens et al., 2008; Smith et al., 2008; Doyle et al., 2011a; Romppel et al., 2012a). However, none of these studies have ever included never-depressed patients at baseline, all of them evaluated depressive symptoms rather than disorders, most of them (Martens et al., 2008; Smith et al., 2008; Doyle et al., 2011a; Romppel et al., 2012a) evaluated the prediction of stability of depression over time rather than its onset, and some studies (Martens et al., 2008; Pedersen et al., 2006) did not adjusted for the baseline severity of depressive symptoms. Moreover, some authors (Romppel et al., 2012a) concluded that the low sensitivity and low positive predictive value of the Type D Scale (DS14) in predicting depression in cardiac patients limit its potential usefulness in clinical practice.

The aforementioned studies did not clarify whether TDP represents a predictive power on the onset of depression after an ACS, leading some authors (Doyle et al., 2007) to suggest that more knowledge on depressive vulnerabilities in CAD patients is needed.

The present study aims to verify whether, in never-depressed subjects at their first ACS, TDP predicts the development of a depressive disorder (major and minor depression), after controlling for the effect of baseline severity of depressive and anxious symptoms.

2. Method

The Local Ethics Committee approved the study protocol and the study was conducted according to the Helsinki Declaration. All patients provided informed consent.

2.1. Sample

The study sample was selected among patients who were consecutively admitted to the Coronary Intensive Care Unit of the University Hospital of Parma, from January 2009 to March 2012, for an ACS.

Patients were included in the study if at the time of enrolment: (1) their age was over 18 years; (2) they were a native Italian speaker or were proficient in Italian; (3) they were at their first acute coronary episode; (4) they had no previous or current major depressive episode according DSM-IV (American Psychiatric Association (1994)); (5) they had no substance abuse or dependence; (6) they did not show cognitive impairment as demonstrated by a Mini Mental State Examination (MMSE) (Folstein et al., 1975) lower than 25; (7) they did not take any psychotropic medication.

2.2. Assessment

All patients underwent the following evaluations at baseline: (1) a brief socio-demographic interview; (2) the Primary Care Evaluation of Mental Disorder (PRIME-MD) (Spitzer et al., 1994); (3) the Hospital Anxiety and Depression Scale (HADS) (Zigmond and Snaith, 1983); (4) the Type-D personality Scale-14 (DS14).

At baseline patients were also interviewed by an expert psychiatry to confirm if the PRIME-MD answers fit their clinical condition and to exclude the presence of previous MD episodes.

The same evaluations were re-administered to all patients after 1, 2, 4 and 6 months of follow-up.

The PRIME-MD is a structured interview designed to diagnose mental disorder according DSM-IV. A part of PRIME-MD evaluates the presence of nine depressive symptoms in the previous 2 weeks. Each symptom is rated on a four-point scale (from "not at all" to "most of the days"). Moreover, the PRIME-MD also rates on a four-point scale the difficulty in daily functioning due to the depressive symptoms (from "not at all" to "extremely difficult").

The PRIME-MD has showed good specificity (98%) and sensitivity (73%) in detecting MD in primary care (Spitzer et al., 1999).

A patient was defined depressed, according PRIME-MD, if, at any evaluation time, (1) he/she fulfilled the criteria for a major depressive episode (MD) (i.e. at least five depressive symptoms in the last 2 weeks, of which one should be depressed mood or loss of pleasure, were present for "more than half of the days" or "almost all the days". Moreover, these symptoms should make daily functioning "very difficult" or "extremely difficult"); or (2) he/she fulfilled the criteria for a minor depressive episode (md), (i.e. at least two but less than five depressive symptoms in the last 2 weeks, of which one should be depressed mood or loss of pleasure, had to be present for "more than half of the days" or "almost all the days" and making daily functioning "very difficult" or "extremely difficult".

A patient was defined non-depressed if he/she did not satisfy the criteria for MD or md at any evaluation during the follow-up period. Patients who had at least one MD or md episode over the follow up (regardless of the number of episodes) were classified, respectively, as MD and md. One patient who developed both MD and md episodes, at different time points, was included in the MD group only.

The HADS consists of a 14-item self-administered scale for the evaluation of anxiety and depression in non-psychiatric samples. Each item is rated on a five-point scale ranging from zero to four. The seven items on the depression subscale are largely based on the hedonic state, as five of its items are related to the loss of pleasure. The seven items on anxiety subscale refer mainly to psychic manifestations of anxiety. Therefore, HADS generates two subscale scores: the anxiety score and the depression score. A score of >10 for both subscales defines the presence of depression or anxiety. HADS has been considered more consistent in evaluating depression in ACS patients

for containing less somatic symptoms that could be more readily influenced by health status (Doyle et al., 2006).

DS14 was specifically developed to assess Negative Affectivity (NA) and Social Inhibition (SI). These two subscales, each consisting of seven items, evaluate respectively the tendency to experience feeling of sadness, dysphoria, anxiety and irritability (NA), discomfort in social interactions, lack of social poise, and the tendency to avoid confrontation in social interaction leading to non-expression of emotion (SI). Each item is rated from zero (false) to four (true) on a 5-point Likert-type scale. Scores higher than 10 on both NA and SI indicate the presence of type D personality.

2.3. Treatment

Concerning treatment of depression, only patients with depressive symptoms severe enough (on clinical judgment) to represent a risk for the health (severe inappetence with very low caloric intake, severe insomnia, agitation or suicide ideation) were referred to a psychiatrist and properly treated.

2.4. Overview of statistical analyses

After computing the rates of patients classified as MD, md, and never-depressed over the course of follow-up, the baseline differences between these groups were evaluated using Fisher exact test (F*) for categorical variables (i.e. gender, occupational and educational status, presence of Type D personality) and one-way ANOVA with Bonferroni correction for continuous variables (i.e. age, HADS scores).

To verify whether NA and SI scores at baseline predicted the development of a depressive disorder throughout the 6 months follow-up, a logistic regression (step wise method) was used. In the analysis family status (being widowed), NA and SI scores and their interaction, and HADS scores entered as independent variables, and presence or absence of a depressive disorder entered as dependent variable. We carried out all the analysis using SPSS software (version 20.0, IBM SPSS Statistics).

3. Results

3.1. Patient characteristics

Three hundred four patients met the inclusion criteria, and among them, 289 accepted to participate in the study. During the follow-up period 18 moved outside the study area, 15 refused further psychiatric evaluations, two passed away and 11 continued the rehabilitation treatment in a different hospital. Therefore only 250 were re-evaluated at the follow-up. The study sample included 203 males (81.2%) and 47 females (18.8%) with a mean age of 61.1 ± 11.2 years (range 32–87yrs).

3.2. Depressive disorder

Throughout the follow-up period, MD was diagnosed in 12 patients (4.8%) and md in 18 patients (7.2%), whereas 220 (88%) did not show any depressive symptoms during the 6 months of follow-up. Interestingly, md symptoms were already present at baseline in seven out of 12 patients who developed MD and 12 out of 18 patients who were affected by md during follow-up.

Baseline socio-demographic and clinical characteristic of MD, md and non-depressed patients are shown in Table 1.

3.3. Treatment

Among the MD patients, 10 received a Selective Serotonin Reuptake Inhibitors in the outpatients service, one patient with melancholic features was admitted into our psychiatric ward, while another patient attended a brief day-hospital hospitalization. All the treated patients started a therapy regimen in the month following the onset of symptoms.

3.4. Type D personality

At baseline the rate of TDP was higher in patients with MD and md than in non-depressed patients (Table 1). However, the rate of TDP was especially higher in patients who already showed at baseline

Table 1
Socio-demographic and clinical characteristics of the sample.

Socio-demographic and clinical variables	Major depression n. 12	Minor depression n. 18	No depression n.220		
Gender (male)	8 (66.7%)	12 (66.7%)	183 (83.2%)	F*=4.8	p=.08
Age (years)	64.7 ± 10.7	63.1 ± 12.5	60.7 ± 11.1	F=1.05	p=.34
Education				F*=3.1	p=.79
Primary school	3 (25.0%)	3 (16.7%)	27 (12.3%)		
Secondary school	5 (41.7%)	8 (44.4%)	86 (39.1%)		
College graduated	3 (25.0%)	5 (27.8%)	84 (38.2%)		
University graduated	1 (8.3%)	2 (11.1%)	23 (10.5%)		
Family status				F*=22.9	p < .001
Never married	1 (8.3%)	1 (5.6%)	28 (12.7%)		
Married/Living together	8 (66.7%)	8 (44.1%)	167 (75.9%)		
Separated/Divorced	2 (16.7%)	2 (11.1%)	17 (7.7%)		
Widowed	1 (8.3%)	7 (38.9%)	8(3.6%)		
Occupation				F*=3.3	p=.67
Unemployed	0	1(5.6%)	6(2.7%)		
Retired	6 (50.0%)	9 (50.0%)	101 (45.9%)		
Housewife	0	2 (11.1%)	12 (5.5%)		
Employed	6 (50.0%)	6 (33.3%)	101 (45.9%)		
baseline HADS-D score	10.9 ± 2.8	9.4 ± 3.7	7.4 ± 4.4	F=6.1	p=.003
baseline HADS-A score	10.0 ± 2.1	10.5 ± 4.2	9.9 ± 5.0	F=.13	p=.87
md symptoms present at baseline	7(58.3%)	12 (66.7%)	–	F*=93.1	p < .001
TDP yes at baseline	8 (66.7%)	9(50.0%)	62(28.2%)	F*=10.2	p=.005

Hospital anxiety and depression scale: depression (**HADS-D**), anxiety (**HADS-A**).
md : minor depression.
F*= Fisher exact test.

md symptoms (14 out of 19; 73.7%) than in patients without md (65 out of 231; 28.1%) ($F^* = 15.4$; $df = 1$; $p < .001$). In patients without md at baseline, the same TDP rate was found in those who developed a depressive disorder, both MD and md, (3 out of 11; 27.3%) and those who maintained a non-depressed condition (62 out of 220; 28.2%) ($F^* = .004$; $df = 1$; $p = 1.00$) during follow-up.

Predictors of Depressive Disorders. The logistic regression showed that the baseline HADS-D score increased the risk for a depressive disorder (MD or md) throughout the follow-up period (Table 2). Neither the DS14 NA score nor the DS14 SI score represented a risk factors for a post-ACS depressive disorder (Table 2). The same result was obtained if the presence of TDP (χ^2 wald $= 2.4$; OR 1.91, CI 95%.84–4.68; $p = .11$), rather than the NA and SI scores, was used.

4. Discussion

In this study we evaluated whether TDP predicts the development of depressive disorders in a sample of patients with no history of Major Depression at their first episode of ACS. To our knowledge, this is the first study evaluating these subjects since previous studies included patients with CAD and depression regardless of the duration of their diseases. This means that our findings could not be explained by the effect of long lasting depressive or coronary illnesses, excluding the possibility that our results could not represent a "scar" of the abovementioned disorders, since a previous study demonstrated an association between TDP and a history of depression (Starrenburg et al., 2013).

The different rate of TDP in patients depressed at baseline or who developed depressive symptoms during the follow-up suggest that, in patients developing depressive disorder during follow-up, the presence of md symptoms at baseline may be associated with a high rate of TDP. Moreover, NA and SI scores found at baseline did not predict the development of depressive disorder (both MD and md), if the severity of depressive and anxiety symptoms was controlled. The same result was obtained if the presence of TDP, rather than the NA and SI scores, was considered. Altogether, our findings suggest that, in our patients, NA and SI (or TDP) do not represent a risk factor for developing a depressive disorder after adjusting for the severity of the depressive and anxious symptoms at baseline.

Our results are a confirmation of the long-recognized high association among the NA component of TPD, and the self-reported measures of depressive symptoms (Kudielka et al., 2004; Bergvik et al., 2010; Coyne et al., 2011; Coyne and de Voogd, 2012; Howard

Table 2
Baseline predictor of depressive disorder (both major and minor depression), during follow-up (logistic regression analysis).

Independent variables	B	wald	OR	CI 95%	p
Step 1					
Family status (widowed)	2.52	15.5	12.5	3.5–44.0	< .001
NA	.09	5.9	1.10	1.01–1.18	.01
SI	.08	2.2	1.08	.97–1.21	.13
Step 2					
Family status (widowed)	2.52	15.2	12.4	3.5–44.1	< .001
NA	.14	2.0	1.15	.94–1.41	.15
SI	.12	1.6	1.13	.93–1.38	.20
NA–SI interaction	−.004	.28	.99	.98–1.01	.59
Step 3					
Family status (widowed)	2.38	12.5	10.8	2.9–40.7	< .001
NA	.10	1.0	1.11	.91–1.35	.29
SI	.10	1.0	1.10	.91–1.34	.30
NA–SI interaction	−.003	.16	.99	.98–1.01	.68
HADS-A	−.11	2.5	.89	.78–1.02	.11
HADS-D	.21	6.6	1.23	1.05–1.44	.01

and Hughes, 2012; Starrenburg et al., 2013; Tully and Penninx, 2012). Moreover our study supports the view proposed by Smith (Smith, 2011) that "the version of TDP, which remains after removing the influence of depressive symptoms or anxiety, no longer resembles as closely the construct of original interest".

Some authors suggest that TDP is a stable trait, stress-independent (Denollet, 2005; de Jonge et al., 2007; Kupper et al., 2011; Romppel et al., 2012b), being also not so much influenced by mood changes (Denollet et al., 1996; de Jonge et al., 2007; Denollet and Pedersen, 2008; Yu et al., 2010;).

The results of this study do not support this view. If TDP was a personality trait, then there should be a higher rate in patients who develop a depressive disorder and, therefore, NA and SI scores should predict the onset of depression, after adjusting for the severity of depression and anxiety. These conditions were not found in our patients, since the premorbid rate of TDP in patients who became depressed (27%) was equal to that found in patients who maintained a non-depressed condition during the follow-up period (28%). NA and SI scores did not predict the development of depression after controlling for the severity of anxious and depressive symptoms at baseline. Therefore, our study does not confirm the results of previous studies which suggested that TDP is a risk factor for the development (Pedersen et al., 2006; Kuijpers et al., 2007; Spindler et al., 2009; De Fazio et al., 2012) or co-occurrence (Martens et al., 2008; Smith et al., 2008; Doyle et al., 2011a; Romppel et al., 2012a) of depressive symptoms in cardiac patients.

In most of these previous studies (Kuijpers et al., 2007; Spindler et al., 2009; Dannemann et al., 2010; De Fazio et al., 2012, Starrenburg et al., 2013) TDP and depression were evaluated at the same time; owing to the fact these constructs are highly correlated and partially overlapping (Denollet et al., 2010) no conclusion could reliably be drawn. Indeed in the aforementioned studies the hypothesis has been that TDP is a risk factor for depression, and this is what was tested. However, it was never satisfactorily excluded that depression could also exert an effect on NA and SI levels.

Support for the hypothesis that TDP is a risk factor for depression comes from previous prospective studies (Pedersen et al., 2006; Martens et al., 2008; Smith et al., 2008; Doyle et al., 2011a; Romppel et al., 2012a). However, comparison between our study and these studies is made difficult by the different methodologies used: in fact, none of these studies have included never-depressed patients at baseline, all of them evaluated depressive symptoms (even evaluated from a categorical point of view: i.e. HADS-D cut-off score higher than 7) rather than disorders; most of them (Martens et al., 2008; Smith et al., 2008; Doyle et al., 2011a; Romppel et al., 2012a) evaluated the prediction of stability of depression over time rather than its onset; and some studies (Pedersen et al., 2006; Martens et al., 2008) were not adjusted for the baseline severity of depressive symptoms.

Our results notwithstanding, further larger prospective studies are needed to clarify whether TDP represents a risk factor for depression, controlling for the baseline severity of depressive symptoms.

This study has some limitations. Firstly, the small number of depressed patients in this sample limits the reliability of our results. Nonetheless, such a small sample reflects the strict inclusion criteria of the study. The exclusion of patients with history of coronary artery diseases or depression allowed us to conclude that our findings could not be the effect of long lasting depressive or coronary illnesses and allowed us to infer some conclusion regarding premorbid TDP. Nevertheless, precaution should be used in drawing firm conclusions from our results and the present data needs to be verified by using larger samples.

Secondly, the sample includes mostly male patients, thus limiting this study's applicability to females, who are at higher risk of developing depression. Since a previous study (Hausteiner et al., 2010) demonstrated that the risk for TDP was higher in depressed

man (OR=3.2) than in depressed women (OR=2.4) we can assume that our data could be valid for females too.

In conclusion, our data suggests that TDP (using both categorical and dimensional approaches) does not predict the development of depressive disorders for patients with no previous or current Major Depressive Episodes, on experiencing their first case of ACS when the baseline depression severity is controlled. Our data suggests that in further studies the results of the DS14 need to be controlled for the severity of depressive symptoms beforehand, in order to conclude that TDP is a risk factor for the onset of a depressive disorder.

References

American Psychiatric Association, 1994. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. American Psychiatric Association, Washington, DC:.

Asendorpf, J.B., 1993. Social Inhibition: a general developmental perspective. In: Traue, HC, Pennebaker, JW (Eds.), Emotion, Inhibition and Health. Hogrefe & Huber, Toronto, pp. p80–p99.

Barth, J., Schumacher, M., Herrmann-Lingen, C., 2004. Depression as a risk factor for mortality in patients with coronary heart disease: a meta-analysis. Psychosom. Med. 66, 802–813.

Baumeister, H., Hutter, N., Bengel, J., Härter, M., 2011. Quality of life in medically ill persons with comorbid mental disorders: a systematic review and meta-analysis. Psychother. Psychosom. 80, 275–286.

Baumeister, H., Kriston, L., Bengel, J., Härter, M., 2010. High agreement of self-report and physician-diagnosed somatic conditions yields limited bias in examining mental-physical comorbidity. J. Clin. Epidemiol. 63, 558–565.

Bergvik, S., Sørlie, T., Wynn, R., Sexton, H., 2010. Psychometric properties of the Type D scale (DS14) in Norwegian cardiac patients. Scand. J. Psychol. 51, 334–340.

Carney, RM., 1998. Psychological risk factors for cardiac events: could there be just one? Circulation 97, 128–129.

Coyne, J.C., de Voogd, J.N., 2012. Are we witnessing the decline effect in the Type D personality literature? What can be learned?. J. Psychosom. Res. 73, 401–407.

Coyne, J.C., Jaarsma, T., Luttik, M.L., van Sonderen, E., van Veldhuisen, D.J., Sanderman, R., 2011. Lack of prognostic value of type D personality for mortality in a large sample of heart failure patients. Psychosom. Med. 73, 557–562.

Dannemann, S, Matschke, K, Einsle, F, Smucker, MR, Zimmermann, K, Joraschky, P, Weidner, K, Köllner, V., 2010. Is type-D a stable construct? An examination of type-D personality in patients before and after cardiac surgery. J. Psychosom. Res. 69, 101–109.

De Fazio, P., Caroleo, M., Rizza, P., Cerminara, G., De Serio, D., Indolfi, C., Segura-García, C., 2012. Specific personality traits and coping styles predict affective symptoms in early post acute coronary syndrome inpatients. Int. J. Psychiatry Med. 44, 119–132.

de Jonge, P., Denollet, J., van Melle, J.P., Kuyper, A., Honig, A., Schene, A.H., Ormel, J., 2007. Associations of type-D personality and depression with somatic health in myocardial infarction patients. J. Psychosom. Res. 63, 477–482.

de Jonge, P., van den Brink, R.H., Spijkerman, T.A., Ormel, J., 2006. Only incident depressive episodes after myocardial infarction are associated with new cardiovascular events. J. Am. Coll. Cardiol. 48, 2204–2208.

Denollet, J., Conraads, V.M., 2011. Type D personality and vulnerability to adverse outcomes in heart disease. Clevel. Clin. J. Med. 78 (Suppl 1), S13–S19.

Denollet, J., Pedersen, S.S., 2008. Prognostic value of Type D personality compared with depressive symptoms. Arch. Intern. Med. 168, 431–432.

Denollet, J., Schiffer, A.A., Spek, V., 2010. A general propensity to psychological distress affects cardiovascular outcomes: evidence from research on the type D (distressed) personality profile. Circ. Cardiovasc. Q. Outcomes 3, 546–557.

Denollet, J., Sys, S.U., Stroobant, N., Rombouts, H., Gillebert, T.C., Brutsaert, D.L., 1996. Personality as independent predictor of long-term mortality in patients with coronary heart disease. Lancet 347, 417–421.

Denollet, J., 2005. DS14: standard assessment of negative affectivity, social inhibition, and Type D personality. Psychosom. Med. 67, 89–97.

Doyle, F., Conroy, R., McGee, H., 2007. Challenges in reducing depression-related mortality in cardiac populations: cognition, emotion, fatigue or personality? Health Psychol. Rev. 1, 137–172.

Doyle, F., McGee, H., Delaney, M., Motterlini, N., Conroy, R., 2011a. Depressive vulnerabilities predict depression status and trajectories of depression over 1 year in persons with acute coronary syndrome. Gen. Hosp. Psychiatry 33, 224–231.

Doyle, F., McGee, H.M., Conroy, R.M., Delaney, M., 2011b. What predicts depression in cardiac patients: sociodemographic factors, disease severity or theoretical vulnerabilities? Psychol. Health 26, 619–634.

Doyle, F., McGee, H.M., De La Harpe, D., Shelley, E., Conroy, R., 2006. The Hospital Anxiety and Depression Scale depression subscale, but not the Beck Depression Inventory-Fast Scale, identifies patients with acute coronary syndrome at elevated risk of 1-year mortality. J. Psychosom. Res. 60, 461–467.

Folstein, M.F., Folstein, S.E., McHugh, P.R., 1975. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. Psychiatr. Res. 12, 189–198.

Frasure-Smith, N., Lespérance, F., Gravel, G., Masson, A., Juneau, M., Talajic, M., Bourassa, M.G., 2000. Depression and health-care costs during the first year following myocardial infarction. J. Psychosom. Res. 48, 471–478.

Frasure-Smith, N., Lespérance, F., 2010. Depression and cardiac risk: present status and future directions. Heart 96, 173–176.

Frasure-Smith, N., Lespérance, F., 2005. Reflections on depression as a cardiac risk factor. Psychosom. Med. 67 (Suppl 1), S19–S25.

Gest, S.D., 1997. Behavioral inhibition: stability and associations with adaptation from childhood to early adulthood. J. Personal. Soc. Psychol. 72, 467–475.

Härter, M., Baumeister, H., Reuter, K., Jacobi, F., Höfler, M., Bengel, J., Wittchen, H.U., 2007. Increased 12-month prevalence rates of mental disorders in patients with chronic somatic diseases. Psychother. Psychosom. 76, 354–360.

Hausteiner, C., Klupsch, D., Emeny, R., Baumert, J., Ladwig, K.H., 2010. Clustering of negative affectivity and social inhibition in the community: prevalence of type D personality as a cardiovascular risk marker. Psychosom. Med. 72, 163–171.

Howard, S., Hughes, B.M., 2012. Construct, concurrent and discriminant validity of Type D personality in the general population: associations with anxiety, depression, stress and cardiac output. Psychol. Health 27, 242–258.

Kaptein, K.I., de Jonge, P., van den Brink, R.H., Korf, J., 2006. Course of depressive symptoms after myocardial infarction and cardiac prognosis: a latent class analysis. Psychosom. Med. 68, 662–668.

Kudielka, B.M., von Känel, R., Gander, M.L., Fischer, J.E., 2004. The interrelationship of psychosocial risk factors for coronary artery disease in a working population: do we measure distinct or overlapping psychological concepts? Behav. Med. 30, 35–43.

Kuijpers, P.M., Denollet, J., Wellens, H.J., Crijns, H.M., Honig, A., 2007. Noncardiac chest pain in the emergency department: the role of cardiac history, anxiety or depression and Type D personality. Eur. J. Cardiovasc. Prev. Rehabil. 14, 273–279.

Kupper, N., Boomsma, D.I., de Geus, E.J., Denollet, J., Willemsen, G., 2011. Nine-year stability of type D personality: contributions of genes and environment. Psychosom. Med. 73, 75–82.

Kupper, N., Denollet, J., de Geus, E.J., Boomsma, D.I., Willemsen, G, 2007. Heritability of type-D personality. Psychosom. Med. 69, 675–681.

Lespérance, F., Frasure-Smith, N., 1996. Negative emotions and coronary heart disease: getting to the heart of the matter. Lancet 347, 414–418.

Martens, E.J., Kupper, N., Pedersen, S.S., Aquarius, A.E., Denollet, J., 2007. Type-D personality is a stable taxonomy in post-MI patients over an 18-month period. J. Psychosom. Res. 63, 545–550.

Martens, E.J., Smith, O.R.F., Winter, J., Denollet, J., Pedersen, S.S., 2008. Cardiac history, prior depression and personality predict course of depressive symptoms after myocardial infarction. Psychol. Med. 38, 257–264.

McGee, H.M., Doyle, F., Conroy, R.M., De La Harpe, D., Shelley, E., 2006. Impact of briefly-assessed depression on secondary prevention outcomes after acute coronary syndrome: a one-year longitudinal survey. BMC Health Serv. Res. 6, 9.

Nicholson, A., Kuper, H., Hemingway, H., 2006. Depression as an aetiologic and prognostic factor in coronary heart disease: a meta-analysis of 6362 events among 146 538 participants in 54 observational studies. Eur. Heart J. 27, 2763–2774.

Ormel, J., Von Korff, M., Burger, H., Scott, K., Demyttenaere, K., Huang, Y.Q., Posada-Villa, J., Pierre Lepine, J., Angermeyer, M.C., Levinson, D., de Girolamo, G., Kawakami, N., Karam, E., Medina-Mora, M.E., Gureje, O., Williams, D., Haro, J. M., Bromet, E.J., Alonso, J., Kessler, R., 2007. Mental disorders among persons with heart disease - results from World Mental Health surveys. Gen. Hosp. Psychiatry 29, 325–334.

Parashar, S., Rumsfeld, J.S., Spertus, J.A., Reid, K.J., Wenger, N.K., Krumholz, H.M., Amin, A., Weintraub, W.S., Lichtman, J., Dawood, N., Vaccarino, V., 2006. Time course of depression and outcome of myocardial infarction. Arch. Intern. Med. 166, 2035–2043.

Parker, G.B., Hilton, T.M., Walsh, W.F., Owen, C.A., Heruc, G.A., Olley, A., Brotchie, H., Hadzi-Pavlovic, D., 2008. Timing is everything: the onset of depression and acute coronary syndrome outcome. Biol. Psychiatry 64, 660–666.

Pedersen, S.S., Ong, A.T., Sonnenschein, K., Serruys, P.W., Erdman, R.A., van Domburg, R.T., 2006. Type D personality and diabetes predict the onset of depressive symptoms in patients after percutaneous coronary intervention. Am. Heart J. 151 (367), e1–e6.

Pelle, A.J., Denollet, J., Zwisler, A.D., Pedersen, S.S., 2009. Overlap and distinctiveness of psychological risk factors in patients with ischemic heart disease and chronic heart failure: are we there yet? J. Affect. Disord. 113, 150–156.

Romppel, M., Herrmann-Lingen, C., Vesper, J.M., Grande, G., 2012a. Type D personality and persistence of depressive symptoms in a German cohort of cardiac patients. J. Affect. Disord. 136, 1183–1187.

Romppel, M., Herrmann-Lingen, C., Vesper, J.M., Grande, G., 2012b. Six year stability of Type-D personality in a German cohort of cardiac patients. J. Psychosom. Res. 72, 136–141.

Rudisch, B., Nemeroff, C.B., 2003. Epidemiology of comorbid coronary artery disease and depression. Biol. Psychiatry 54, 227–240.

Smith, O.R., Pedersen, S.S., Van Domburg, R.T., Denollet, J., 2008. Symptoms of fatigue and depression in ischemic heart disease are driven by personality characteristics rather than disease stage: a comparison of CAD and CHF patients. Eur. J. Cardiovasc. Prev. Rehabil. 15, 583–588.

Smith, T.W., 2011. Toward a more systematic, cumulative, and applicable science of personality and health: lessons from type D personality. Psychosom. Med. 73, 528–532.

Spijkerman, T., de Jonge, P., van den Brink, R.H., Jansen, J.H., May, J.F., Crijns, H.J., Ormel, J., 2005. Depression following myocardial infarction: first-ever versus ongoing and recurrent episodes. Gen. Hosp. Psychiatry 27, 411–417.

Spindler, H., Kruse, C., Zwisler, A.D., Pedersen, S.S., 2009. Increased anxiety and depression in Danish cardiac patients with a type D personality: cross-validation of the Type D Scale (DS14). Int. J. Behav. Med. 16, 98–107.

Spitzer, R.L., Kroenke, K., Williams, J.B.W., 1999. Validation and utility of a self-report version of PRIME-MD: the PHQ Primary Care Study. JAMA 282, 1737–1744.

Spitzer, R.L., Williams, J.B.W., Kroenke, K., Linzer, M., deGruy 3rd, F.V., Hahn, S.R., Brody, D., Johnson, J.G., 1994. Utility of a new procedure for diagnosing mental disorders in primary care: the PRIME-MD 1000 study. JAMA 272, 1749–1756.

Starrenburg, A.H., Kraaier, K., Pedersen, S.S., van Hout, M., Scholten, M., van der Palen, J., 2013. Association of psychiatric history and type D personality with symptoms of anxiety, depression, and health status prior to ICD implantation. Int. J. Behav. Med. 20, 425–433.

Svansdottir, E., van den Broek, K.C., Karlsson, H.D., Gudnason, T., Denollet, J., 2012. Type D personality is associated with impaired psychological status and unhealthy lifestyle in Icelandic cardiac patients: a cross-sectional study. BMC Public Health 12, 42.

Thombs, B.D., Bass, E.B., Ford, D.E., Stewart, K.J., Tsilidis, K.K., Patel, U., Fauerbach, J.A., Bush, D.E., Ziegelstein, R.C., 2006. Prevalence of depression in survivors of acute myocardial infarction. J. Gen. Intern. Med. 21, 30–38.

Tully, P.J., Penninx, B.W., 2012. Depression and anxiety among coronary heart disease patients: can affect dimensions and theory inform diagnostic disorder-based screening? J. Clin Psychol., http://dx.doi.org/10.1002/jclp.21828.

Watson, D., Pennebaker, J.W., 1989. Health complaints, stress, and distress: exploring the central role of negative affectivity. Psychol. Rev. 96, 234–254.

Yu, D.S., Thompson, D.R., Yu, C.M., Pedersen, S.S., Denollet, J., 2010. Validating the type D personality construct in Chinese patients with coronary heart disease. J. Psychosom. Res. 69, 111–118.

Zigmond, A.S., Snaith, R.P., 1983. The hospital anxiety and depression scale. Acta Psychiatr. Scand. 67, 361–370.