

Clinical Events in Coronary Patients Who Report Low Distress: Adverse Effect of Repressive Coping

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Objective: Coronary artery disease (CAD) patients who report low distress are considered to be at low psychological risk for clinical events. However, patients with a repressive coping style may fail to detect and report signals of emotional distress. The authors hypothesized that repressive CAD patients are at risk for clinical events, despite low self-rated distress. **Design:** This was a prospective 5- to 10-year follow-up study, with a mean follow-up of 6.6 years. At baseline, 731 CAD patients filled out Trait-Anxiety (distress), Marlowe-Crowne (defensiveness), and Type D scales; 159 patients were classified as “repressive,” 360 as “nonrepressive,” and 212 as “Type D.” **Main Outcome Measures:** The primary endpoint was a composite of total mortality or myocardial infarction (MI); the secondary endpoint was cardiac mortality/MI. **Results:** No patients were lost to follow-up; 91 patients had a clinical event (including 35 cardiac death and 32 MI). Repressive patients reported low levels of anxiety, anger and depression at baseline, but were at increased risk for death/MI (21/159 = 13%) compared with nonrepressive patients (22/360 = 6%), $p = .009$. Poor systolic function, poor exercise tolerance, 3-vessel disease, index MI and Type-D personality—but not depression, anxiety or anger—also independently predicted clinical events. After controlling for these variables, repressive patients still had a twofold increased risk of death/MI, $OR = 2.17$, 95% $CI = 1.10–4.08$, $p = .025$). These findings were replicated for cardiac mortality/MI. **Conclusion:** CAD patients who use a repressive coping style are at increased risk for clinical events, despite their claims of low emotional distress. This phenomenon may cause an underestimation of the effect of stress on the heart.

Keywords: CAD, depression, Type-D personality, repressive coping, prognosis

Depression is a frequent comorbidity in medical patients (Whooley & Simon, 2000), and emotional distress associated with depression and anxiety has been related to coronary artery disease (CAD) (Barth, Schumacher, & Herrmann-Lingen, 2004; Creed, 1999; de Jonge et al., 2006; Strik, Denollet, Lousberg, & Honig, 2003). As a consequence, CAD patients who report little distress are considered to be at low risk for clinical events. However,

individuals greatly differ in their threshold for responding to negative stimuli with self-reports of emotional distress (Gross, 2002) and in their ability to recognize emotional stimuli (Lane, Sechrest, Riedel, Shapiro, & Kaszniak, 2000). Individuals with a repressive coping style typically report low levels of emotional distress (Broomfield & Turpin, 2005; Denollet, 1991; Derakshan & Eysenck, 1997; Giese-Davis, Sephton, Abercrombie, Duran, & Spiegel, 2004; Grossman, Watkins, Ristuccia, & Wilhelm, 1997; Lane, Merikangas, Schwartz, Huang, & Prusoff, 1990, 2000; Movius & Allen, 2005; Myers, Brewin, & Power, 1998; Pauls & Stemmler, 2003; Rutledge & Linden, 2003).

Repressive coping is the tendency to avoid/repress negative emotions (Rutledge & Linden, 2003). It protects against psychiatric disorder (Lane et al., 1990), but is also associated with less accurate detection of sadness/fear (Lane et al., 2000), less eye movements toward threatening stimuli (Broomfield & Turpin, 2005), increased blood pressure (Grossman et al., 1997), decreased heart rate variability (Movius & Allen, 2005; Pauls & Stemmler, 2003), cortisol dysregulation (Giese-Davis et al., 2004), and poor adherence to health recommendations (Jamner, Schwartz, & Leigh, 1988; Levine et al., 1987). Hence, repressive individuals may show overt behavioral and physiological signs of distress despite their claims of low distress.

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We previously hypothesized that repressive patients' tendency to minimize distress causes the underdiagnosis of high-risk CAD patients with low levels of distress (Denollet, 1991). Hence, research may underestimate the effect of stress on the heart; patients reporting little distress typically have been assigned a low-risk status whereas, in fact, the low-risk reference group in these studies may include a subgroup of "false negative" patients who use a repressive coping style. The aim of the present 5- to 10-year prospective follow-up study was to test this a priori hypothesis. We predicted that, given their tendency to be unaware of internal signals of distress, repressive CAD patients would be at risk for clinical events.

Method

Patients

This study involves 731 CAD patients (656 men/75 women; $M = 56.0 \pm 8.0$ years) from the University Hospital of Antwerp. They were recruited in two studies that were designed to examine the effect of emotional distress and repressive coping on cardiac prognosis. The design of both studies was similar; methodological details were described previously (Denollet et al., 1996, Denollet, Vaes, & Brutsaert, 2000). In the first study, 303 CAD patients (Denollet et al., 1996) were screened for decreased left ventricular ejection fraction (LVEF) with ventricular angiography; the study included an additional 106 patients (Denollet, 1993) with echocardiographic screening of LVEF. In the second study, a new sample of 322 CAD patients (Denollet et al., 2000) was examined. The pooled data from these studies included 392 patients (54%) with a myocardial infarction (MI) and 535 (73%) who had coronary artery bypass graft surgery (CABG) or percutaneous coronary intervention (PCI), with 196 patients experiencing both a MI and invasive intervention. Patients with other life-threatening diseases were excluded. At baseline, all patients had an exercise stress test and provided informed consent. The study was approved by the local hospital ethics committee.

Assessment

Repressive coping. The Marlowe-Crowne Social Desirability (MCSD) scale (Crowne & Marlowe, 1960) has been shown to be a valid defensiveness scale in a number of experimental and clinical studies (Broomfield & Turpin, 2005; Denollet, 1991; Derakshan & Eysenck, 1997; Giese-Davis et al., 2004; Grossman et al., 1997; Lane et al., 1990, 2000; Movius & Allen, 2005; Pauls & Stemmler, 2003). Weinberger, Schwartz, and Davidson (1979) provided construct validity for distinctions among low-anxious, high-anxious, and repressive styles as three general patterns of coping with threatening situations.

The State Trait Anxiety Inventory (STAI) is a self-report measure consisting of two scales developed to measure the level of general state and trait anxiety (van der Ploeg, Defares, & Spielberger, 1980). In the current study we included the trait scale of the STAI. The STAI has been demonstrated to have adequate validity and reliability (Knight, Waal-Manning, & Spears, 1983).

Patients who avoid/repress negative emotions typically score low on the STAI (van der Ploeg et al., 1980) but high on the MCSD (Crowne & Marlowe, 1960) scale. Both measures were

used in this study to define repressive coping. According to previously published cutoff scores (Denollet, 1991), 159 CAD patients (22%) were classified as repressive (STAI ≤ 42 and MCSD ≥ 22).

End points. According to previous research on the effect of emotional distress and its treatment in CAD (Schneiderman et al., 2004), the primary endpoint was a composite of total mortality or nonfatal MI. The secondary endpoint was cardiac mortality or nonfatal MI. As described previously (Denollet et al., 1996, 2000), mortality/MI data were derived from hospital records and the patient's attending physician was involved in the classification of cause of death. The follow-up interval varied between 5 and 10 years ($M = 6.6 \pm 1.7$ y).

Risk factors. LVEF, exercise tolerance and extent of CAD are indices of disease severity that are powerful predictors of clinical events in this sample of CAD patients (Denollet et al., 1996, 2000). Overtly impaired ($\leq 44\%$) but also borderline decreased (45%–54%) LVEF have been associated with poor prognosis (Gottdiener et al., 2002); decreased systolic function was defined as LVEF $\leq 54\%$. Poor exercise tolerance is associated with progression of atherosclerosis (Lakka et al., 2001) and was defined by a median split for peak work load on a symptom-limited exercise test (i.e., ≤ 140 and ≤ 120 W for younger and older men; ≤ 100 and ≤ 80 W for younger and older women, respectively). A great extent of CAD was defined as 3 vessels with $\geq 70\%$ reduction in internal diameter. We also controlled for age, sex, Type-D personality, and clinical indices of cardiac risk. Clinical indices included an index MI, anterior MI, CABG or PCI, β -blocker and angiotensin-converting enzyme (ACE)-inhibitor therapy, hypertension, hyperlipidemia, and smoking.

Anxiety, depression, and anger. Repressive patients were hypothesized to score in the nondistressed range of the State Anxiety (van der Ploeg et al., 1980), HPPQ Depression scale (Erdman, Duivendoorn, Verhage, Kazemier, & Hugenholtz, 1986) and Trait Anger (van der Ploeg, Defares, & Spielberger, 1982) scales; these scales have previously been used to document the role of emotional distress on cardiac prognosis (Denollet & Brutsaert, 1998). Patients were distressed if they scored in the upper quartile; that is, ≥ 49 on the anxiety scale, ≥ 22 on the depression scale, and ≥ 21 on the anger scale.

Distressed personality (Type-D). We previously showed that Type-D patients from the current sample are at increased risk for clinical events (Denollet et al., 1996, 2000). According to the cutoff scores on the STAI (van der Ploeg et al., 1980) and Heart Patients Psychological Questionnaire (HPPQ) Social Inhibition (Erdman et al., 1986) scales used in these studies, 212 patients (29%) in the current study were classified as Type-D (STAI ≥ 43 and HPPQ ≥ 12). In contrast to repressive patients, Type-D patients are well aware of their level of emotional distress as indicated by high scores on distress measures. Therefore, repressive coping would also have to predict clinical events above and beyond the effect of Type-D personality (Denollet et al., 1996, 2000).

Reference group. Patients with a nonrepressive coping style were used as a reference group, and included both (a) patients with low scores on both trait-anxiety and defensiveness (STAI ≤ 42 and MCSD ≤ 21), and (b) patients with a high score on trait-anxiety but a low score on social inhibition (STAI ≥ 43 and HPPQ ≤ 11). These patients were conceptualized as nonrepressive because their

Table 1
Baseline Measures of Emotional Distress According to Death/MI at Follow-up

Baseline measures	Event-free (<i>N</i> = 640)	Death/MI (<i>N</i> = 91)	Odds ratio [95% confidence interval]	<i>p</i>
Univariate analyses				
Negative emotions				
Depression	25% (161)	35% (32)	1.61 [1.01–2.57]	.044
Anxiety	25% (160)	31% (28)	1.33 [0.83–2.15]	.24
Anger	24% (152)	30% (27)	1.35 [0.83–2.20]	.22
Personality				
Type-D personality	26% (164)	53% (48)	3.24 [2.07–5.07]	.0001
Multivariable analysis				
Predictors of death/MI (<i>n</i> = 91)				
Depression			1.15 [0.63–2.10]	.66
Anxiety				.24
Anger			1.04 [0.60–1.79]	.89
Type-D personality			3.51 [2.12–5.83]	.0001

Note. MI = myocardial infarction. Number of subjects appears in parentheses.

strategy for regulating emotions is in accordance with their levels of distress based on their self-reports.

Statistical Analyses

T test, χ^2 test, and logistic regression were used to analyze differences in baseline characteristics as a function of repressive coping. Multivariable logistic regression analysis was used to examine the effect of disease severity on 5- to 10-year prognosis. Logistic regression models were also constructed to investigate the prognostic value of repressive coping in addition to disease severity and Type-D personality. All variables were entered simultaneously in these final regression models. Analyses were performed using SPSS for Windows version 12.0.

Results

No patients were lost to follow-up; 91 patients (12%) had a clinical event (death or MI), and 67 had a cardiac event (35 cardiac death and 32 MI). All deaths were attributable to natural causes. The rate of clinical events of patients who score low on both trait-anxiety/ defensiveness (15/206 = 7%) and those who score

high on trait-anxiety but low on social inhibition (7/154 = 5%) was not significantly different, $p = .28$. Hence, merging these subgroups into one reference group ($n = 360$; 49% of total sample) of nonrepressive patients was warranted.

At baseline, repressive patients were less likely to have suffered a MI compared with nonrepressive patients (50% vs. 61%, $p = .014$). In addition, they were somewhat older (57.5 ± 7.8 vs. 55.6 ± 8.0 year, $p = .007$) and more likely to have been treated invasively (81% vs. 71% $p = .011$) because of 3-vessel disease. They did not differ from nonrepressive patients in terms of gender, LVEF, exercise tolerance, anterior MI, use of β -blockers and ACE-inhibitors, hypertension, hyperlipidemia, or smoking. Given their tendency to repress signals of distress, repressive patients were hypothesized to claim to experience no distress. In this study, they were significantly less likely to report distress compared with other patients (see Figure 1). Only a small minority of repressive patients was in the distressed range of anxiety, anger ($n = 4$) or depression ($n = 5$) as opposed to one third of the other patients.

Repressive patients had more than 2 times the risk for death/MI as compared to nonrepressive patients (Figure 2, left). This adverse effect of repressive coping was not accounted for by demographic differences ($p = .009$, after adjustment for age and gender). These findings were replicated when using cardiac events as an endpoint (Figure 2, right); that is, repressive patients had an increased risk of cardiac death/MI, adjusting for age and gender ($p = .018$).

Symptoms of depression and Type-D personality, but not anxiety or anger, predicted poor prognosis in univariate analysis (see Table 1). Patients who reported depressive symptoms had a 60% increase in risk. As reported previously (Denollet et al., 1996, 2000), Type-D patients had a more than threefold increased risk of clinical events (23%) as compared to non-Type-Ds (8%). A multivariable regression model indicated that Type-D personality was the only measure of emotional distress that independently predicted prognosis (Table 1, bottom).

In order to examine whether the prognostic role of repressive coping was independent of the effects of Type-D, depression, anxiety and anger, all of these variables were entered simultaneously in a logistic regression model. Repressive coping and Type-D personality were retained as independent predictors of death/MI, while depression, anxiety and anger were not (see Table

Table 2
Repressive Coping Versus Emotional Distress as Independent Predictor

Clinical endpoint	Odds ratio [95% confidence interval]	<i>p</i>
Death/MI (<i>n</i> = 91)		
Repressive coping	2.39 [1.25–4.56]	.009
Type-D personality	4.63 [2.60–8.27]	.0001
Depression	1.20 [0.65–2.23]	.56
Anxiety	0.73 [0.39–1.35]	.73
Anger	1.16 [0.66–2.04]	.60
Cardiac events (<i>n</i> = 67)		
Repressive coping	2.35 [1.11–4.99]	.026
Type-D personality	4.73 [2.44–9.14]	.0001
Depression	1.24 [0.62–2.51]	.55
Anxiety	0.74 [0.37–1.48]	.39
Anger	1.15 [0.61–2.17]	.68

Note. MI = myocardial infarction.

Table 3
Demographic/Clinical Characteristics According to Death/MI at Follow-up

Baseline Characteristics	Event-free (N = 640)	Death/MI (N = 91)	Odds ratio [95% confidence interval]	p
Univariate analyses				
Demographic				
Age (mean ± SD)	56.0 ± 8.0 y	55.8 ± 7.9 y	0.99 [0.97–1.02]	.781
Gender (male)	90% (574)	90% (82)	1.05 [0.50–2.18]	.901
Disease severity				
Decreased LVEF ^a	27% (174)	47% (43)	2.40 [1.54–3.75]	.0001
Poor exercise tolerance ^b	35% (225)	63% (57)	3.09 [1.96–4.87]	.0001
Three-vessel disease	33% (214)	52% (47)	2.13 [1.37–3.31]	.001
Index MI at baseline	57% (367)	69% (63)	1.67 [1.04–2.68]	.032
Anterior MI	24% (152)	33% (30)	1.58 [0.98–2.54]	.059
Clinical variables				
CABG/PCI	74% (477)	64% (58)	0.60 [0.38–0.95]	.031
β-blocker therapy	52% (331)	53% (48)	1.04 [0.67–1.62]	.854
ACE-inhibitors	6% (36)	6% (5)	0.98 [0.37–2.55]	.961
Hypertension	27% (173)	25% (23)	1.04 [0.72–1.50]	.832
Hyperlipidemia	33% (213)	31% (28)	0.89 [0.55–1.43]	.634
Smoking	20% (126)	23% (21)	1.22 [0.72–2.07]	.451
Multivariable analysis				
Predictors of death/MI (n = 91)				
Decreased LVEF			1.95 [1.19–3.17]	.008
Poor exercise tolerance			2.84 [1.76–4.58]	.0001
Three-vessel disease			2.19 [1.35–3.55]	.001
Index MI at baseline			1.94 [1.13–3.34]	.016
Predictors of cardiac events (n = 67)				
Decreased LVEF			2.38 [1.37–4.13]	.002
Poor exercise tolerance			2.73 [1.59–4.71]	.0001
Three-vessel disease			1.96 [1.13–3.41]	.017
Index MI at baseline			2.26 [1.19–4.32]	.013

Note. Number of subjects appears in parentheses. MI = myocardial infarction; LVEF = left ventricular ejection fraction; CABG = coronary artery bypass surgery; PCI = percutaneous coronary intervention; ACE = angiotensin-converting enzyme.

^aLeft ventricular ejection fraction ≤54%.²⁵

^b≤140/≤120 Watt for younger/older men; ≤100/≤80 Watt for younger/older women.

2). These findings were replicated when using continuous scores of depression, anxiety and anger as predictors. Furthermore, repressive coping and Type-D personality were independent predictors of cardiac death/MI as a secondary endpoint (Table 2, bottom).

Patients who died or had a nonfatal MI also differed from patients with an event-free survival in several medical characteristics at baseline. They were more likely to perform worse on markers of disease severity, and less likely to have been treated invasively with CABG/PCI (see Table 3). Multivariable logistic regression analysis yielded decreased LVEF, poor exercise tolerance, three-vessel disease and index MI as independent predictors of death/MI (Table 3, bottom). These same indicators of disease severity also predicted cardiac events.

To determine whether repressive coping was an independent psychological predictor of death/MI, repressive coping, Type-D and disease severity were all entered simultaneously in a regression model. Repressive coping was associated with a twofold increased risk of death/MI after adjustment for disease severity, and predicted clinical events above and beyond Type-D personality (see Table 4). Repressive coping also independently predicted cardiac death/MI (Table 4, bottom). Decreased LVEF, poor exercise tolerance, three-vessel disease, and index MI remained independent predictors of death/MI and cardiac events in these final analyses.

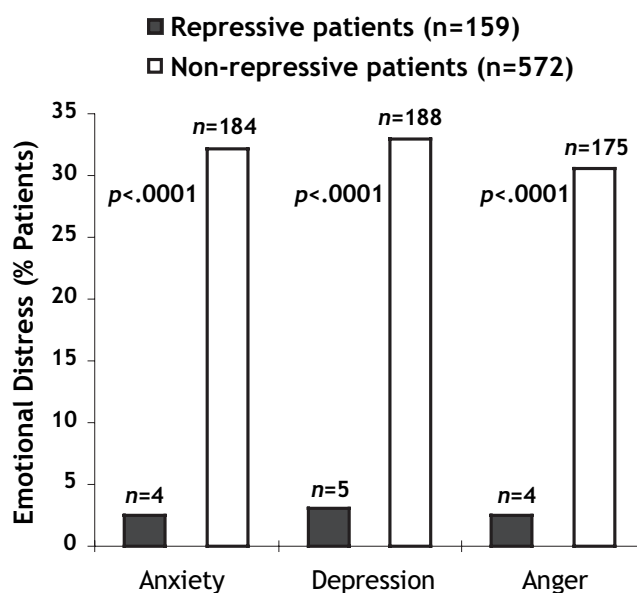


Figure 1. Lack of emotional distress in repressive patients, as indicated by very small percentages of these patients reporting anxiety, depression or anger. Number of patients who score in the distressed range of anxiety, depression or anger are presented on top of each bar.

Table 4
Repressive Coping as Independent Predictor of 5- to 10-Year Prognosis

Clinical endpoint	Odds ratio [95% confidence interval]	<i>p</i>
Death/MI (<i>n</i> = 91)		
Repressive coping	2.17 [1.10–4.08]	.025
Gender (male)	1.21 [0.55–2.66]	.639
Age	0.98 [0.95–1.01]	.269
Type-D personality	3.80 [2.17–6.64]	.0001
Decreased LVEF ^a	1.81 [1.10–3.00]	.021
Poor exercise tolerance ^b	2.63 [1.61–4.31]	.0001
Three-vessel disease	2.22 [1.33–3.68]	.002
Index MI at baseline	1.89 [1.09–3.28]	.024
Cardiac events (<i>n</i> = 67)		
Repressive coping	2.16 [1.01–4.65]	.047
Gender (male)	2.17 [0.72–6.54]	.168
Age	0.97 [0.94–1.00]	.074
Type-D personality	3.96 [2.08–7.53]	.0001
Decreased LVEF ^a	2.23 [1.27–3.94]	.006
Poor exercise tolerance ^b	2.56 [1.46–4.49]	.001
Three-vessel disease	2.01 [1.12–3.61]	.020
Index MI at baseline	2.14 [1.11–4.13]	.023

MI = acute myocardial infarction; LVEF = left ventricular ejection fraction.

^aLeft ventricular ejection fraction $\leq 54\%$.²⁵

^b $\leq 140/\leq 120$ Watt for younger/older men; $\leq 100/\leq 80$ Watt for younger/older women.

Discussion

The findings of this study clearly showed that repressive CAD patients were at a twofold increased risk of long-term mortality or MI, despite their claim to experience low levels of distress. These findings were confirmed after adjustment for depression, anxiety, anger, Type-D personality, and severity of the cardiac disease, and were replicated when looking at cardiac events as a secondary endpoint.

Accumulating evidence indicates that individuals who experience feelings of distress are at risk for clinical events, including MI and cardiac death (Barth et al., 2004; Creed, 1999; de Jonge et al., 2006; Strik et al., 2003). As a consequence, there is an implicit assumption that patients scoring low on distress measures can be regarded as low-risk individuals in terms of stress-related CAD. However, our findings suggest that this assumption may not apply to a subgroup of CAD patients who use a repressive coping style. Although repressive patients reported very low levels of depression, anger, and anxiety, they had an increased risk of 5- to 10-year clinical events.

The present findings should be interpreted with some caution. There were significant differences on several baseline characteristics between repressive and other patients but we did control for these differences in multivariable analyses. The present findings may not generalize to women, as female patients only comprised a minority of the sample. Finally, defensiveness was assessed by the MCSD scale that has been shown to detect susceptibility to unconscious forms of distress in experimental research (Broomfield & Turpin, 2005; Grossman et al., 1997; Lane et al., 1990; Movius & Allen, 2005; Pauls & Stemmler, 2003); the use of other measurement tools (physiological measurement, brain imaging, behavioral assessment) is needed to further examine the role

of unconscious emotions in the clinical course of CAD. However, the present findings confirm our a priori hypothesis (Denollet, 1991) that repressive coping would predict clinical events in patients who report low levels of distress but score high on the tendency to minimize this distress.

Our findings may have important implications for clinical research and practice. Considering the impact for clinical research, these findings suggest that the adverse effect of stress and negative emotions may have been underestimated in previous research. Individuals reporting little distress typically have been assigned a low-risk status whereas, in fact, the low-risk reference group in these studies includes a subgroup of “false negative” individuals who are characterized by repressive coping. The tendency of repressive patients to report low levels of distress may go some way toward explaining the lack of an association between self-reported distress and cardiac events in some studies (Dickens et al., 2004).

A number of behavioral and physiological pathways may explain the worse clinical outcome in repressive patients. For example, repressive coping has been associated with self-reports of medication allergies (Jamner et al., 1988) and poorer recovery in the year following discharge in CAD patients: repressive patients were more noncompliant with medical recommendations and required more days of rehospitalization (Levine et al., 1987). Further, repressive individuals have been found to exhibit a less favorable cardiovascular function than nonrepressives, putatively being the consequence of a hyperresponsiveness of the sympathetic nervous system to stressful events. They have shown elevated heart rate and blood pressure responses to laboratory stressors (Derakshan & Eysenck, 1997; Grossman et al., 1997), enhanced reductions of heart rate variability (Pauls & Stemmler, 2003), and cortisol dysregulation (Giese-Davis et al., 2004). Repressive coping is also associated with elevated ambulatory blood pressure (Nyklicek, Vingerhoets, & van Heck, 1999) and has been shown to predict the incidence of hypertension (Rutledge & Linden, 2000).

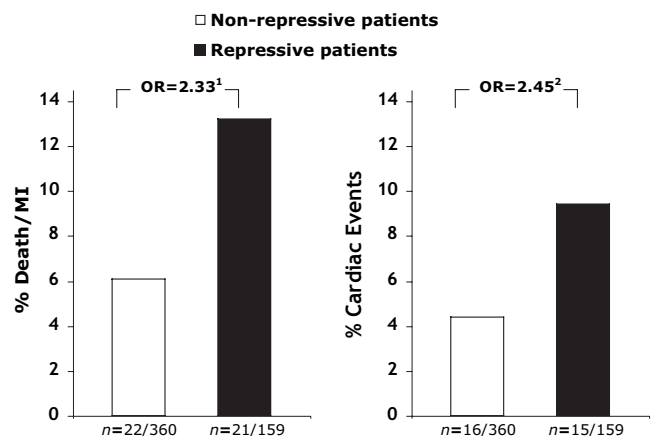


Figure 2. Percentage of repressive patients versus nonrepressive patients who had a clinical event. The association between repressive coping and clinical events after 5–10 year follow-up was analyzed for all cause mortality/MI (left) and for cardiac death/MI as cardiac events (right), respectively. ¹95%CI [1.23–4.41], *p* = .009; adjusted for age and gender. ²95%CI [1.17–5.15], *p* = .018; adjusted for age and gender.

In terms of clinical intervention, CAD is a life-threatening disease causing much patient burden. An important objective of self-management interventions for CAD patients is to help them cope with their chronic condition. However, there is some evidence suggesting that we need to consider the timing and nature of behavioral intervention strategies carefully in repressive patients. Some of these interventions have been associated with unfavorable medical outcomes in repressive CAD patients (Frasure-Smith et al., 2002; Shaw, Cohen, Doyle, & Palesky, 1985), possibly due to the fact that the intervention may have interfered with these patients' normal coping strategy to minimize distress.

Recently, we have argued that we need to learn more about factors that may modulate the impact of negative emotions on cardiac prognosis (Denollet, Pedersen, Vrints, & Conraads, 2006). The active inhibition of consciously experienced negative emotions, for example, may be a source of chronic stress that increases susceptibility to clinical events in CAD (Denollet, Pedersen, Ong, Erdman, Serruys, & van Domburg, 2006). The present findings suggest that repressive coping may modify the risk associated with patients' self-reports of low distress. We found clinical evidence that repressive CAD patients are at risk for death/MI. This phenomenon may cause an underestimation of the effect of stress on the heart, and possibly undertreatment of some high-risk patients. Inclusion of repressive coping as a modulating factor may lead to a better understanding of the relationship between emotional distress and prognosis in patients with CAD.

References

- Barth, J., Schumacher, M., & Herrmann-Lingen, C. (2004). Depression as a risk factor for mortality in patients with coronary heart disease: A meta-analysis. *Psychosomatic Medicine*, *66*, 802–813.
- Broomfield, N. M., & Turpin, G. (2005). Covert and overt attention in trait anxiety: A cognitive psychophysiological analysis. *Biological Psychology*, *68*, 179–200.
- Creed, F. (1999). The importance of depression following myocardial infarction. *Heart*, *82*, 406–408.
- Crowne, D. P., & Marlowe, D. (1960). A new scale of social desirability independent of psychopathology. *Journal of Consulting Psychology*, *24*, 349–354.
- de Jonge, P., Ormel, J., van den Brink, R. H., van Melle, J. P., Spijkerman, T. A., Kuijper, A., et al. (2006). Symptom dimensions of depression following myocardial infarction and their relationship with somatic health status and cardiovascular prognosis. *American Journal of Psychiatry*, *163*, 138–144.
- Denollet, J. (1991). Negative affectivity and repressive coping: Pervasive influence on self-reported mood, health, and coronary-prone behavior. *Psychosomatic Medicine*, *53*, 538–556.
- Denollet, J. (1993). Biobehavioral research on coronary heart disease: Where is the person? *Journal of Behavioral Medicine*, *16*, 115–141.
- Denollet, J., & Brutsaert, D. L. (1998). Personality, disease severity and the risk of long-term cardiac events in patients with a decreased ejection fraction after myocardial infarction. *Circulation*, *97*, 167–173.
- Denollet, J., Pedersen, S. S., Ong, A. T. L., Erdman, R. A. M., Serruys, P. W., & van Domburg, R. T. (2006). Social inhibition modulates the effect of negative emotions on cardiac prognosis following percutaneous coronary intervention in the drug-eluting stent era. *European Heart Journal*, *27*, 171–177.
- Denollet, J., Pedersen, S. S., Vrints, C. J., & Conraads, V. M. (2006). Usefulness of Type-D personality in predicting five-year cardiac events above and beyond concurrent symptoms of stress in patients with coronary heart disease. *American Journal of Cardiology*, *97*, 970–973.
- Denollet, J., Sys, S. U., Stroobant, N., Rombouts, H., Gillebert, T., & Brutsaert, D. L. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *Lancet*, *347*, 417–421.
- Denollet, J., Vaes, J., & Brutsaert, D. L. (2000). Inadequate response to treatment in coronary heart disease: Adverse effects of Type-D personality and younger age on 5-year prognosis and quality of life. *Circulation*, *102*, 630–635.
- Denollet, J., Pedersen, S. S., Ong, A. T., Erdman, R. A., Serruys, P. W., & van Domburg, R. T. (2006). Social inhibition modulates the effect of negative emotions on cardiac prognosis following percutaneous coronary intervention in the drug-eluting stent era. *European Heart Journal*, *27*, 171–177.
- Derakshan, N., & Eysenck, M. W. (1997). Interpretive biases for one's own behavior and physiology in high-trait-anxious individuals and repressors. *Journal of Personality and Social Psychology*, *73*, 816–825.
- Dickens, C. M., McGowan, L., Percival, C., Douglas, J., Tomenson, B., Cotter, L., et al. (2004). Lack of a close confidant, but not depression, predicts further cardiac events after myocardial infarction. *Heart*, *90*, 518–522.
- Erdman, R. A., Duivenvoorden, H. J., Verhage, F., Kazemier, M., & Hugenholtz, P. G. (1986). Predictability of beneficial effects in cardiac rehabilitation: A randomized clinical trial of psychosocial variables. *Journal of Cardiopulmonary Rehabilitation*, *6*, 206–213.
- Frasure-Smith, N., Lespérance, F., Gravel, G., Masson, A., Juneau, M., & Bourassa, M. G. (2002). Long-term survival differences among low-anxious, high-anxious and repressive copers enrolled in the Montreal heart attack readjustment trial. *Psychosomatic Medicine*, *64*, 571–579.
- Giese-Davis, J., Sephton, S. E., Abercrombie, H. C., Duran, R. E., & Spiegel, D. (2004). Repression and high anxiety are associated with aberrant diurnal cortisol rhythms in women with metastatic breast cancer. *Health Psychology*, *23*, 645–650.
- Gottdiener, J. S., Panza, J. A., St John Sutton, M., Bannon, P., Kushner, H., & Weissman, N. J. (2002). Outcome of congestive heart failure in elderly persons: Influence of left ventricular systolic function. The Cardiovascular Health Study. *Annals of Internal Medicine*, *137*, 631–639.
- Gross, J. J. (2002). Emotion regulation: Affective, cognitive and social consequences. *Psychophysiology*, *39*, 281–291.
- Grossman, P., Watkins, L. L., Ristuccia, H., & Wilhelm, F. H. (1997). Blood pressure responses to mental stress in emotionally defensive patients with stable coronary artery disease. *American Journal of Cardiology*, *80*, 343–346.
- Jamner, L. D., Schwartz, G. E., & Leigh, H. (1988). The relationship between repressive and defensive coping styles and monocyte, eosinophile, and serum glucose levels: Support for the opioid peptide hypothesis of repression. *Psychosomatic Medicine*, *50*, 567–575.
- Knight, R., Waal-Manning, H., & Spears, G. (1983). Some norms and reliability data for the state-trait anxiety inventory and the Zung self-rating depression scale. *British Journal of Clinical Psychology*, *22*, 245–249.
- Lakka, T. A., Laukkanen, J. A., Rauramaa, R., Salonen, R., Lakka, H. M., Kaplan, G. A., & Salonen, J. T. (2001). Cardiorespiratory fitness and the progression of carotid atherosclerosis in middle-aged men. *Annals of Internal Medicine*, *134*, 12–20.
- Lane, R. D., Merikangas, K. R., Schwartz, G. E., Huang, S. S., & Prusoff, B. A. (1990). Inverse relationship between defensiveness and lifetime prevalence of psychiatric disorder. *American Journal of Psychiatry*, *147*, 573–578.
- Lane, R. D., Sechrest, L., Riedel, R., Shapiro, D. E., & Kaszniak, A. W. (2000). Pervasive emotion recognition deficit common to alexithymia and the repressive coping style. *Psychosomatic Medicine*, *62*, 492–501.
- Levine, J., Warrenburg, S., Kerns, R., Schwartz, G., Delaney, R., Fontana,

- A., et al. (1987). The role of denial in recovery from coronary heart disease. *Psychosomatic Medicine*, *49*, 109–117.
- Movius, H. L., & Allen, J. J. (2005). Cardiac vagal tone, defensiveness, and motivational style. *Biological Psychology*, *68*, 147–162.
- Myers, L. B., Brewin, C. R., & Power, M. J. (1998). Repressive coping and the directed forgetting of emotional material. *Journal of Abnormal Psychology*, *107*, 141–148.
- Nyklíček, I., Vingerhoets, A. J., & van Heck, G. L. (1999). Elevated blood pressure and self-reported symptom complaints, daily hassles, and defensiveness. *International Journal of Behavioral Medicine*, *6*, 177–189.
- Pauls, C. A., & Stemmler, G. (2003). Repressive and defensive coping during fear and anger. *Emotion*, *3*, 284–302.
- Rutledge, T., & Linden, W. (2000). Defensiveness status predicts 3-year incidence of hypertension. *Journal of Hypertension*, *18*, 153–159.
- Rutledge, T., & Linden, W. (2003). Defensiveness and 3-year blood pressure levels among young adults: The mediating effect of stress-reactivity. *Annals of Behavioral Medicine*, *25*, 34–40.
- Schneiderman, N., Saab, P. G., Catellier, D. J., Powell, L. H., DeBusk, R. F., Williams, R. B., et al., for the ENRICHD Investigators (2004). Psychosocial treatment within sex by ethnicity subgroups in the Enhancing Recovery in Coronary Heart Disease clinical trial. *Psychosomatic Medicine*, *66*, 475–483.
- Shaw, R. E., Cohen, F., Doyle, B., & Palesky, J. (1985). The impact of denial and repressive style on information gain and rehabilitation outcomes in myocardial infarction patients. *Psychosomatic Medicine*, *47*, 262–273.
- Strik, J. J., Denollet, J., Lousberg, R., & Honig, A. (2003). Comparing symptoms of depression and anxiety as predictors of cardiac events and increased health care consumption following myocardial infarction. *Journal of the American College of Cardiology*, *42*, 1801–1807.
- van der Ploeg, H. M., Defares, P. B., & Spielberger, C. D. (1980). *ZBV. A Dutch-Language Adaptation of the Spielberger State-Trait Anxiety Inventory*. Lisse, The Netherlands: Swets & Zeitlinger.
- van der Ploeg, H. M., Defares, P. B., & Spielberger, C. D. (1982). *ZAV. A Dutch-Language Adaptation of the Spielberger State-Trait Anger Scale*. Lisse, The Netherlands: Swets & Zeitlinger, 1982.
- Weinberger, D. A., Schwartz, G. E., & Davidson, R. J. (1979). Low-anxious, high-anxious and repressive coping styles: Psychometric patterns and behavioral and physiological responses to stress. *Journal of Abnormal Psychology*, *88*, 369–380.
- Whooley, M. A., & Simon, G. E. (2000). Managing depression in medical outpatients. *New England Journal of Medicine*, *343*, 1942–1950.

Correction to Chae and Yoshikawa (2008)

In the article, “Perceived Group Devaluation, Depression, and HIV-Risk Behavior Among Asian Gay Men” by David H. Chae and Hirokazu Yoshikawa (*Health Psychology*, 2008, Vol. 27, No. 2, pp. 140-148), the second sentence of the **Results** portion of the abstract should read:

Among participants most attracted to Whites, group devaluation was associated with higher levels of nonprimary partner UAI; but was associated with lower levels of nonprimary partner UAI among those most attracted to non-Whites.

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