Personality as independent predictor of long-term mortality in patients with coronary heart disease

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Summary

Background Emotional distress has been related to mortality in patients with coronary heart disease (CHD), but little is known about the role of personality in long-term prognosis. We postulated that type-D personality (the tendency to suppress emotional distress) was a predictor of long-term mortality in CHD, independently of established biomedical risk factors.

Methods We studied 268 men and 35 women with angiographically documented CHD, aged 31–79 years, who were taking part in an outpatient rehabilitation programme. All patients completed personality questionnaires at entry to the programme. We contacted them 6–10 years later (mean 7·9) to find out survival status. The main endpoint was death from all causes.

Findings At follow-up, 38 patients had died; there were 24 cardiac deaths. The rate of death was higher for type-D patients than for those without type-D (23 [27%]/85 vs 15 [7%]/218; p<0·00001). The association between type-D personality and mortality was still evident more than 5 years after the coronary event and was found in both men and women. Mortality was also associated with impaired left ventricular function, three-vessel disease, low exercise tolerance, and the lack of thrombolytic therapy after myocardial infarction. When we controlled for these biomedical predictors in multiple logistic regression analysis, the impact of type-D remained significant (odds ratio 4·1 [95% CI 1·9–8·8]; p=0·0004). In this group of CHD patients, type-D was an independent predictor of both cardiac and non-cardiac mortality. Social alienation and depression were also related to mortality, but did not add to the predictive power of type-D.

Interpretation We found that type-D personality was a significant predictor of long-term mortality in patients with established CHD, independently of biomedical risk factors. Personality traits should be taken into account in the association between emotional distress and mortality in CHD.

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Introduction

Research on the prognosis and treatment of patients with coronary heart disease (CHD) has identified several important biomedical prognostic factors.[1,2] A relation between personality and CHD has also been suggested.[3] Psychosocial factors may affect longevity,[4] and particular factors such as social alienation[5] and depression[6] have been associated with a poor outcome in patients who survived myocardial infarction. However, little is known about the role of personality factors in prognosis in CHD or about the prognostic value of psychosocial stress more than 5 years after the coronary event.

We examined the role of personality in 6–10-year mortality of CHD patients, with control for biomedical prognostic factors such as the severity of cardiac disease. This study focused on the tendency to inhibit self-expression in social interaction (social inhibition[7]) and the tendency to experience negative emotions (negative affectivity[8]) because these personality traits may promote social alienation and depression.

Patients and methods

Patients

268 men and 35 women, aged 31–79 years (mean 55·4 [SD 7·9] years) were selected from a consecutive series of CHD patients enrolled in the Antwerp cardiac rehabilitation programme between January, 1985, and December, 1988. Patients were eligible for this study if they had experienced a coronary event within the 2 months before entry to the programme. 182 patients had had myocardial infarctions within the previous 2 months and 121 coronary patients without recent myocardial infarction had undergone coronary bypass surgery or angioplasty. We did not exclude patients with a previous myocardial infarction or impaired left ventricular function. The only criteria for exclusion were that another serious disease was present at baseline (renal failure, cancer) or that no assessment of left ventricular ejection fraction had been done at baseline.

All patients underwent a standard treatment regimen—an outpatient rehabilitation programme comprising 36 sessions of electrocardiographically monitored aerobic exercise training and six psychosocial counselling sessions in groups of patients and spouses. Individual medical and psychological counselling tailored the rehabilitation programme to the needs of each patient. The presence of patients in the Antwerp rehabilitation programme is not a function of severity of cardiac disorder, but largely reflects the attitude of referring physicians towards cardiac rehabilitation. Standard medical care during follow-up was similar for all patients; it consisted of a routine cardiological check-up every 6 months. Women were included in this study because cardiac mortality after myocardial infarction is independent of sex[9] and there is preliminary evidence that psychosocial factors may be related to long-term mortality in women with premature myocardial infarction.[10] A subset of 93 patients also took part in a pilot study on the relation between personality and 2–5-year mortality.[11]

Type-D personality

This study was preceded by psychological studies defining distinctly different personality types in patients with CHD.[11,12]

Personality types can be delineated either through empirical induction (relying on statistical procedures) or through theoretical deduction (relying on specific assumptions). We used a combined inductive-deductive approach to analyse the role of personality in the context of CHD.

We initially used cluster analysis, a multivariate statistical procedure that is specifically designed to classify subjects into homogeneous subtypes. This inductive approach yielded a discrete personality type of CHD patient characterised by high scores on both negative affectivity and social inhibition.[12] External measures not involved in the clustering showed that these patients had high scores for emotional distress and low scores for subjective well-being. The reliability of this personality type was shown across parallel datasets, and follow-up assessment showed that these patients were still experiencing substantial distress 15 months after the initial assessment.[12]

On the basis of this empirically generated model, a subsequent study[11] used a deductive approach to define the distressed personality type or type-D in patients with CHD (table 1). By analogy with cluster analysis, a median split on measures of negative affectivity and social inhibition was used as a definition of type-D—ie, patients with type-D personality tend simultaneously to experience negative emotions and inhibit self-expression (as indicated by scores above the medians for these traits). Type-D is therefore characterised by the chronic suppression of negative emotions. Preliminary findings suggested that type-D, in fact, was associated with depression, social alienation, and 2–5-year mortality in middle-aged men who survived myocardial infarction.[11]

The definition of type-D was derived before the mortality outcome was known in this study. We postulated for this study that type-D was an independent predictor of long-term mortality in CHD. First, inhibition of emotional expression may promote disease.[13] Suppressed anger, for example, has been associated with hypertension, incidence of CHD, and mortality.[13] Second, type-D patients are prone to depression and social alienation, two psychosocial factors that may increase mortality in CHD.[5,6]

A median split on the trait scale of the state-trait anxiety inventory[14] and the social inhibition scale of the heart patients psychological questionnaire[15] was used to classify 85 patients as type-D (ie, trait-anxiety $\geqslant 43$ and social inhibition $\geqslant 12$) and 218 patients as not type-D. Among patients with CHD, these scales are valid measures of negative affectivity and social inhibition, respectively.[8,12]

Prognostic factors

Left ventricular function and the extent of coronary obstructive disease are powerful predictors of mortality for CHD patients.[2] To ensure that any observed personality effects were independent of disease severity, we included indices of impaired left ventricular function (left ventricular ejection fraction $\leqslant 40\%$ calculated from ventricular angiography) and extent of coronary disease (severe=three vessels with 70% or greater reduction in internal diameter).

Biomedical risk assessment also included non-invasive measurement and clinical data.[1,2] Clinical risk factors included older age ($\geqslant 56$ years), history of myocardial infarction, one or more previous myocardial infarctions, anterior location of myocardial infarction, no thrombolysis after myocardial infarction, no aspirin therapy or no β-blocker therapy at discharge from the rehabilitation programme, poor compliance with the exercise regimen, failure to quit smoking, and history of hyperlipidaemia. A poor exercise tolerance was defined by a median split for peak workload on a symptom-limited exercise test 6 weeks after the coronary event ($\leqslant 140$ W and $\leqslant 120$ W for younger and older men; $\leqslant 100$ W and $\leqslant 80$ W for younger and older women, respectively). We recorded the frequency of angiography, coronary artery bypass surgery, and angioplasty during follow-up.

Social alienation and depression were measured by the Millon behavioural health inventory.[16] Patients were classified as being prone to social alienation if they scored above the median ($\geqslant 7$) for the social alienation scale of the inventory, which measures

	Personality trait	
	Negative affectivity	Social inhibition
Definition		
Cluster analysis	Mean T-score >60	Mean T-score >55
Theoretical model	High score*	High score*
Assessment		
Self-report scales	Trait-anxiety scale of STAI	Social inhibition scale of HPPQ
Correlates		
Clinical	Intrapsychic; tends to worry and take gloomy view of things, often feels unhappy or irritated	Interpersonal; tends to keep others at distance, often inhibits expression of true feelings
Psychometric	Symptoms of depression and chronic tension, low subjective well-being	Low levels of perceived social support

*Defined by median split of scores on corresponding trait measure.
T-score=standardised score (mean 50 [SD10]). STAI=state-trait anxiety inventory; HPPQ=heart patients psychological questionnaire.

Table 1: **Definition of type-D personality**

perception of lack of social support. The premorbid pessimism and future despair scales of the inventory measure cognitive dimensions of depression. Patients were classified as being prone to depression if they scored above the median for both scales ($\geqslant 10$ and $\geqslant 12$, respectively).

In addition, use of benzodiazepines was used as a non-test marker of emotional distress. Use of benzodiazepines has been related to poor prognosis in myocardial infarction patients.[11]

Procedure

At entry to the rehabilitation programme, all patients completed psychological questionnaires. The psychological status of chronically ill patients represents enduring personality traits rather than the demands of their medical condition. Depression and the association between depression and mortality[6] are independent of disease severity in myocardial infarction patients. Type-D was not associated with disease severity in this study: among myocardial infarction patients, type-D and non-type-D patients did not differ in left ventricular ejection fraction (51 vs 55%, p=0·10) or percentage with three-vessel disease (29 vs 27%, p=0·71); similarly, there was no difference between type-D and non-type-D among coronary artery bypass graft or angioplasty patients in left ventricular ejection fraction (60 vs 64%, p=0·10) or percentage with three-vessel disease (65% vs 60%, p=0·62).

Follow-up, as determined retrospectively by when the patient joined the rehabilitation programme, varied between 6 and 10 years (mean 7·9). Between September and December, 1994, we contacted participants and their families to find out whether participants were still alive. The main endpoint in this study was death from all causes. In secondary analyses, causes of death were divided into cardiac and non-cardiac categories. Mortality data were derived from hospital records and the patient's attending physician was always involved in the classification of cause of death.

Statistical analysis

We initially stratified patients by covariates of CHD severity to examine the effect of type-D on the risk of death within risk strata. Next, we did univariate analyses after exclusion of deaths during the first 5 years of follow-up, and after stratification by cause of death and sex. Differences in biomedical and psychosocial characteristics according to vital status were analysed for total, cardiac, and non-cardiac mortality. Dichotomous variables were analysed by the χ^2 test and Fisher's exact test; continuous variables by unpaired t tests.

To assess the prognostic value of type-D in addition to biomedical and psychosocial prognostic factors, we used multiple logistic regression analysis. Criteria for entry ($p \leqslant 0·10$) and removal ($p > 0·10$) were based on the likelihood ratio test. Multivariate analyses were repeated with cardiac death and non-cardiac death as endpoints. Patients were stratified by exercise

	Type-D		Non-type-D		p*
	No of patients	No (%) deaths	No of patients	No (%) deaths	
All participants	85	23 (27%)	218	15 (7%)	<0·00001
Participants who did not take part in pilot study†	61	13 (21%)	149	8 (5%)	0·0005
Left ventricular ejection fraction					
≥41%	64	14 (22%)	190	11 (6%)	0·0002
≤40%	21	9 (43%)	28	4 (14%)	0·02
Extent of coronary disease					
1-2 vessels affected	48	8 (17%)	131	6 (5%)	0·01
3 vessels affected	37	15 (40%)	87	9 (10%)	0·0001
Sex					
Male	71	18 (25%)	197	14 (7%)	0·00005
Female	14	5 (36%)	21	1 (5%)	0·028
Cause of death					
Cardiac	77	15 (20%)	212	9 (4%)	0·00003
Non-cardiac	70	8 (11%)	209	6 (3%)	0·009

*Type-D vs non-type-D. †93 participants in pilot study excluded.

Table 2: **Mortality according to personality type**

tolerance and personality type to examine the effect of poor physical and mental health on the risk of death (χ^2 test).

Results

No patient was lost to follow-up. After 6–10 years of follow-up, 38 (14%) of the 303 patients had died; there were 24 cardiac and 14 non-cardiac deaths. All deaths were attributable to natural causes; there were no deaths caused by accident, homicide, or suicide. There was no significant difference in mortality between men and women (p=0·26) or between myocardial infarction and coronary artery bypass/angioplasty patients (p=0·77). Hence, pooling of participants in one category for further statistical analyses was justified.

Personality was significantly associated with an increased risk of death. The risk of death was nearly four times higher in type-D than in non-type-D patients (table 2). When the 93 subjects of the pilot study were excluded, type-D patients still had a four times higher risk of death.

This personality effect was not accounted for by the severity of cardiac disease; the association between type-D personality and mortality remained significant after adjustment for left ventricular function and extent of coronary obstructive disease (table 2).

There were 14 deaths that occurred more than 5 years after the coronary event. The risk of death after more than 5 years of follow-up was three times greater in type-D patients than in non-type-D patients (7 [10%]/69 vs 7 [3%]/210; p=0·032).

Type-D was associated with mortality in both men and women (table 2) and was associated with both cardiac death and non-cardiac death. The death rate was lower for patients scoring high on trait-anxiety but low on social inhibition than for type-D patients (4 [6%]/66 vs 27%; p=0·0008) and did not differ from that for patients scoring low on trait-anxiety (11 [7%]/152; p=0·75). Likewise, the rate of death for patients scoring high on social inhibition but low on trait-anxiety was smaller than that for type-D patients (5 [6%]/81 vs 27%; p=0·0003) and did not differ from that for patients scoring low on social inhibition (10 [7%]/137; p=0·75). Accordingly, it was not trait-anxiety or social inhibition per se but rather the interaction of trait-anxiety with social inhibition that was predictive of mortality.

Baseline characteristics	% (number of subjects)		p
	Vital status at 6–10 years		
	Alive (n=265)	Dead (n=38)	
Demographic data			
Age >55 years	54% (144)	66% (25)	0·18
Male	89% (236)	84% (32)	0·26
White collar worker	59% (157)	58% (22)	0·87
Disease severity			
Impaired left ventricular function*	14% (36)	34% (13)	0·001
Three-vessel disease	38% (100)	63% (24)	0·003
Non-invasive and clinical data			
Poor exercise tolerance†	43% (114)	76% (29)	0·0001
History of MI	71% (188)	76% (29)	0·49
One or more previous MI	11% (30)	24% (9)	0·03
Anterior location of MI	29% (77)	40% (15)	0·19
Thrombolysis after MI	16% (30)	3% (1)	0·05
Aspirin therapy	63% (167)	53% (20)	0·22
β-blocker therapy	49% (129)	55% (21)	0·45
Angiography after rehabilitation	14% (36)	13% (5)	0·94
CABG/PTCA after rehabilitation	9% (24)	13% (5)	0·29
Compliance with exercise regimen	92% (243)	82% (31)	0·05
Failure to quit smoking	23% (61)	21% (8)	0·79
History of hyperlipidaemia	30% (80)	21% (8)	0·25
Psychosocial factors			
Type-D personality	23% (62)	61% (23)	0·00001
Social alienation	42% (112)	66% (25)	0·006
Depression	39% (103)	63% (24)	0·005
Use of benzodiazepines	19% (51)	34% (13)	0·03

*Left ventricular ejection fraction ≤40%; †≤140% and ≤120 W for younger and older men; ≤100 and ≤80 W for younger and older women. MI=myocardial infarction; CABG=coronary artery bypass surgery; PTCA=percutaneous transluminal coronary angioplasty.

Table 3: **Baseline biomedical and psychosocial characteristics according to vital status**

Patients who died differed from survivors in several biomedical and psychosocial characteristics that have previously been related to mortality in coronary patients (table 3). They were more likely to have impaired left ventricular function, three-vessel disease, poor exercise tolerance, previous myocardial infarctions, characteristics of social alienation, depressive symptoms, and benzodiazepine use, and they were slightly less likely to have been treated with thrombolysis and to adhere to the exercise regimen. Cardiac death was associated with type-D personality (p<0·0001), poor exercise tolerance (p<0·001), impaired left ventricular function, three-vessel disease, previous myocardial infarction, depression (p<0·01), history of myocardial infarction, anterior myocardial infarction (p<0·05), and lack of thrombolysis treatment (p=0·05). Non-cardiac death was associated with type-D personality, lack of perceived social support (p<0·01), and poor exercise tolerance (p<0·05).

To find out whether type-D was an independent predictor of 6–10-year mortality, we first entered biomedical and psychosocial factors (but not personality type) in a stepwise logistic regression model. This initial analysis yielded a group of independent predictors that included impaired left ventricular function, three-vessel disease, poor exercise tolerance, lack of thrombolysis treatment after myocardial infarction, hyperlipidaemia, and depressive symptoms. Next, we added type-D to this logistic regression model. The final model included type-

Variable	Odds ratio (95% CI)	p
Type-D personality	4·1 (1·9–8·8)	0·0004
Impaired left ventricular function	3·0 (1·1–8·0)	0·03
Three-vessel disease	2·4 (1·1–5·2)	0·03
Poor exercise tolerance	2·4 (1·1–5·3)	0·03
Thrombolysis after myocardial infarction	0·2 (0·03–1·2)	0·07

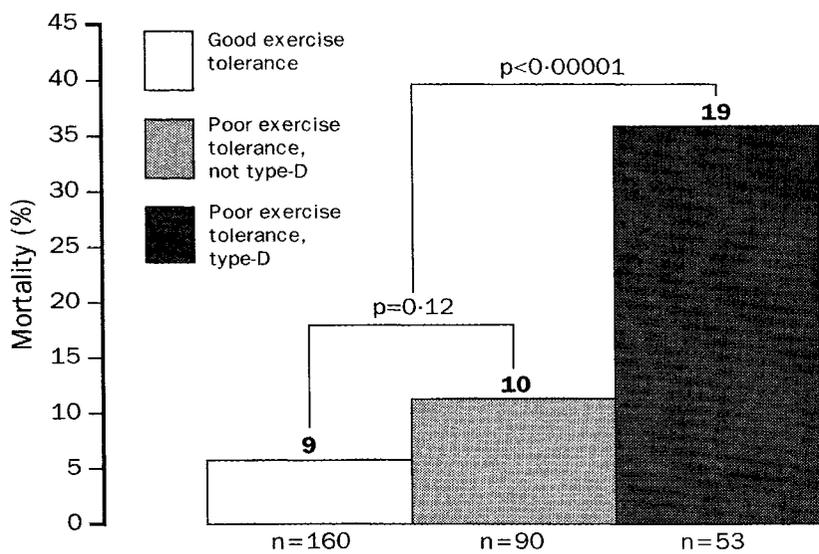Table 4: **Predictors of 6–10 year mortality by multiple logistic regression**

Figure: **Percentage of patients who died, according to exercise tolerance and type-D personality**
Number of deaths given at top of bar. Patients with poor exercise tolerance were stratified by type-D personality. Patients with good exercise tolerance were pooled (type-D and not type-D).

D but not depression (table 4). Type-D was therefore a significant prognostic factor independent of biomedical prognostic factors, and depression, social alienation, and use of benzodiazepines did not add significantly to the predictive power provided by type-D. Post-hoc analyses showed that type-D patients were more likely to report depression and social alienation (p<0·00001) and were more likely to use benzodiazepines (p=0·0004) than non-type-D patients.

Multiple logistic regression analyses yielded three independent risk factors for cardiac mortality—poor exercise tolerance (odds ratio 4·3 [95% CI 1·6–11·6]; p=0·004), type-D personality (3·8 [1·4–10·0]; p=0·0067), and history of myocardial infarction (4·7 [1·3–17·8]; p=0·021)—and two independent risk factors for non-cardiac mortality—type-D personality (6·4 [1·7–23·4; p=0·0054) and three-vessel disease (3·6 [1·0–12·9]; p=0·046).

As reported previously,[17] CHD patients with good exercise tolerance had a low mortality risk in this study (4 [13%]/32 type-D *vs* 5 [4%]/128 non-type-D; p=0·08). However, patients with poor exercise tolerance but not type-D did not differ significantly in mortality from patients with good exercise tolerance (figure). By contrast, patients with both poor exercise tolerance and type-D personality had more than four times the risk of death of patients with either good exercise tolerance or no type-D personality.

Discussion

We found that type-D personality was associated with long-term mortality in men and women with established CHD, after adjustment for the severity of cardiac disease. Consistent with previous reports, various biomedical and psychosocial factors were also associated with mortality. Multivariate analysis showed that type-D was a significant predictor of mortality independently of biomedical prognostic factors. In addition, this analysis showed that neither social alienation nor depression added significantly to the predictive power provided by type-D.

These findings are important for two reasons. First, we found evidence that biomedical factors and personality are independent predictors of mortality in CHD patients. We focused on the interplay of two broad, stable personality traits—the tendency to experience negative emotions and the tendency to inhibit self-expression. The presence of only one of these tendencies had no effect; it was the interaction of the two that had an adverse effect on prognosis. Hence, our findings are relevant to both emotional stress and the chronic suppression of emotions.[13]

Second, although others have shown that psychosocial stress is an independent predictor during the first years after myocardial infarction[5,6] we found evidence that the adverse effect of psychosocial stress on prognosis may last much longer. The association between type-D and mortality was still evident 5 years after the coronary event. Hence, it may be valuable to examine further the effect of personality in long-term follow-up studies of CHD patients. The strength of the association between type-D and mortality in this study, as shown by an adjusted odds ratio of 4·1, warrants additional exploration in confirmatory research.

Caution is needed, however, in interpretation of the implications of this study. Cardiac rehabilitation may improve survival[1] and emotional well-being;[18] whether type-D would predict mortality in a less selected population of CHD patients than our patients from a rehabilitation programme remains unclear. Furthermore, our data cannot show that personality was a direct factor in causing mortality. The study was designed to examine the prognostic power of type-D but not to uncover mechanisms that may account for any associations between personality and mortality. We can therefore only speculate about possible mechanisms that might explain this association.

Personality might promote disease directly through pathophysiological mechanisms. Coronary spasm and activated blood platelets have an important role in the progression of CHD and arterial thrombosis.[19] Both coronary spasm[20] and platelet release[19] may be potentiated by exposure to mental stress. Accordingly, silent myocardial ischaemia may develop during mental stress testing at low heart rates in patients with CHD,[21] and patients with ischaemia during mental stress testing are likely to have increased ischaemia during sedentary activities in daily life.[22] Silent ischaemia due to coronary spasm can, in turn, initiate potentially fatal arrhythmias. There is evidence that depressed patients with CHD have lower than normal heart-rate variability,[23] which may predispose to ventricular fibrillation. In our study personality was related to both cardiac and non-cardiac mortality, which suggests that psychosocial stress is associated with various pathophysiological states.

Personality might also promote disease indirectly through health-related behaviours. Failure to change risk factors and poor treatment adherence[24] are related to a greater extent of coronary disease and an increased risk of death in patients with CHD. The tendency of type-D patients to inhibit behaviour in social interaction also means that they may decrease the availability of social support. Lack of social support is likely to potentiate the role of psychosocial stress in the progression of CHD.[25] In addition, social inhibition may impair communication with doctors, which may in turn hinder effective treatment.[26]

A third possible mechanism is that there is a third variable that is a primary cause of both personality traits and premature mortality. Type-D would be merely a behaviour manifestation of an underlying biological or genetic variable that predisposes an individual to adverse

health outcomes. CHD incidence[27] and longevity have been associated with underlying genetic causes. There is evidence that about 50% of individual differences in personality traits such as negative affectivity and social inhibition is due to genetic factors.[28]

We do not know what therapeutic interventions, if any, would help to decrease mortality in type-D patients with CHD. It is certainly premature to conclude that psychotherapeutic interventions or psychotropic agents are indicated for these patients. Inclusion of type-D as an individual difference variable in therapeutic trials may help to answer the question of whether type-D patients could benefit from specific interventions. In any case, a link between personality and mortality does not mean that the patient is at fault for not being able to control the disease.

Although our study precludes definitive conclusions, the findings support the notion that stable personality factors may be related to mortality in patients with CHD.[3] Others have shown that adverse health outcomes in CHD are related to social isolation[5] and depression[6] as well as emotional exhaustion,[29] and that personality may predict longevity across the lifespan.[30] We found evidence that CHD patients with type-D had a four-fold risk of death compared with non-type-D patients.

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