



Review Paper

Type D personality, cardiac events, and impaired quality of life: a review

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Background Psychological distress has been associated with the pathogenesis and progression of coronary heart disease (CHD) but little is known about the determinants of distress as a coronary risk factor. Although it has become unfashionable to focus on personality factors since research on Type A behaviour yielded inconsistent findings, personality may comprise a major explanatory factor of individual differences in stress-related CHD. This article focuses on Type D – the *distressed* – personality, which describes patients who experience increased negative emotions and tend to inhibit the expression of these emotions in social interactions.

Methods The article reviews research on Type D personality in the context of CHD.

Results Accumulating evidence indicates that cardiac patients with the Type D personality are at increased risk for cardiovascular morbidity and mortality (odds ratios ranging from 4.1–8.9, $P < 0.0001$) independent of standard cardiac risk factors. Type D patients are also at increased risk for psychological distress, clustering of psychosocial risk factors, impaired quality of life, and seem to benefit less from medical and invasive treatment. Preliminary evidence suggests that physiological hyper-reactivity and activation of pro-inflammatory cytokines may be responsible for the detrimental effect of Type D personality on cardiac prognosis.

Conclusions There is an urgent need to adopt a personality approach in the identification of patients at risk for stress-related cardiac events. Type D is a stable personality construct that may be of special interest not only in CHD, but in other chronic cardiac conditions as well. *Eur J Cardiovasc Prevention Rehab* 10:241–248 © 2003 Lippincott Williams & Wilkins.

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Keywords: cardiac events, coronary heart disease, depression, Type D personality, quality of life, prognosis

Introduction

Acute and chronic psychological distress has been associated with the pathogenesis of coronary heart disease (CHD) [1,2] but little is known about the determinants of distress as a coronary risk factor. Although broad and stable personality traits may have much explanatory power, few studies on the interface between cardiology and psychology have included personality factors. This may in part be

due to the controversy surrounding the Type A coronary-prone behaviour pattern, which made it unfashionable to include personality factors in CHD research.

The present article reviews evidence from 10 articles, published between 1996 and 2003, on the more recent Type D personality construct (Table 1). In this review, we will first briefly evaluate the similarities and differences between the Type A behaviour patterns (TABP) and the Type D personality construct. Subsequently, we will review research on Type D personality in the context of CHD with a view to presenting evidence that Type D is an important factor in explaining individual differences in

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Table 1 Chronological view of studies published on the Type D personality construct

Authors	Year	Ref	Subjects	Study design	Main endpoint
Denollet <i>et al.</i>	1996	19	303 CHD patients	Prospective follow-up	All cause mortality
Denollet <i>et al.</i>	1998	30	87 MI patients with LVEF \leq 50%	Prospective follow-up	Cardiac death/non-fatal MI
Denollet	1998	31	246 CHD patients	Prospective follow-up	Cancer
Denollet	1998	14	100 CHD patients	Cross-sectional	Negative and positive affect
Denollet <i>et al.</i>	2000	29	319 CHD patients	Prospective follow-up	Cardiac death/non-fatal MI
Denollet	2000	17	734 hypertensives	Cross-sectional	Depressive affect
Appels <i>et al.</i>	2000	32	99 SCA and 119 CHD patients	Case-control	Sudden cardiac arrest
Pedersen <i>et al.</i>	2001	26	171 CHD patients	Intervention study	Symptoms of exhaustion
Denollet <i>et al.</i>	2003	33	42 heart failure patients	Cross-sectional	Inflammatory markers
Habra <i>et al.</i>	2003	36	173 undergraduates	Cross-sectional	Cardiovascular and neuroendocrine reactivity

SCA, sudden cardiac arrest; CHD, coronary heart disease; MI, myocardial infarction; LVEF, left ventricular ejection fraction.

cardiovascular morbidity and mortality. We will then focus on Type D personality as a determinant of quality of life and as a moderator of treatment. Finally, we will discuss the clinical implications of the findings on Type D.

Contrasting Type A and Type D

Type A behaviour pattern

The Type A behaviour pattern was derived from clinical observations by Friedman and Rosenman [3] that behavioural factors may influence serum cholesterol levels independent of diet. It was defined as 'an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and if required to do so, against the opposing efforts of other things or other persons' [4]. The validity of TABP was initially confirmed in two prevalence studies showing higher serum cholesterol levels and clinical manifestations of CHD in Type A relative to Type B – with the latter designating the absence of TABP [3,5]. These two studies led the way for the Western Collaborative Group Study, a prospective study, which found that TABP predicted the incidence of CHD independent of traditional biomedical risk factors [6]. This finding was also confirmed by independent research teams, and sparked an evaluation of the construct by a panel of biomedical and behavioural scientists, after which the behaviour pattern was formally accepted as a risk factor for CHD on a par with traditional biomedical risk factors [7].

Subsequent studies failing to show a relationship between TABP and CHD, however, suggest that the panel may have been too premature in their conclusions [8,9]. Later research showed that only parts of TABP might have a toxic effect on the pathogenesis of CHD with hostility appearing to be the most potent component [10]. In addition, a recent review indicates that Type A/hostility may play an aetiological but not a prognostic role in the pathogenesis of CHD [2]. Finally, it is important to note that although TABP has often been referred to as a personality type, the TABP construct was specifically

designed to *avoid* any associations with global personality traits [11]. Therefore, it is particularly paradoxical that the controversy surrounding the TABP construct has led to the avoidance of personality factors in CHD research.

Type D personality

Contrary to the TABP construct, Type D personality – also called the *distressed* personality – was derived from existing personality theory and empirical evidence, including cluster and factor analyses [12,13]. The taxonomy is based on two broad and stable traits, those of *negative affectivity* and *social inhibition* [14]. Negative affectivity denotes the tendency to experience increased negative distress across time and situations [15], whereas social inhibition refers to the tendency to inhibit the expression of these negative emotions in social interactions [16]. A high score on both traits denotes those with a Type D personality. From a clinical point of view, Type D patients tend to worry, to take a gloomy view of life, and to feel tense and unhappy. They are more easily irritated and in general are less likely to experience positive mood states. At the same time, they are less likely to share these negative emotions with other people for fear of rejection and disapproval. Type D patients also generally have fewer personal ties with other people and tend to feel uncomfortable when with strangers [17].

Type D can be considered a chronic psychological risk factor insofar as Type D patients tend to deal with emotions in a characteristic way. The construct was originally developed in Belgian cardiac patients in an attempt to investigate the role of personality traits in CHD outcome [14]. It is important to note that the construct emphasizes normal personality traits rather than psychopathology. Hence, Type D reflects a relatively homogeneous subgroup that has a clear basis in psychological theory [17], whereas TABP reflects a 'heterogeneous hodgepodge' of behavioural symptoms [11].

The wealth of studies that have investigated the role of negative emotions in the pathogenesis of CHD, however,

begs the question as to whether the Type D construct merely represents *old wine in new bottles* [18]?

It is correct that the Type D construct measures negative emotions, but it is the joint effects of the tendency to experience negative emotions and to inhibit self-expression of these emotions rather than negative emotions *per se* that has a deleterious effect on health. This has been demonstrated in a 6–10 year follow-up study of 303 patients with CHD, where the mortality rate of patients with negative effect, but low inhibition (6%), did not differ significantly from that for low negative effect patients (7%) [19]. In other words, the Type D construct adds to research on stress-related CHD in that the way people cope with negative emotions may be as important as the experience of negative emotions *per se*. However, the way Type Ds characteristically deal with emotions is different from other emotional coping styles, such as denial [20] and repression [21–23]. Repression, for example, refers to *low distress* and the *unconscious* exclusion of negative emotions from awareness, whereas Type Ds experience *high interpersonal distress* while *consciously* trying to suppress these emotions [14]. Thus, the answer as to whether the Type D construct merely represents *old wine in new bottles* is an emphatic *no*.

Type D as a determinant of psychological distress

Type D personality has been associated with a variety of emotional and social difficulties, including depressive symptoms, chronic tension, anger, pessimism, lack of perceived social support, and a low level of subjective well-being [12–14,19]. It is particularly noteworthy that Type D patients display a relative absence of positive emotions indexed by low positive effect, low levels of self-esteem, and general dissatisfaction with life [14].

Symptoms of fatigue and vital exhaustion also seem to be more prevalent in cardiac patients with the Type D personality. Vital exhaustion is a mental state that is defined as extreme fatigue, increased irritability, and demoralization [24]. It is a risk factor on a par with hyperlipidaemia that predicts cardiac mortality up to 40 months post-myocardial infarction [24,25]. In a study of 171 patients with ischaemic heart disease, Type D was associated with a six-fold increased risk of vital exhaustion at baseline and more than a four-fold risk at 6 weeks following implementation of invasive or conservative treatment, despite a reduction in symptoms of angina following treatment [26].

The results of the latter study support the notion that chronic psychological risk factors (Type D) may promote the development of episodic risk factors (vital exhaustion), as suggested by Kop [27].

Moreover, difficulties experienced by Type D patients are not only limited to psychological distress but span behaviours and symptoms, including the chronic use of benzodiazepines, persistent complaints of chest pain, and failure to return to work [12,28]. Overall, these findings underscore the validity of the Type D construct as a ‘distressed’ personality profile leading to increased vulnerability for emotional and social difficulties.

Cardiovascular morbidity and mortality

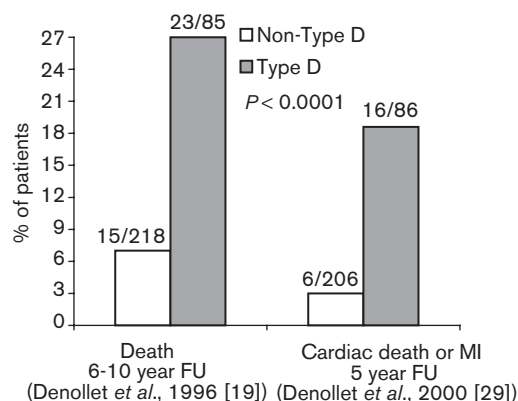
As noted above, Type D personality is an important determinant of emotional and social difficulties in patients with CHD. Eventually, these difficulties may have serious implications for the health of patients with established CHD resulting in hard-core medical endpoints in terms of morbidity and mortality.

Type D as an independent predictor of cardiac events

The first study ($n = 105$) to suggest that Type D personality has deleterious effects on health was published in 1995 [28]. Of all deaths, 73% occurred in patients with the Type D personality. When focusing on mortality due to cardiac causes, Type D was associated with a six-fold attenuated risk compared with non-Type D. Type D personality also added significantly to the predictive power of mortality above and beyond a model comprising the biomedical risk factors of low exercise tolerance, previous or anterior myocardial infarction (MI), smoking, and age.

These preliminary results were confirmed one year later in a study of 303 CHD patients, which was an extension of the 1995 study in order to increase sample size and the length of follow-up [19]. Mortality was higher for Type D patients compared with non-Type D patients (27 versus 7%; $P < 0.00001$) (Fig. 1, left), and the impact of Type D

Fig. 1



Type D personality as a long-term predictor of death and cardiac events in patients with coronary heart disease. (Number of patients are presented on top of each bar). FU, follow-up; MI, myocardial infarction.

on cardiac and non-cardiac mortality remained significant [odds ratio (OR) = 4.1; 95% confidence interval (CI): 1.9–8.8] when adjusting for impaired left ventricular function, three-vessel disease, low exercise tolerance, and the lack of thrombolytic therapy following MI.

In 2000, these results were replicated in an independent sample of 319 patients with established CHD that examined the effect of Type D personality on cardiac mortality and non-fatal MI [29]. Type D was an independent predictor of cardiac mortality and non-fatal MI (OR = 8.9; 95% CI: 3.2–24.7) and also of a composite endpoint of cardiac mortality, non-fatal MI, coronary artery bypass graft surgery (CABG), and percutaneous transluminal coronary angioplasty (PTCA) (OR = 4.5; 95% CI: 2.3–8.5) at 5 years follow-up (Fig. 1, right). Type D personality comprised a risk factor on a par with left ventricular dysfunction for both endpoints. Hence, the consistency of these findings indicates that Type D is an independent predictor of long-term medical endpoints in cardiac patients in general.

Type D and poor prognosis in special groups

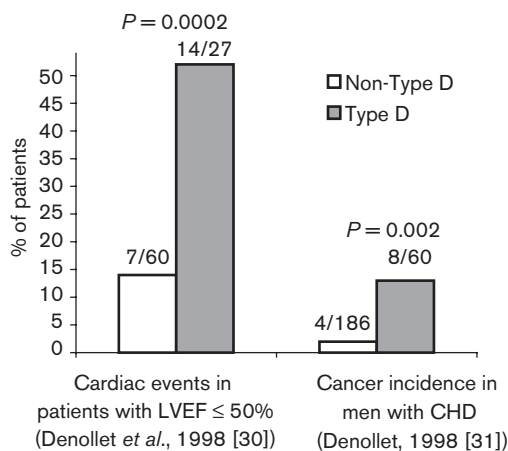
Contrary to previous Type D studies that focused on heterogeneous groups of cardiac patients, a number of studies have focused on Type D and prognosis in special interest groups. One subgroup study focused on patients with a decreased left ventricular ejection fraction (LVEF), a second subgroup studied the risk of cancer in men with CHD, and a third studied victims of sudden cardiac arrest.

The first subgroup study published in 1998 focused on a homogeneous sample of 87 CHD patients with a poor prognosis, as indicated by a LVEF 50% following MI (Fig. 2, left) [30]. Type D was identified as an independent predictor of a composite endpoint of mortality due to cardiac causes and non-fatal MI [relative risk (RR) = 4.7; 95% CI: 1.9–11.8] together with LVEF 30% at a follow-up of 6–10 years (mean = 7.9 years) [30]. In this subgroup study, anxiety, depression, Type B behaviour, and anger did not add to the predictive power of Type D personality in patients with a decreased LVEF [30].

A second subgroup study [31] investigated the ability of the Type D construct to predict the development of cancer in patients with established CHD (Fig. 2, right). Type D (OR = 7.2; 95% CI: 2.9–18.1) and older age (OR = 4.6; 95% CI: 1.5–14.3) were identified as independent predictors of the development of cancer. The development of cancer was unrelated to cardiac severity measured by means of left ventricular dysfunction [31].

A third subgroup study focusing on victims of sudden cardiac arrest (SCA) was also the first to corroborate the

Fig. 2



Type D personality as a long-term predictor of hard medical end-points in patient subgroups. (Number of patients are presented on top of each bar). CHD, coronary heart disease; LVEF, left ventricular ejection fraction.

findings of Type D by an independent research group. Using a proxy for the Type D construct, Appels *et al.* [32] investigated its effect on SCA. Next of kin of the SCA victims were asked to complete the Maastricht Interview Vital Exhaustion (as a measure of negative affectivity of the Type D construct) and a question on the *closedness* (as a measure of social inhibition of the Type D construct) of the SCA victim. Based on the interviews with the next of kin, patients scoring high on negative affectivity and social inhibition were at a seven-fold increased risk of sudden cardiac arrest controlling for standard biomedical risk factors [32]. This suggests that Type D or the proxy that Appels *et al.* [32] used for Type D may be an antecedent of SCA.

Pathophysiological mechanisms

Evidence of mechanisms that may be responsible for the relationship between Type D personality and the pathogenesis of CHD is just beginning to emerge. A preliminary study of 42 men with chronic heart failure has identified interactions with the immune system as one possible mechanism [33]. In the latter study, Type D was identified as an independent predictor of increased circulating levels of pro-inflammatory cytokine tumour necrosis factor (TNF)- α (OR = 9.5, 95% CI 2.1–43.8, $P = 0.004$) and TNF- α soluble receptors 1 and 2 (OR = 6.1, 95% CI 1.4–25.8, $P = 0.014$) adjusting for ischaemic aetiology and severity of heart failure [33]. TNF- α and its soluble receptors have been associated with the pathogenesis of CHF [34,35], with TNF- α receptor 1 emerging as the strongest and most accurate predictor of mortality independent of follow-up duration and clinical variables [35].

Physiological hyper-reactivity may comprise another mechanism. In a study of 173 (mean age = 20.4 years) healthy undergraduates, the inhibition component of the Type D construct was associated with heightened blood pressure reactivity, and both social inhibition (SI) and negative affectivity (NA) were related to greater cortisol reactivity to stress [36]. In the latter study, it was the separate components of the Type D construct rather than the joint effects of the components that were associated with heightened reactivity to stress. Although this is contrary to the results found in CHD patients, it is possible that the synergistic effect of NA and SI only become pronounced with age [36]. As suggested by the authors, a reactivity study with cardiac patients to investigate whether disease status influences the physiological reactivity of Type D may shed further light on this finding [36].

Effect on quality of life and treatment

Taken together, the studies presented above point to a consistent deleterious effect of Type D personality on prognosis in patients with established CHD, and suggest some pathophysiological mechanisms that may explain this link. Type D personality may also have an adverse effect on softer endpoints, like quality of life and the effect of treatment according to self-reported somatic symptoms.

Type D personality and quality of life

There is preliminary evidence to suggest that Type D personality may be a determinant of quality of life (QoL). In a prospective study, Type D patients were at a two-fold (OR = 2.2; 95% CI: 1.2–3.8) increased risk of reporting poor perceived health at 5 years follow-up compared with non-Type D patients [29]. It should be noted, however, that this result is based on a relatively small sample of 104 patients and that larger studies are required to further confirm this association.

In the future, the need to identify determinants of QoL is likely to increase. Mortality rates from CHD have declined considerably in the last two decades due to the proliferation of new treatment options, including new pharmacological, interventional, and surgical therapies [37]. Thus, due to a better prognosis for cardiac patients and an ageing population, the number of patients with CHD is increasing. As a consequence, study endpoints are gradually shifting from the more hard-core endpoints of mortality and re-infarction to softer endpoints such as QoL. With this shift in endpoints a further need for determinants of QoL will ensue, and personality traits may provide much explanatory power of individual differences in QoL in patients with established CHD [38].

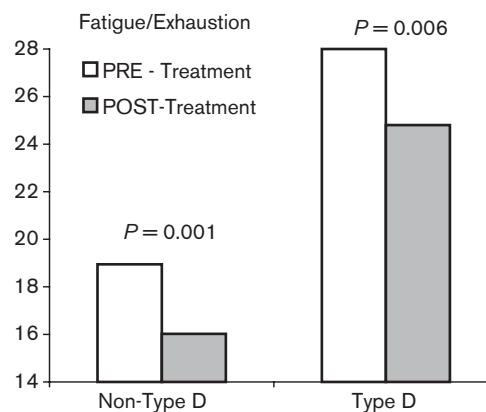
Type D – a moderator of treatment

Type D personality is not only associated with increased emotional distress, cardiac events, and adverse QoL, but

may also moderate the effects of pharmacologic and cardiac invasive treatments [26]. Despite an improvement in symptoms of fatigue and exhaustion following conservative and invasive treatment, Type D patients with ischaemic heart disease were at more than a four-fold risk of feeling fatigued following implementation of conservative or invasive treatment adjusting for type of treatment and symptoms of angina compared with their non-Type D counterparts (Fig. 3). Type D patients also reported more symptoms of angina compared with non-Type D patients following CABG/PTCA or conservative treatment, even though intervention in general resulted in a significant reduction in symptoms of angina [26,38]. In other words, Type D patients benefit from cardiovascular treatment in terms of experiencing a reduction in psychological distress, somatic complaints, and symptoms of angina, but their levels of distress and somatic health complaints are still far exceeding those of non-Type D patients.

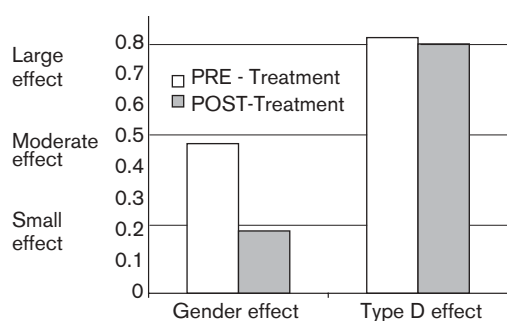
Gender and Type D personality both comprise individual difference variables, but whereas gender is routinely included in cardiac research, personality is largely ignored. Is this exclusion of personality as an individual difference variable justified? One way of approaching this issue is to look at the clinical significance of both Type D personality and gender effects on psychological distress by means of Cohen's [39] effect size index. In the Pedersen and Middel [26] study of 171 patients with ischaemic heart disease, the gender effect on symptoms of fatigue was moderate prior to coronary angiography [effect size (*ES*) = 0.48] and small (*ES* = 0.20) following conservative and invasive treatment (Fig. 4, left). By contrast, the effect of Type D personality on fatigue was large both at baseline (*ES* = 0.82) and following intervention (*ES* = 0.80) (Fig. 4, right). This points to a stable

Fig. 3



Symptoms of fatigue/exhaustion before and after invasive or medical treatment, stratified by Type D personality (Pedersen & Middel, 2001 [26]).

Fig. 4



Effect size estimates indicating the clinical significance of gender and Type D personality as moderators of self-reported fatigue/exhaustion before and after cardiac treatment.

effect of Type D personality on symptoms of fatigue and exhaustion, even following appropriate invasive and medical cardiac treatment. Although this is based on only one study, the results support the contention that personality variables may be important in explaining individual differences in outcome.

Clinical implications

Identification of cardiac patients at risk for adverse health outcomes and modification of this risk comprise the primary objectives of secondary prevention. It is particularly important to be able to identify subgroups of patients in whom psychosocial risk factors tend to cluster, as clustering substantially increases the risk of cardiac events [1]. A recent review on the role of psychological factors in cardiovascular disease has suggested that the identification of patients at risk for clustering may be enhanced if focus is directed at chronic stress in research and in clinical practice [1]. The importance of focusing on chronic stress is further attenuated, if one accepts the notion that chronic psychological risk factors may promote the development of episodic psychological risk factors and influences the manner of responding to acute factors, as proposed by Kop [27]. Personality dispositions tap into chronic attributes of individuals and as such may be of great benefit. This is exemplified by the empirical evidence on Type D personality, which suggests that the taxonomy may have much explanatory power in terms of accounting for individual differences in cardiovascular morbidity and mortality. This evidence also shows that Type D comprises a risk factor on a par with left ventricular dysfunction, a traditional biomedical risk factor for future cardiovascular events in patients with CHD.

Cardiovascular risk factors have also been shown to act in synergy such that the combined risk may be greater than the sum of these factors operating in isolation. There is preliminary evidence to suggest that Type D personality

may also act in synergy with other risk factors. In a study of 319 CHD patients, patients were stratified according to variables that were identified as independent predictors of 5-year prognosis, namely age ≤ 55 years, LVEF $\leq 50\%$, and Type D personality [29]. Patients with two or three risk factors had a four-fold attenuated risk of adverse outcome compared with patients with no or one risk factor present. It is noteworthy that mean estimated medical costs were also almost three times higher (\$10 400 versus \$3600; $P < 0.0001$) in patients with two or three risk factors compared with patients with no or one risk factor present. In other words, psychological risk factors may not only act in synergy with more established risk factors but their presence may also lead to increased medical costs. Depression has also been associated with increased medical costs (41% higher in depressed compared with non-depressed; $P < 0.004$) [40].

Based on the presented evidence, the use of the Type D Personality Scale or the DS14 [41] for risk stratification may identify patients at high risk for clustering of psychosocial risk factors and future cardiovascular events. The brevity of the scale makes it particularly suitable to use as a screening instrument in clinical practice and to include in research protocols, since it poses a minimal burden to patients. However, improvement in outcome not only requires the identification of patients at risk for adverse outcome, but also modification of the factors that pose these patients at risk in the first place. The question is – is Type D personality at all modifiable, since personality is considered to have stable effects on behaviour?

First of all, it is important to bear in mind that just because patients with this psychological profile deal with emotional stress in a particular way, this does not imply that their level of emotional stress cannot be reduced. Secondly, psychosocial interventions targeting general emotional distress, depression, TABP, and anger/hostility have been successful in reducing cardiovascular morbidity and mortality [42–45]. Results from the Recurrent Coronary Prevention Project targeting TABP were particularly encouraging since a follow-up study showed that the effect of psychosocial intervention on the cardiac recurrence rate may be maintained several years post-treatment [46]. Type D patients clearly match this psychological profile. Hence, although interventions specifically directed at modifying Type D personality and its deleterious effects on health outcomes are not yet available, indirect evidence suggests that it is possible to modify the effect of this chronic psychological risk factor.

Conclusion

Type D personality has been associated with increased morbidity and mortality in patients with established

CHD. The construct comprises a risk factor on a par with left ventricular dysfunction – an established biomedical risk factor. However, the construct may also be salient in other chronic medical conditions, as it has been shown to predict depressive effect in hypertensives [17], the development of cancer in men with CHD [31], and increased levels of pro-inflammatory cytokines in patients with heart failure [33]. Type D patients are also at increased risk for impaired quality of life, and seem to benefit less from medical and invasive treatment.

The Type D personality construct was originally developed in Belgian cardiac patients, but evidence is beginning to emerge that this construct is equally applicable to cardiac patients from other nationalities, including Denmark, Germany, Hungary and Italy [47]. As a final note, we would like to stress that this review does not purport to argue that Type D personality is *the* psychological risk factor in the pathogenesis of CHD. Rather, accumulating evidence from this review indicates the urgent need to adopt a personality approach in order to optimize the identification of patients at risk for stress-related cardiac events.

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