Psychological Risk Factors In Heart Disease: What Type D Personality Is (Not) About

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Comments on the article by Pedersen and Middel:
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There is a growing number of studies showing that psychological factors may increase the risk of coronary events and cardiac death [1,2]. Depression in particular is often considered to be the most pathogenic factor. Yet, as pointed out by Carney et al. [3], some fundamental questions about depression as a cardiac risk factor remain unanswered, including the issue whether this risk is restricted to individuals with major depression or also extends to patients with less severe depression. In fact, some studies [4,5] showing that the diagnosis of clinical depression does not add to the predictive power of -quite often- relatively mild levels of depressive symptoms or negative affect [6] force us to pose the question: "Is this risk due to negative affect rather than clinical depression?"

This brings us with respect to depression to the most fundamental issue of all, that is, the fact "…that we know so little about this apparently lethal exposure variable" [7, p. 1731]. Stating that depression is a risk factor for cardiac events because the self-report scale that was used to predict these events includes the term "depression" in its label [4,5] may lead to misleading conclusions. Consider, for example, the observation of Lespérance et al. that “… there was a tendency toward higher cardiac event rates among patients with mild to moderate levels of depressive symptoms on the BDI who did not meet the diagnostic criteria for major depression than among those who did meet the criteria.” [5, p. 1357]. In his 1990 book on personality and disease, Friedman clearly stated that "To be sure that we are spiraling ahead in our understanding rather than circling towards old dead ends, careful and constant evaluations of our concepts is crucial". [8, p. 11]. Obviously, there is an urgent need to address these conceptual issues in psychosomatic research on cardiac disease.

The investigation by Pedersen and Middel [9] published in this issue contributes to the evaluation of two potentially relevant concepts. This study investigated the relationship between Type D personality and vital exhaustion in patients with angiographically documented coronary artery disease. Vital exhaustion represents a mental state characterized by fatigue, demoralization and irritability [10], and has been associated with an increased risk of cardiac events following coronary angioplasty [11]. Type D represents a personality profile characterized by both the tendency to experience negative emotions and the propensity to inhibit self-expression in social interaction, and has also been connected with an increased risk of fatal and non-fatal cardiac events in patients with documented coronary heart disease [12].
Pedersen and Middel [9] investigated whether Type D was a predictor of vital exhaustion in 217 coronary patients who were scheduled for coronary angioplasty (33%), bypass surgery (29%), or conservative medical treatment (38%). As predicted by theory [12], Type D patients were at a substantially increased risk of suffering from vital exhaustion at baseline (OR 6.35). They were, however, also at increased risk of suffering from exhaustion following medical treatment or revascularization (OR 4.74) despite the fact that treatment was successful in reducing angina pectoris. Accordingly, Type D was a significant predictor of vital exhaustion, even when controlling for all other variables including treatment and signs of angina pectoris.

So far for statistical significance, but what about clinical significance? Let’s consider for one moment the effect of gender versus the effect of Type D in the Pedersen and Middel study [9]. Using Cohen’s criteria for effect size [13], the gender effect on vital exhaustion changed from moderate at baseline (effect size= .48) to small following intervention (effect size= .20). In contrast, the effect of Type D personality was large, not only at baseline (effect size= .82) but also following intervention (effect size= .80). Hence, in terms of clinical significance, the personality effect was substantial and very stable as compared with the observed gender effect. Yet, in studies on stress-related disease gender is quite often included as an individual difference variable while personality is not.

Although these findings are impressive in terms of the stability of the personality effect, the Pedersen and Middel study [9] has a number of limitations. The non-response of as many as 35% of the patients screened for inclusion in the study raises the issue of generalizability of findings. But perhaps more importantly, no objective indices were included to control for cardiac variables such as the severity of cardiac ischemia or the extent of coronary artery calcification. Inclusion of these parameters would significantly enhance the study’s findings. Notwithstanding these limitations, the findings are in keeping with the notion that Type D is a marker of distress and that the Type D scale could serve as a screening instrument for patients at risk. In addition, the findings confirm that Type D may moderate the effects of surgical or medical intervention [12].

The Pedersen and Middel study [9] distinguishes itself from many other psychosomatic studies in coronary patients for two reasons. First, it has been argued that psychosomatic research on coronary syndromes needs to investigate both acute and chronic negative emotions [14]. Vital exhaustion is considered a short-term risk factor while Type D (the tendency to experience
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distress) was designed to reflect a chronic psychological condition. Second, it has also been argued that research should not ignore individual differences in coping with these negative emotions [12]. That is, Type D entails that the tendency to inhibit the expression of emotions and behaviors in social interaction is an additional form of disease promoting stress. By addressing both these issues, Pedersen and Middel [9] provide more empirical evidence for the validity of the Type D construct as an explicit theoretical framework of individual differences in stress-related disease.

Pedersen and Middel [9] also correctly point out the fact that the mixed findings from research on the Type A Behaviour Pattern have caused a decline of interest in the notion that broad and stable personality traits may impact on cardiovascular health. As a result, personality is out of fashion in psychosomatic research on coronary heart disease. In addition, there are a number of misunderstandings about Type D that may impede an optimal integration of this personality construct in psychosomatic research [15,16]. Most of these misconceptions deal with three important and related facets of clinical research and practice: the identification of the target population under study, the target of intervention itself, and the feasibility of intervention. A fourth misconception concerns the very nature of the Type D construct itself. It should be clear from the outset that these false interpretations do not refer to Pedersen and Middel [9]; in fact, these authors provided evidence to counter some of these misconceptions.

First, the misconception that Type D further congests the field [15] implies that Type D would not be useful in the process of identifying the target population; i.e., coronary patients at risk for adverse health outcomes. Other investigators have suggested a variety of psychological risk factors in coronary patients, including, negative affect [6], anxiety [17], anger [18], stress [19], and vital exhaustion [11]. Previous research showed that Type D may be a major determinant of negative affect, anxiety, anger, and stress [12]. In their study, Pedersen and Middel [9] were able to show that Type D is associated with a substantially increased risk of suffering from vital exhaustion as well. These findings indicate that Type D may be very helpful in the process of identifying high-risk coronary patients. Findings from two prospective follow-up studies have provided empirical evidence to support this proposition [20,21].

Second, the misconception that Type D claims to be the one and only psychological risk factor that should be accounted for in coronary heart disease [16] implies that Type D would deny the
importance of specific mood states. This claim was never made in any of the Type D articles. Ironically, this misunderstanding was stated in an editorial comment [16] on a Type D article whereas in the actual Type D article [22] it was clearly asserted that: “… it is important to examine a broader scope of psychosocial factors than has previously been considered in relation to CHD” [p. 167, italics added]. In the very same article, it was also stated that: “clinical diagnoses of affective disorder, self-report measures of negative emotions and personality test scores may be independent predictors of adverse cardiac events” and furthermore that: “… the most powerful prediction scheme is likely to be one that incorporates both biomedical and psycho-social factors, including specific emotions and global personality traits” [22, p. 172, italics added]. Rather than ignoring specific negative emotions, Type D aims at the early detection of patients who are at increased risk of experiencing one of these forms of disease-promoting stress. Pedersen and Middel [9] found that Type D patients experienced symptoms of emotional exhaustion across time and situation (i.e., both pre- and post-treatment). Hence, feelings of fatigue, demoralization and irritability may be a focus of intervention in Type D patients.

The third misconception holds that if Type D is a personality type, it can't be changed. This would imply that Type D represents a static view of the role of psychological stress in heart disease. In contrast, the notion that Type D patients may use a characteristic way to deal with emotional stress does not mean that the level of emotional stress in these patients can not be modified. Reducing anger, alleviating depressive symptoms, and improving social relations have been proposed as targets for behavioral and pharmacological interventions in coronary patients [23]. These targets clearly match the psychological profile of Type D patients. However, important differential treatment effects may be overlooked if one considers only averaged findings. Consider, for example, the decrease in vital exhaustion as a function of revascularization or medical treatment [9]. Pedersen and Middel observed that Type D patients were significantly less exhausted post-intervention than at baseline (p=.006); yet at the same time, they still were at a more than fourfold risk of being emotionally exhausted post-intervention compared with non-Type D patients (OR=4.74). What does this mean in terms of intervention? Can emotional stress be modified in Type D patients? Yes, it can. Is the outcome of intervention the same in Type D and non-Type D patients? No, it clearly differs. By analogy, surgical and medical intervention also lead to a significant decrease in angina pectoris in Type D patients (p=.002), but Type D patients still experienced more signs of angina pectoris post-intervention as compared with their non-Type D counterparts (p=.006). There is now some evidence to suggest
that intervention may improve the psychological risk profile in cardiac patients [24]. Inclusion of Type D in clinical research would make it possible to look at (a) individual differences in emotional stress at baseline and (b) ways differential treatment effects may impact on changes in psychological profile and, eventually prognosis.

Fourth, the misconception that the Type D construct represents old wine in new bottles implies that it fails to tell us something new about personality factors in health and disease [e.g., 25]. Of course, we strongly disagree. Consider, for example, the Type A behaviour pattern. This behaviour pattern is often referred to as a personality type; yet this construct was specifically designed to avoid association with global personality traits. As a result, Type A reflects a "heterogeneous hodgepodge" of behavioural symptoms and signs [26]. By analogy, Type C reflects a very heterogeneous mixture of attitudes, cognitions, emotions, verbal and nonverbal expressive patterns as well as coping strategies which all have been related to cancer risk [27]. As indicated by Scheier and Bridges, we should avoid measuring aspects of behaviour or personality that are too wide or poorly defined [28]. Therefore, the Type D construct was specifically designed to refer to a homogeneous subgroup that is defined by the combination of two broad and stable personality traits that have a clear conceptual basis in psychological theory.

There are more relevant differences between Type C coping and Type D personality. Type C predominantly focuses on inhibited self-expression; Type D places additional emphasis on the tendency to experience negative emotions. Type C has been proposed as the opposite of the Type A behaviour pattern; Type D is basically unrelated to the Type A behaviour pattern. Sometimes, Type D is also confused with specific emotional coping styles like repression. However, repression refers to low negative affect due to an unconscious defensive process wherein negative emotions are excluded from awareness [12]; Type D refers to high levels of negative affect and the conscious suppression of this affect as a deliberate strategy to avoid disapproval by others. These major conceptual differences once more illustrate the importance of construct validation and model-building in research on personality, health, and disease [25].

Psychological risk factors for coronary disease may cluster together [1]; multifaceted constructs like Type D may identify patients who are at risk for this clustering of risk factors [12]. Does this mean that Type D is the psychological pathogenic factor in CHD? Certainly not. More than a decade ago, Dimsdale [26, p. 112] wrote about the relation between personality and coronary
disease, stating that: "... the nature of that influence is far more complex than is conveyed by the simple assertion that Type A behavior is a risk factor for coronary heart disease". It would be naïve to assume that Type D would cover all of this complexity. Among other things, the picture provided by the Type D construct is incomplete. It neither specifies mechanisms that mediate the personality-disease relationship [1] nor takes into account the numerous environmental influences that may moderate this relationship [29].

At the same time, new evidence supports the need to adopt a personality approach in the identification of patients at risk for cardiac events. In their sample of Dutch coronary patients, Pedersen and Middel showed that Type D was a strong predictor of vital exhaustion as a form of disease-promoting stress [9]. Using a different methodology, Appels et al. also observed that the combination of “being exhausted” and “being closed” was a significant risk factor for sudden cardiac death in Dutch coronary patients [30]. Murberg et al. found that neuroticism -or negative affectivity- was associated with an increased mortality risk in a two-year follow-up of Norwegian patients with heart failure [31]. In Canada, the study of Habra et al. indicated that social inhibition as measured by the Type D scale was a better predictor of blood pressure reactivity in male students than hostility or social support [32]. Overall, these findings support the Type D model that was originally developed in Belgian patients [20].

Hence, in addition to measuring specific psychological factors in coronary patients such as depression or social support, it is also important to assess constructs that are based on broad, stable dimensions of normal personality [12]. The paper by Pedersen and Middel in this issue [9] clearly provides more evidence for the notion that this line of research has been neglected too much in the past. But most important, these authors found the courage to approach the issue of psychological factors in heart disease with an open mind. According to McCrae [33], openness to experience includes the permeability in the mind for new and different ideas, even if this includes moving beyond the comfortable boundaries of familiar constructs to consider also alternative constructs. Once again, it seems that personality does matter after all.
References


2 Creed F. The importance of depression following myocardial infarction. Heart 1999; 82:406-408.


9 Pedersen SS, Middel B. Increased vital exhaustion among Type D patients with ischemic heart disease. J Psychosom Res 2001; 50: 000-000, in press.


18 Mendes de Leon CF, Kop WJ, de Swart HB, Bär FW, Appels AP. Psychosocial characteristics and recurrent events after percutaneous transluminal coronary angioplasty. Am J Cardiol 1996; 77:252-255.


33 McCrae RR. Openness to experience: Expanding the boundaries of Factor V. Eur J Person 1994; 8:251-272.