Review

Type D personality: the heart, stress, and cortisol

L. SHER

From the Division of Neuroscience, Department of Psychiatry, Columbia University, New York, USA

Summary

Many studies have demonstrated the role of psychosocial and behavioural risk factors in the aetiology and pathogenesis of cardiovascular disorders. Recently, a new personality construct, the type D or ‘distressed’ personality, has been proposed. Type D behaviour is characterized by the joint tendency to experience negative emotions and to inhibit these emotions while avoiding social contacts with others. The observation that cardiac patients with type D personality are at increased risk for cardiovascular morbidity and mortality underlines the importance of examining both acute (e.g. major depression) and chronic (e.g. certain personality features) factors in patients at risk for coronary events. Both type D dimensions (negative affectivity and social inhibition) are associated with greater cortisol reactivity to stress. Elevated cortisol may be a mediating factor in the association between type D personality and the increased risk for coronary heart disease and, possibly, other medical disorders. Studies of the effect of age on hypothalamic-pituitary-adrenal (HPA) function in healthy humans have produced inconsistent results. This may relate to a different prevalence of type D individuals in study samples (i.e. some type D individuals may have alterations within the HPA axis that are similar to HPA axis changes in depressed patients). Further studies of the psychological and biological features of type D individuals may help develop treatment approaches to improve the psychological and physical health of individuals with type D personality.

Introduction

Many studies have demonstrated the role of psychosocial and behavioural risk factors in the aetiology and pathogenesis of cardiovascular disorders. The most well known of these factors is type A behaviour pattern, which includes ambitiousness, aggressiveness, competitiveness, impatience, muscle tenseness, alertness, rapid and empathic vocal style, irritation, cynicism, hostility, and increased potential for anger. Type A individuals are at increased risk for developing coronary heart disease. Recently, a new personality construct, the type D or ‘distressed’ personality, has been proposed. This construct is a result of an investigation of coping styles in men with coronary heart disease. Type D personality subtype is characterized by the joint tendency to experience negative emotions and to inhibit these emotions while avoiding social contacts with others. In other words, the type D personality is a gloomy, anxious, and socially inept worrier. Type D individuals generally have fewer personal ties with other...
people and tend to feel less comfortable with strangers.6

Type D characteristics

Type D individuals score highly on negative affectivity and social inhibition personality dimensions. Negative affectivity is defined as the ‘tendency to experience negative emotions,’ including depressed mood, anxiety, anger, and hostile feelings.9,10 Individuals scoring high on negative affectivity are not only dysphoric but have a negative view of self, report more somatic symptoms, and have an attention bias towards adverse stimuli.6,7 As Denollet6 astutely notes, individuals who score high on negative affectivity seem to scan the world for signs of impending trouble.

Social inhibition is described as ‘the avoidance of potential ‘dangers’ involved in social interactions such as disapproval or non-reward by others.11 Individuals scoring high on social inhibition frequently feel inhibited, tense, uncomfortable, and insecure when encounter with other people. Both negative affectivity and social inhibition are associated with the perception of a socially unsupportive environment.8,12

Type D is defined as the interaction of negative affectivity (which is closely related to neuroticism) and social inhibition.6–8,12 Social inhibition is a moderator: the prevalence of cardiac events for individuals who score high in negative affectivity but low in social inhibition is less than for that for individuals scoring highly in both components. In other words, the type D concept suggests that the way people cope with negative emotions may be as important as the experience of negative emotions per se.8

Personality type D is assessed with a scale that measures negative affectivity and social inhibition.13–15 Each item is rated according to a 5-point Likert scale from 0 (false) to 4 (true). Patients who score high on both negative affectivity and social inhibition, as determined by a median split, are classified as type D. The psychometric qualities and prognostic power of the scale have proven satisfactory in Belgian cardiac patients with Cronbach’s α of 0.89 and 0.82 and test–retest reliability of 0.78 and 0.87 for the Negative Affectivity and Social Inhibition subscales, respectively.14,16 The two-factor structure and the internal consistency of the Negative Affectivity and Social Inhibition subscales were recently confirmed in studies of Danish and German cardiac patients.13,15

The data on the relation of type D personality with mood and anxiety disorders are limited. There is evidence that type D personality is associated with depressive and anxiety symptoms, and with post-traumatic stress disorder.6,13,17 Type D personality may be related to social phobia and panic disorder, because its clinical and biological correlates could be thus attributed. Type D individuals may also have a predisposition to develop avoidant personality disorder.

Type D and cardiac events

The inhibition of emotions has been associated with higher cardiovascular reactivity,18 lower cardiovascular recovery,19 lower heart rate variability,20 and, in the long term, carotid atherosclerosis,21 incidence of coronary heart disease,22 and cardiac mortality.23 In a sample of patients undergoing cardiac rehabilitation, deaths from cardiac causes were increased four-fold in those with type D personality, even after controlling for conventional risk factors.24 This observation was later replicated in an independent sample of more than 300 patients with coronary heart disease.16 Type D was an independent predictor of cardiac mortality and non-fatal myocardial infarction, and also of a composite endpoint of cardiac mortality, non-fatal myocardial infarction, coronary artery bypass surgery, and percutaneous transluminal coronary angioplasty.

A study of cardiac patients with a decreased left ventricular ejection fraction demonstrated that type D personality was an independent predictor of a composite endpoint of mortality due to cardiac causes together with a decreased left ventricular ejection fraction.25 In this study, type B behaviour, depression, anxiety, and anger did not add to the predictive power of type D personality. Appels et al.26 investigated the effect of type D behaviour on sudden cardiac death. Next-of-kin of the sudden cardiac death victims were interviewed. Patients scoring high on negative affectivity and social inhibition were at seven-fold increased risk of sudden cardiac death, after controlling for biomedically risk factors. Type D personality and older age were independent predictors of the development of cancer in patients with coronary heart disease.27

A recent study suggests that type D personality is associated with increased depressive and anxiety symptoms in patients with an implantable cardioverter defibrillator.17 Another recent study investigated the effect of type D personality on the occurrence of adverse events at 9 months in patients with ischaemic heart disease after percutaneous
Type D, stress, and cortisol

Type D individuals tend to experience negative emotions such as depressed mood, anxiety, anger, hostile feelings, and to inhibit these emotions while avoiding social contacts. Situations involving fear, anxiety, helplessness, and loss of control result in release of cortisol. The relationship between negative affect and cortisol activity has been documented in several studies using structured laboratory stressors, such as public speaking and mental arithmetic and aversive stimulation, and in the scientific literature related to changes in the hypothalamic-pituitary-adrenal (HPA) axis in depressed patients. A recent study has documented relationships among negative affect, positive affect and cortisol in response to naturalistic stressors. Both the experience of a current stressor and anticipating a stressor were associated with increased salivary cortisol levels. Negative affect was associated with higher cortisol levels and positive affect was associated with lower cortisol levels. Another study also found that stressful daily events were associated with increased cortisol secretion in healthy volunteers. Distress, as reflected by the mood states ‘negative affect’ and ‘agitation’, was associated with higher cortisol levels. Mood plays a mediating role in the relationship between stressful events and cortisol secretion. Negative affectivity is not just a confounder, but is related to elevated cortisol secretion during normal daily activities. In a recent study, both type D dimensions (negative affectivity and social inhibition) were associated with greater cortisol reactivity to stress, although the results were not significant in more stringent regression analyses. However, it is reasonable to suggest that there is a difference in HPA regulation in type D individuals and in people with other personality types.

Elevated cortisol and medical illness

Depression appears to be an independent risk factor for the development of coronary heart disease and osteoporosis, and affects the prognosis of these and other medical disorders. Considerable evidence suggests an association between depression and hypertension, peptic ulcers, and diabetes. Elevated cortisol may be a mediating factor in these relationships. Cortisol has many effects that promote coronary heart disease. For example, cortisol inhibits the growth hormone and gonadal axes. Growth hormone deficiency is associated with higher relative risk for premature cardiovascular disease in adults. Cortisol is a potent stimulus to visceral fat. Inhibition of the growth hormone and gonadal axes exacerbates visceral fat accumulation. Excess visceral fat leads to dyslipidaemia and, along with hypercortisolism, to insulin resistance, hyperinsulinism, and their sequelae. Similar mechanisms may increase the vulnerability of type D individuals to cardiac and other medical illnesses. Elevated cortisol may be a mediating factor in the association between type D personality and the increased risk for coronary heart disease and, possibly, other medical disorders. It is important to note that cortisol is not the only mediating factor in this association. A recent study suggests that type D personality is associated with increased circulating levels of cytokine tumour necrosis factor α and its soluble receptors 1 and 2, which are predictors of mortality in chronic heart failure.

HPA function, ageing, and type D personality

Depression is associated with impairment in feedback control of the HPA axis, contributing to higher cortisol levels during episodes of depression. Prolonged exposure to elevated cortisol levels may be neurotoxic, especially for brain regions rich in corticosteroid receptors, and may mediate neuronal vulnerability to stressors.
Recurrent depression is associated with atrophy of the hippocampus and amygdala as well as the prefrontal cortex. A gradual deterioration of hippocampal feedback inhibition of the HPA axis due to down-regulation of glucocorticoid receptors from repeated stress has been demonstrated. Evidence suggests that age and/or length of depression and/or the number of depressive episodes affect HPA regulation in depressed patients. The potentiating or additive effect of age in conjunction with depression on pituitary adrenocortical activity was suggested by a number of studies. Mean 24-h cortisol level increases with age in depression. Elderly depressives who are cortisol non-suppressors after dexamethasone need more time for pituitary adrenocortical normalization to occur than do younger subjects. An increase in post-dexamethasone cortisol levels with age has been reported in major depressive disorder. A significant effect of age on cortisol release in depressed patients has been observed during the combined dexamethasone-corticotropin-releasing hormone test: older patients had higher post-dexamethasone cortisol levels. In patients with endogenous depression, advancing age leads to higher baseline cortisol and a greater likelihood of being a dexamethasone non-suppressor. Cortisol responses to fenfluramine administration in depressed patients increased with the number of major depressive episodes. Other authors have reported similar observations.

Studies of the effect of ageing on HPA function in healthy humans have inconsistent results. We recently found that age did not affect cortisol levels in healthy volunteers, consistent with other reports. Advanced age did not appear to affect the overnight dexamethasone suppression in healthy humans. The 24-h mean cortisol concentration and the number of cortisol peaks as well as their amplitude and duration were studied in healthy volunteers, and no difference between younger and older subjects was found. Basal and corticotropin releasing hormone-stimulated adrenocorticotropic hormone (ACTH) and cortisol secretion, as well as sensitivity of the ACTH-cortisol axis to glucocorticoid feedback suppression, were essentially unaltered with age in healthy men. No difference in the results of combined dexamethasone-corticotropin-releasing hormone test was found between younger and older healthy volunteers, and a recent study suggests that ageing has no effect on cortisol responses to fenfluramine administration in healthy elderly subjects.

However, a number of authors suggest that age does affect HPA regulation in healthy humans. Differences in the results of studies have been be explained by differences in a sample size, screening criteria, and some other factors, such as differences in sleeping patterns. Equivocal results of these studies may be, in part, related to a different prevalence of type D individuals in study samples: i.e. some type D individuals may have alterations within the HPA axis that are similar to HPA axis changes in depressed patients. Future studies of HPA function should control for the presence of type D individuals. Type D individuals should perhaps not participate in psychobiological studies as healthy controls. Studies of HPA function should also control for other personality traits that may affect the HPA axis. For example, individuals with borderline or antisocial personality features may have HPA axis abnormalities.

Do type D individuals need treatment?

Individuals with type D personality are at increased risk for developing psychiatric and medical disorders. Type D personality may be regarded as a psychopathological condition that may affect health and longevity, and requires psychological and/or pharmacological treatment. Are there psychological, pharmacological, or alternative medicine interventions that can help type D individuals? Cognitive behavioural therapy, social skills training, emotional support, interpersonal psychotherapy, progressive muscle relaxation, autogenic training, diaphragmatic breathing, guided imagery, various forms of meditation, hypnosis, biofeedback, exercise, and other treatments may all reduce stress in type D persons and improve their ability to socialize. For example, regular exercise may result in decreased anxiety and depression, greater ease in handling daily stress, longer and more restful sleep, improved sexual functioning, improvement in glucose tolerance and lipid parameters, etc. Antidepressants may possibly help some type D individuals. It has been suggested that treatment with selective serotonin reuptake inhibitors may decrease harm avo,dance (a tendency to respond intensely to signals of aversive stimuli), increase social confidence, and decrease hostility. Further studies of psychological and biological features of type D individuals may help develop treatment approaches to improve psychological and physical health of individuals with type D personality.
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