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Challenges in reducing depression-related mortality in cardiac populations: cognition, emotion, fatigue or personality?

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Depression is associated with increased morbidity and mortality in cardiac patients post-event, however treatment of psychological symptoms has failed to reduce cardiovascular risk. We explore depression and related constructs in terms of construct overlap and intervention potential. Certain depressive symptoms may be more important than others for increasing risk. Depressive cognitions may have lesser importance than somatic symptoms, however there is potential for designing interventions based on distorted illness perceptions. Somatic depressive symptoms and vital exhaustion are associated with increased risk. However, these symptoms may be more indicative of cardiovascular disease severity. A global tendency towards negative affectivity could account for the associations seen with negative affect states, with those of Type D personality demonstrating especially high levels of risk for morbidity/mortality. Positive emotion is associated with better outcomes, and could provide clues of how to intervene with negative emotion. Construct overlap and a dearth of theoretically based studies remain significant challenges. Future research needs to encompass all relevant variables to identify the key symptoms or symptom constellations that increase cardiovascular risk. A more sophisticated understanding of these issues can lead to more precise pinpointing of the ‘cardiotoxic’ aspects of psychological status, and ultimately to more refined interventions.

Keywords: coronary heart disease; depression; mortality; intervention; personality; positive emotion

Background

Depression is more common in medical patients compared to the general population. For patients with myocardial infarction (MI), the prevalence of depression post-event is generally reported as more than double that of comparable general population samples (i.e., 15.5–31.1% versus 3.0–7.3%) (Jacobi et al., 2005; Lett et al., 2004; Thombs et al., 2006). While this higher prevalence of depression was initially regarded as an epiphenomenon – an understandable and transient response to a life-threatening event – there is now an increasing realisation not only that depression may have been present prior to the cardiac event, but that depression

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may actually be important in the aetiology and prognosis of coronary heart disease (CHD).

Depression has been found to be a strong predictor both of the development of CHD and of its subsequent prognosis (Barth, Schumacher, & Herrmann-Lingen, 2004; Lett et al., 2004; van Melle et al., 2004). Several reviews and meta-analyses have concluded that depression not only predicts the development of CHD in initially healthy persons (Rugulies, 2002; Wulsin & Singal, 2003), but in those with established CHD, post-event depression is associated with a relative risk of 1.5–2.5 for recurrent fatal or non-fatal events (Lett et al., 2004; van Melle et al., 2004). The majority of these studies have been conducted on post-coronary inpatients with acute coronary syndrome (ACS; MI or unstable angina). Thus, a call has been made for depression to be recognised as one of the most important potentially modifiable prognostic factors in persons with established CHD (Rumsfeld & Ho, 2005).

However, although the treatment of depression in post-coronary patients has reduced the prevalence of detected depression, it has not demonstrated a reduction in subsequent morbidity or mortality. Adding to the non-significant findings of SADHART and ENRICH (Berkman et al., 2003; Glassman et al., 2002; Lesperance et al., 2007; van Melle et al., 2007), the recent MIND-IT trial also produced no effect (van Melle et al., 2007). MIND-IT evaluated the effectiveness of anti-depressants in a randomised controlled trial of over 2000 patients with MI (van Melle et al., 2007). Patients were assessed for depression with diagnostic interviews in-hospital and at three, six, nine and 12 months post-MI. Cardiac events up to 18 months post-MI were assessed. Treating depression did not improve cardiac endpoints (14% in intervention versus 13% control), depression or quality of life measures, and approximately one-third of patients continued to be depressed 18 months post-event. A failure to even reduce depression scores was important, but also echoes some results of the CREATE trial (Lesperance et al., 2007). CREATE demonstrated that short-term treatment of depression with interpersonal psychotherapy was not efficacious in persons with coronary artery disease (although use of a selective serotonin reuptake inhibitor was effective, especially in those with prior depression). These findings further increase the difficulty of intervening with depression in CHD patients – not only are the established depression treatments unsuccessful for reducing depression, they also fail to reduce cardiovascular risk. Thus, van Melle et al. (2007) echoed the call of previous researchers who question the validity and homogeneity of depression in ACS (Frasure-Smith & Lesperance, 2003; van Melle et al., 2007).

The failure of these trials suggests that models of depression based on psychiatric disorder, as is the pattern to date, may not be optimal for the detection and management of depression in CHD populations. Alternative psychological theories of depression, which have yet to be assessed in CHD patients, may point the way to more successful interventions (Davidson, Rieckmann, & Lesperance, 2004). The psychiatric approach, which considers depression as a distinct disorder, may also be less useful than an approach which considers the spectrum of well-being, running from depression to positive health (Keyes, 2004). Depression certainly exhibits a dose–response relationship with CHD morbidity with an observable gradient of risk across the spectrum of depressive symptoms (Bush et al., 2001; Doyle, McGee, De La Harpe, Shelley, & Conroy, 2006; Lett et al., 2004; Rugulies, 2002; Wulsin & Singal, 2003). To date, however, few studies have examined the positive end of the

spectrum: a link between well-being and reduced risk of cardiovascular events. Blazer and Hybels (2004) and Ostir, Markides, Peek and Goodwin (2001a) are two studies that have demonstrated an association between positive affect and decreased risk.

90 An excellent paper by Davidson et al. (2004) outlines how theories of depression may influence the development of cardiovascular disease. The authors suggest that the usefulness (or otherwise) of various psychological treatments for depressive symptoms post-event will differ depending on the proximal cause of depression in each patient. The identification of proximal causes of depression may indicate a difference between those patients with and those patients without spontaneously remitting depression. For example, those patients with cognitive distortions, low positive reinforcement and interpersonal problems are more likely to stay depressed post-event than those who do not display any of these vulnerabilities (Davidson et al., 2004). Thus, treatment could focus on those with non-remitting depression. The interested reader is referred to the original paper for a more in-depth discussion. The present article expands on these ideas by incorporating research on other related psychological constructs that also predict morbidity/mortality in CHD patients when assessed during or after the acute hospitalisation phase. Research on vital exhaustion and Type D personality has also yielded significant results in risk prediction post-event (Appels, 2004; Denollet, 2000; Denollet & Van Heck, 2001; Pedersen & Denollet, 2003), whereas modifying distorted illness perceptions has been shown to reduce angina re-occurrence in patients with MI (Petrie, Cameron, Ellis, Buick, & Weinman, 2002). The extent of overlap between depression and these variables has not been consistently established, and thus previous findings on depression may be limited by not assessing these alternative paradigms. Constructs are defined in Table 1, along with the details of prognostic studies in those with established CHD (examples only are given for depression, as this has been systematically reviewed recently, e.g., Barth et al. (2004), Frasure-Smith and Lesperance (2006) and Nicholson, Kuper, and Hemingway (2006), but systematic searches were conducted for the remaining constructs). In an accompanying editorial to the MIND-IT trial, Carney and Freedland (2007) called for research which could refine our understanding of depression in CHD patients, and research which could pinpoint exact aspects of depression which have harmful effects. Other authors have concluded that a more global measure of negative affectivity could be a more appropriate conceptualisation, and highlight significant overlap between many negative emotions which are currently considered separately in the literature (Suls & Bunde, 2005). Thus, including these related constructs will highlight the most appropriate aspects that can inform interventions to reduce cardiovascular morbidity/mortality post-event. This paper provides a brief outline of the following areas in relation to CHD patients: cognitive distortions (depression and illness perceptions), positive emotion, vital exhaustion (fatigue), Type D personality. This paper briefly addresses the following: (1) a description of each construct; (2) how the construct relates to depression and/or cardiovascular outcomes; and (3) interventions derived from each model and their (potential) treatment for depression (and possible impact on subsequent cardiovascular outcomes).

125 We do not claim that the presented concepts represent the full spectrum of negative affect in CHD patients, but the overlap among these constructs provides a starting point to integrate the literature.

Table 1. Definitions of depression and related constructs, and examples of studies investigating associations with cardiovascular morbidity and/or mortality in samples with established CHD.

Construct	Definition	Study examples	Study design	Effect size (adjusted analysis unless stated otherwise)	Measurement tool	Comments
Depression	Major Depressive Disorder (DSM-IV): one of either depressed mood or anhedonia for two weeks, along with at least four of the following symptoms: weight/appetite loss/gain; insomnia/hypersomnia; psychomotor retardation or agitation; fatigue; feelings of worthlessness or guilt; difficulty concentrating; suicidal ideation	Barth et al. (2004)	Meta-analysis of 20 studies of CHD patients, up to 15 years follow-up	HR =1.76 for mortality two years post-event (OR =ns for mortality within two years post-discharge)	Not applicable	Reported that either interview or questionnaire were equivalent predictors of outcomes
		de Jonge, Ormel, et al. (2006)	2466 MI patients followed for mean 2.5 years	HR =3.91 for somatic/ affective symptoms only	BDI (21-item). Factor analysis divided BDI into cognitive/affective, somatic/affective and appetitive symptoms	Association significant when controlling for other symptoms of depression
		Frasure-Smith et al. (1995)	222 MI patients followed for 18 months	OR =ns for DIS interview (in adjusted analysis) (OR =6.64 (questionnaire)	Modified National Institute of Mental Health DIS (BDI) (21 item)	BDI scale was significant predictor of outcomes at 18 months, but DIS was not
		Nicholson et al (2006)	Meta-analysis of 54 studies ($n = 146,538$) of aetiology and prognosis	RR =1.53 for eight adjusted studies, unadjusted RR =1.80 for 34 prognostic studies	Not applicable	Evidence of publication bias. Non-adjusted results often published, and a substantial attenuation of risk seen after adjustment for left ventricular function

Table 1 (Continued)

Construct	Definition	Study examples	Study design	Effect size (adjusted analysis unless stated otherwise)	Measurement tool	Comments
		van Melle et al. (2004)	Meta-analysis of 6367 MI patients (3343 patients from studies with cardiac mortality as outcome) followed for average 13.7 months	OR =2.59 (unadjusted) for cardiac mortality	Not applicable	Association with mortality was more pronounced in the older studies (OR, 3.22 before 1992) than in the more recent studies (OR, 2.01 after 1992). Reported that either interview or questionnaire were equivalent predictors of outcomes
Vital exhaustion	Feelings of excess fatigue, energy loss, irritability and demoralisation, which appear unrelated to (cardiac) illness severity	Irvine et al. (1999)	671 MI patients followed for two years	RR =1.31 (per unit increase on scale) for association between fatigue and sudden cardiac death	Assessed fatigue with Yale scale (not vital exhaustion), and depression (BDI)	Depression also significant predictor, but inclusion of fatigue attenuated the association of depressive symptoms with SCD
		Koertge et al. (2002)	110 women with MI followed for five years	HR =2.24 for cardiac death for those scoring above median on vital exhaustion	MQ (18 items). This version has more depression items than later versions	Women assessed 3–6 months post-MI. Results adjusted for coronary stenosis and chest pain severity
		Kop, Appels, Mendes de Leon, de Swart, and Bar (1994)	127 PCI patients followed for 1.5 years	OR =2.34 for cardiac morbidity/mortality	MQ (21 items)	Other psychological variables not reported
		Mendes de Leon, Kop, de Swart, Bar, and Appels (1996)	149 PCI patients followed for 1.5 years	RR =2.57 for recurrent cardiac events	MQ (21 item)	Anger was also predictive of events, but composite of anger and vital exhaustion was most predictive

Table 1 (Continued)

Construct	Definition	Study examples	Study design	Effect size (adjusted analysis unless stated otherwise)	Measurement tool	Comments
Type D personality	Tendency to experience increased negative distress and to then inhibit the expression of these negative emotions in social settings, for fear of disapproval or rejection	Pedersen, Denollet, Daemen, et al. (2007)	534 PCI patients followed for two years	HR =2.73 for cardiac events/death for total vital exhaustion score, but fatigue items were non-significant when analysed independently	MQ (21 item)	Divided MQ into fatigue, depression and hopelessness. Depression (adjusted HR =2.69) was cardiotoxic, with hopelessness (adjusted HR =3.36) item most significant
		Denollet et al. (1996)	303 CHD patients followed for 6–10 years	OR =4.1 for mortality	Composite personality questionnaire assessing social inhibition and negative affectivity analysed (using median split) via theoretical and statistical procedures	Type D predictive of cardiac and non-cardiac mortality. Type D associated with depression
		Denollet and Brutsaert (1998)	87 MI patients followed for 6–10 years	RR =4.7 for cardiac events	Type D assessed by combination of various scales (using 75 th percentile as cut-off for anxiety and anger scales).	Specific emotions (depression, anxiety, anger) did not add to the predictive power of Type D
		Denollet et al. (2000)	319 cardiac patients followed for five years	OR =8.9 for cardiac death or non-fatal myocardial infarction	DS16 (16-item scale assessing Type D personality), ZDS and Dutch version of STAI assessed depression and anxiety	Depression and anxiety non-significant in multivariate analysis

Table 1 (Continued)

Construct	Definition	Study examples	Study design	Effect size (adjusted analysis unless stated otherwise)	Measurement tool	Comments
Positive affect	Positive moods or emotions characterised by engagement and energy (although no fixed definition in the literature, and measurement tools vary widely and may include optimism)	Denollet, Pedersen, Ong, et al. (2006) and Pedersen et al. (2004)	875 PCI patients followed for nine months	HR = 1.92 for major adverse cardiac event in those with high negative affectivity <i>and</i> high inhibition (Type D) only. OR = 5.31 for MI/mortality	DS14 (14-item scale assessing Type D personality). HADS (7-item depression and seven-item anxiety scales) assessed negative affect (anxiety and depression). Scales completed six months post-PCI	Negative affect did not explain the association with increased risk
		Denollet, Pedersen, Vrints, and Conraads (2006)	337 CHD patients followed for five years	OR = 2.9 for cardiac death, MI or revascularisation	DS16, GHQ (12-item)	Controlled for psychological stress symptoms (GHQ)
		Pedersen, Denollet, Ong, et al. (2007)	358 PCI patients treated with drug-eluting stents	HR = 2.61 for cardiac morbidity or mortality	DS14 (14 items)	Controlled for history of coronary artery disease, multivessel disease, diabetes, hypercholesterolemia, hypertension, renal impairment and smoking
		Barefoot et al. (2000)	1250 patients with coronary artery disease diagnosed by coronary angiography	HR = 1.10 (ns) for (reverse-scored) well-being, adjusted for negative affect and somatic symptoms	Zung Self-rating Depression Scale (20-item), factor analysed into well-being, negative affect, somatic and appetite symptoms. Hopelessness assessed by one MMPI item	Negative affect (HR = 1.30), continued to predict mortality in adjusted analysis, but other constructs did not. Marginal significant for hopelessness item (HR = 1.40, $p = 0.06$) when controlling for negative affect in analysis of 867 patients

Table 1 (Continued)

Construct	Definition	Study examples	Study design	Effect size (adjusted analysis unless stated otherwise)	Measurement tool	Comments
		Brummett et al. (2005)	866 PCI patients followed for mean 11.4 years	HR =0.8, but HR =ns when controlling for depression	NEO-PI eight-item positive emotion and eight-item depression scales	Depression remained significant when controlling for positive emotion

Note: BDI, Beck Depression Inventory; CES-D, Centre for Epidemiological Studies Depression Scale; DIS, Diagnostic interview schedule; DS14, DS16, Type D personality scales; DSM-IV, Diagnostic and Statistical Manual for Mental Disorders – IV; GHQ, General Health Questionnaire; HADS, Hospital Anxiety and Depression Scale; HR, hazard ratio; IPQ, Illness Perception Questionnaire; MIVE, Maastricht interview for vital exhaustion; MQ, Maastricht Questionnaire (assesses vital exhaustion); NEO-PI, Neuroticism, openness, extraversion personality inventory; ns, non-significant; OR, Odds ratio; PCI, percutaneous coronary intervention; RR, relative risk; STAI, State-trait anxiety inventory; ZDS, Zung Depression Scale.

Cognitions – depression

135 Much of the research into the association between depression and CHD has been
atheoretical, providing no clear insight into how interventions may be derived from
the findings. Few studies have been conducted on the antecedents of depression in
CHD patients. There also remains the question of whether depression itself
140 influences outcomes, or whether outcomes are influenced by some other aspect of
somaticised symptoms of disease severity (Lane, Carroll, Ring, Beevers, & Lip, 2000;
Lane, Carroll, Ring, Beevers, & Lip, 2002; Lane, Lip, & Carroll, 2004). The proximal
causes of depression in post-coronary patients may influence the development of
CHD, the usefulness of a particular treatment, and the potential for spontaneous
remission in this population (Davidson et al., 2004).

Cognitive theory of depression

145 The cognitive model is one of the most commonly utilised models of depression
(Beck, 1991, 2005; Kanter, Callaghan, Landes, Busch, & Brown, 2004). It evolved
through an observation of depressed patients' verbalisation of thoughts. Beck (1991,
2005) and Beck, Rush, Shaw, and Emery (1979) noted that depressed patients had
150 various distorted thoughts and beliefs, and that negative mood was usually a
consequence of these thoughts. Cognitive theory forms a model where distorted self-
concepts (which may be latent) determine personal meaning in several situations, but
increase vulnerability to depression. Stressful life events can then trigger depressive
episodes.

Associations with depression and CHD

155 Support for this theory is seen in a large body of research which has demonstrated
that vulnerable individuals process information dysfunctionally (Alloy et al., 2000;
Beck et al., 1979; Dobson & Ottenbreit, 2004; Ingram, Odom, & Mitchusson, 2004).
Cognitive models of depression have been investigated in cardiac patients (Berkman
et al., 2003; Carney et al., 2004; de Jonge, Ormel, et al., 2006; Dijkstra et al., 2002;
160 Rieckmann et al., 2006; Roberts, 1989; Shnek, Irvine, Stewart, & Abbey, 2001).
Rieckmann et al. (2006) demonstrated that mildly to moderately/severely depressed
patients with ACS had significantly higher levels of dysfunctional attitudes
compared to non-depressed ACS patients. Although depression has been associated
with morbidity and mortality following CHD, subsequent research questioned
165 whether cognitive symptoms of depression were associated with mortality, or
whether only somatic symptoms predicted outcomes (Table 1) (de Jonge, Ormel, et
al., 2006; Doyle et al., 2006). Furthermore, there is also evidence to suggest that
symptoms of depression in post-MI patients are both quantitatively and qualitatively
different from symptoms observed in psychiatric patients, with cardiac patients
170 showing fewer distorted cognitions (Martens et al., 2006).

Interventions

Cognitive therapy assumes that thought precedes mood, and treatment enables
patients to correct false self-beliefs that lead to depressive moods or behaviours
(Beck, 1991; Beck et al., 1979). Patients learn to substitute healthier thoughts for

175 distorted thoughts, and mood improves. Cognitive therapy is a well-established and
successful treatment for depression (Butler, Chapman, Forman, & Beck, 2006).
Subsequent manifestations of cognitive therapy also included some behavioural
180 principles, evolving to cognitive-behavioural therapy (CBT) (Kanter et al., 2004).

The ENRICHD trial attempted to determine whether mortality and recurrent
185 infarction were reduced by treating depression and low perceived social support with
CBT (supplemented with antidepressants when indicated) (Berkman et al., 2003).
There was no significant difference in morbidity or mortality between usual care and
intervention groups over an average follow-up of 29 months, even though a
significant reduction in depressive symptoms was seen. The authors concluded
190 that a substantial improvement in depression in the control group could have hidden
the effects of the intervention. However, alternative explanations are also plausible.
The spontaneous remission of depressive symptoms in the control group may, for
example, indicate that much of the depression measured in ENRICHD was a
relatively transient response to a major negative life event which resolved
spontaneously over time – more akin to adjustment disorder than major depression,
195 minor depression or dysthymia (Frasure-Smith & Lesperance, 2003). Furthermore,
since major depressive disorder is a heterogeneous disease, it may be that only a
proportion of the treatment group benefited from improved cardiovascular outcomes
(i.e., a sub-group for whom CBT was most appropriate, other sub-groups may have
benefited from interpersonal therapy or operant therapy) (Davidson et al., 2004).
Indeed, Rieckmann et al. (2006) demonstrated that depressed ACS patients exhibited
cognitive, interpersonal and behavioural vulnerabilities to depression, and that these
200 vulnerabilities were independently associated with depression. Therefore, the trial
would not have been powered to find the difference in the sub-group for which it was
most suited. Also, since cognitive depressive symptoms are not associated with
subsequent mortality (de Jonge, Ormel, et al., 2006; Doyle et al., 2006), it is perhaps
unsurprising that the treatment of these symptoms through CBT had no impact on
cardiovascular outcomes.

205 **Cognitions – illness**

Self-regulatory model (SRM) of illness perceptions

Another area of cognitive distortions (related to illness) has shown promise in the
development of effective interventions, and is based on the self-regulatory model
(SRM) of illness perceptions (Leventhal, Diefenbach, & Leventhal, 1992; Leventhal,
210 Meyer, & Nerenz, 1980; Weinman & Petrie, 1997; Weinman, Petrie, Moss-Morris, &
Horne, 1996). This theory postulates that individuals are active problem solvers, who
make cognitive and emotional representations of illness in order to make sense of its
potential threat to their health, and then determine how to respond (Fortune,
Richards, Griffiths, & Main, 2004; Horne & Weinman, 1999; Leventhal, Leventhal,
& Cameron, 2001; Moss-Morris et al., 2002). These coping behaviours are then
215 reappraised in terms of efficacy (e.g., reducing symptoms) along with the illness
representation. The model thus combines three stages and a continuous feedback
circuit (Broadbent, Petrie, Main, & Weinman, 2006; Horne, 1997; Leventhal et al.,
2001). Illness representations are made up of several components (see Table 1).
'Distorted' or incorrect illness perceptions may have implications for how patients

220 behave during secondary prevention. For example, if a patient believes that a virus
caused their heart attack (despite repeated explanations otherwise), they may be less
likely to adhere to anti-hypertensive medication.

Associations with depression – theoretical commonalities

225 There are potential theoretical links between depression and illness perceptions.
First, diagnosis with a life-threatening illness is a common antecedent of depression,
and of course may be the cause of new or evolving illness perceptions, as with CHD
patients. Second, there is the possibility that both depression and illness perceptions
are evolving not only from the same threatening life event, but also at the same time,
230 each influencing the other (e.g., depressed patients are known to catastrophise illness
symptoms and demonstrate poor psychological adjustment (Moss-Morris, Petrie, &
Weinman, 1996; Petrie, Moss-Morris, & Weinman, 1995)). Illness representations
can be formulated and modified at either or both of two stages of illness appraisals:
disease occurrence (cause) or disease management (coping) (Schiaffino & Cea, 1995;
Weinman et al., 1996). Therefore, potential depressive-related distortions could
235 occur during either or both of these phases. Third, it is possible that people who
blame themselves for negative events will also blame themselves for their CHD (a
cognition that could be reinforced by explanations of lifestyle factors as being key
risk factors for CHD). Indeed, some researchers have stated that individual
differences in adjustment to illness can be better explained by a single dimension –
maladaptive thinking – than by specific illness cognitions (Evers et al., 2001).

240 Research has also demonstrated that depression and illness perceptions predict
similar health behaviours in patient populations, such as non-adherence to medica-
tion and non-attendance at cardiac rehabilitation programmes (French, Cooper, &
Weinman, 2006; Horne & Weinman, 1999; Ziegelstein et al., 2000). The items of the
Brief Illness Perception Questionnaire (IPQ) (Broadbent et al., 2006) are listed in
245 Table 2, alongside symptoms of depression assessed by various scales and interviews,
including the diagnostic classifications of DSM-IV and ICD-10. Table 2 demon-
strates potential overlap between the constructs outlined in this paper. In the case of
illness perceptions, it could be argued that up to five of the nine items assess
depressive cognitions (including the symptom of depressed mood). Thus, potential
250 interactions between illness perceptions and depression deserve critical investigation.

Associations with depression – research findings

255 Researchers have shown significant associations between various illness perceptions
and negative affectivity (Broadbent et al., 2006; Moss-Morris et al., 2002). Other
studies have specifically related distorted illness perceptions to depression in CHD
(French, Lewin, Watson, & Thompson, 2005; Grace, Krepostman, et al., 2005).
Furthermore, depression as an illness can also be considered within the SRM.
Fortune et al. (2004) studied the SRM of depression in a convenience sample of 101
women with current or past depression. Beck Depression Inventory (BDI) scores
260 correlated significantly with scales of the IPQ (which assesses the SRM (Weinman et
al., 1996)), and the IPQ was also able to discriminate between women who were and
those who were not depressed. In terms of IPQ subscales, currently depressed women
had a stronger depression identity, a more chronic timeline, less perceived control

Table 2. Comparison of overlapping symptoms of various constructs by questionnaire/interview.

Symptom type assessed by interviews or scales	DSM-IV Major Depressive Disorder (American Psychiatric Association, 2000)	ICD-10 (Mild, moderate or severe depressive episode) (World Health Organisation, 1993)	Maastricht Interview for Vital Exhaustion (Meesters & Appels, 1996)	BDI-II (Beck et al., 1996)	Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977)	Hospital Anxiety and Depression Scale (depression subscale) (Zigmond & Snaith, 1983)	Brief-Illness Perception Questionnaire (Broadbent et al., 2006)	Global Mood Scale (positive and negative affect rated on a five-point Likert scale) (Denollet, 1993)	DS-14 (Type D personality) (Denollet, 2005)
Significant depressed mood	Significant depressed mood	Depressed mood	(5) Do you feel dejected? (20) Do you sometimes cry or feel like crying?	(1) Sadness. (10) Crying	(3) I felt that I could not shake off the blues even with help from my family or friends. (6) I felt depressed. (12) I was happy. (17) I had crying spells. (18) I felt sad	(6) I feel cheerful	(8) How much does your illness affect you emotionally? (e.g., does it make you angry, scared, upset or depressed?	(17) Cheerful	(4) I often feel unhappy. (7) I take a gloomy view of things. (9) I am often in a bad mood. (13) I am often down in the dumps
Anhedonia	Loss of interest or pleasure (anhedonia)	Loss of interest and enjoyment (anhedonia)	(15) Have you noticed a decrease in your sexual appetite or a decrease in the desire to make love?	(4) Loss of pleasure. (12) Loss of interest. (21) Loss of interest in sex	(16) I enjoyed life	(2) I still enjoy the things I used to enjoy. (4) I can laugh and see the funny side of things. (10) I have lost interest in my appearance. (14) I can enjoy a good book, radio or TV programme			

Table 2 (Continued)

Symptom type assessed by interviews or scales	DSM-IV Major Depressive Disorder (American Psychiatric Association, 2000)	ICD-10 (Mild, moderate or severe depressive episode) (World Health Organisation, 1993)	Maastricht Interview for Vital Exhaustion (Meesters & Appels, 1996)	BDI-II (Beck et al., 1996)	Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977)	Hospital Anxiety and Depression Scale (depression subscale) (Zigmond & Snaith, 1983)	Brief-Illness Perception Questionnaire (Broadbent et al., 2006)	Global Mood Scale (positive and negative affect rated on a five-point Likert scale) (Denollet, 1993)	DS-14 (Type D personality) (Denollet, 2005)
Anergia, fatigue or energy loss	Loss of energy or fatigue	Reduced energy leading to increased fatigue and diminished activity (anergia)	(1) Do you often feel tired? (2) Do you feel listless? (3) Do you feel weak all over or without energy? (6) Do you sometimes have the feeling that your body is like a battery that is losing its power? (7) Do you have the feeling that you have not been accomplishing much lately? (8) Do you ever wake up with a feeling of exhaustion and fatigue? (9) Do you wake up repeatedly during the night?	(15) Loss of energy. (20) Tiredness or fatigue	(7) I felt that everything I did was an effort. (20) I could not get 'going'	(8) I feel as if I am slowed down		(1) Weary. (2) Active. (3) Worn out. (4) Dynamic. (7) Hard-working. (8) Feeble. (9) Lively. (10) Physically weak. (11) Listless. (12) Tired. (13) Enterprising. (18) Fatigued. (19) Weakened	

Table 2 (Continued)

Symptom type assessed by interviews or scales	DSM-IV Major Depressive Disorder (American Psychiatric Association, 2000)	ICD-10 (Mild, moderate or severe depressive episode) (World Health Organisation, 1993)	Maastricht Interview for Vital Exhaustion (Meesters & Appels, 1996)	BDI-II (Beck et al., 1996)	Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977)	Hospital Anxiety and Depression Scale (depression subscale) (Zigmond & Snaith, 1983)	Brief-Illness Perception Questionnaire (Broadbent et al., 2006)	Global Mood Scale (positive and negative affect rated on a five-point Likert scale) (Denollet, 1993)	DS-14 (Type D personality) (Denollet, 2005)
Weight/appetite loss/gain	Substantial weight/appetite loss/gain	Diminished appetite		(18) Changes in appetite	(2) I did not feel like eating; my appetite was poor				
Disturbed sleep patterns	Insomnia or hypersomnia	Disturbed sleep		(16) Changes in sleep pattern	(11) My sleep was restless				
Psychomotor retardation/agitation	Psychomotor retardation/agitation	Considerable distress or agitation, retardation (severe depressive episode)		(11) Agitation					
Feeling worthless, guilty	Feelings of worthlessness or excessive guilt	Reduced self-esteem and self-confidence. Ideas of guilt and unworthiness	(14) Do you have the feeling these days that you just do not have what it takes anymore?	(3) Past failure. (5) Guilty feelings (6) Punishment feelings. (7) Self-dislike. (8) Self-criticalness. (14) Worthlessness	(4) I felt that I was just as good as other people. (9) I thought my life had been a failure			(15) Insecure. (20) Self-confident	

Table 2 (Continued)

Symptom type assessed by interviews or scales	DSM-IV Major Depressive Disorder (American Psychiatric Association, 2000)	ICD-10 (Mild, moderate or severe depressive episode) (World Health Organisation, 1993)	Maastricht Interview for Vital Exhaustion (Meesters & Appels, 1996)	BDI-II (Beck et al., 1996)	Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977)	Hospital Anxiety and Depression Scale (depression subscale) (Zigmond & Snaith, 1983)	Brief-Illness Perception Questionnaire (Broadbent et al., 2006)	Global Mood Scale (positive and negative affect rated on a five-point Likert scale) (Denollet, 1993)	DS-14 (Type D personality) (Denollet, 2005)
Difficulty concentrating or indecisiveness	Diminished ability to think or concentrate, or indecisiveness	Reduced concentration and attention	(16) Has it become harder lately to solve a mental task or problem that requires much concentration? (17) Do you have increasing difficulty in concentrating on a single subject for long?	(19) Concentration difficulty. (13) Indecisiveness	(5) I had trouble keeping my mind on what I was doing			(5) Bright	
Suicidal ideation	Suicidal ideation, recurrent thoughts of death or attempted suicide	Ideas or acts of self-harm or suicide	(23) Do you want to be dead at times?	(9) Suicidality					
Hopelessness		Bleak and pessimistic views of the future	(19) Do you believe that you have come to a 'dead end'? (22) Did you experience a feeling of hopelessness recently?	(2) Pessimism	(8) I felt hopeful about the future	(12) I look forward with enjoyment to things	(2) How long do you think your illness will continue? (4) How much do you think your treatment can help your illness?	(6) Helpless	

Table 2 (Continued)

Symptom type assessed by interviews or scales	DSM-IV Major Depressive Disorder (American Psychiatric Association, 2000)	ICD-10 (Mild, moderate or severe depressive episode) (World Health Organisation, 1993)	Maastricht Interview for Vital Exhaustion (Meesters & Appels, 1996)	BDI-II (Beck et al., 1996)	Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977)	Hospital Anxiety and Depression Scale (depression subscale) (Zigmond & Snaith, 1983)	Brief-Illness Perception Questionnaire (Broadbent et al., 2006)	Global Mood Scale (positive and negative affect rated on a five-point Likert scale) (Denollet, 1993)	DS-14 (Type D personality) (Denollet, 2005)
Irritability		Irritability	(4) Do you blow up more easily than before? (12) Do little things irritate you more lately than they used to? (13) Do minor hassles irritate you more easily the last few months?	(17) Irritability	(1) I was bothered by things that usually don't bother me				(5) I am often irritated
Worry or anxiety					(10) I felt fearful		(1) How much does your illness affect your life? (6) How concerned are you about your illness?	(14) Relaxed	(2) I often make a fuss about unimportant things. (12) I often find myself worrying about something

Table 2 (Continued)

Symptom type assessed by interviews or scales	DSM-IV Major Depressive Disorder (American Psychiatric Association, 2000)	ICD-10 (Mild, moderate or severe depressive episode) (World Health Organisation, 1993)	Maastricht Interview for Vital Exhaustion (Meesters & Appels, 1996)	BDI-II (Beck et al., 1996)	Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977)	Hospital Anxiety and Depression Scale (depression subscale) (Zigmond & Snaith, 1983)	Brief-Illness Perception Questionnaire (Broadbent et al., 2006)	Global Mood Scale (positive and negative affect rated on a five-point Likert scale) (Denollet, 1993)	DS-14 (Type D personality) (Denollet, 2005)
Other symptoms			(10) Do you have the feeling that you cannot cope with everyday problems as well as you used to? (11) Do you feel you want to give up trying? (18) Do you shrink from your regular work as if it were a mountain to climb? (21) Do you feel defeated or disillusioned?		(14) I felt lonely. (15) People were unfriendly. (19) I felt that people disliked me		(3) How much control do you feel you have over your illness? (5) How much do you experience symptoms from your illness? (7) How well do you feel you understand your illness? (9) Please list in rank-order the three most important factors that you believe caused your illness		

Table 2 (Continued)

Symptom type assessed by interviews or scales	DSM-IV Major Depressive Disorder (American Psychiatric Association, 2000)	ICD-10 (Mild, moderate or severe depressive episode) (World Health Organisation, 1993)	Maastricht Interview for Vital Exhaustion (Meesters & Appels, 1996)	BDI-II (Beck et al., 1996)	Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977)	Hospital Anxiety and Depression Scale (depression subscale) (Zigmond & Snaith, 1983)	Brief-Illness Perception Questionnaire (Broadbent et al., 2006)	Global Mood Scale (positive and negative affect rated on a five-point Likert scale) (Denollet, 1993)	DS-14 (Type D personality) (Denollet, 2005)
Social inhibition					(13) I talked less than usual			(16) Sociable	(1) I make contact easily when I meet people. (3) I often talk to strangers. (6) I often feel inhibited in social interactions. (8) I find it hard to start a conversation. (10) I am a closed kind of person. (11) I would rather keep other people at a distance. (14) When socialising, I do not find the right things to talk about

AQ2

Note: DSM-IV-TR. Five or more symptoms during the same two-week period, at least one symptom being depressed mood or anhedonia. ICD differentiation between mild, moderate and severe. Mild: at least two of main symptoms, plus at least two others (present for at least two weeks). Moderate: at least two of the main symptoms, plus at least three (preferably four) of the other symptoms. Severe: all three of the typical symptoms, plus at least four other symptoms. Considerable distress/agitation, unless retardation is a marked feature. Some items may be negatively scored. The following symptom clusters have been strongly associated with increased cardiovascular risk in adjusted analyses: depressed mood; anhedonia; fatigue; hopelessness; social inhibition.

265 and more negative scores on the consequences subscale. In terms of the IPQ
causation subscale, currently depressed women were significantly more likely to
endorse a self-blame item ('My illness is largely due to my own behaviour'). This item
may be important for establishing a link between depression and illness cognitions.

270 A recent cross-sectional study investigated associations between illness percep-
tions, depression and gender in 661 patients with ACS (Grace, Krepostman, et al.,
2005). For women, higher levels of depressive symptoms were related to perceiving a
chronic timeline, whereas for men more depressive symptoms related to perceiving a
chronic timeline, more negative consequences, and lower cure/control (Grace,
Krepostman, et al., 2005). Furthermore, those who believed that psychological
275 factors, such as stress or personality, caused their ACS were more likely to be
depressed. Illness perceptions accounted for a significant variation in depressive
symptoms in this study, but prospective studies are needed to investigate whether
these differences in perceptions reflect poorer outcomes in the depressed ACS
population.

Associations with CHD

280 The validity of the SRM, or any model, in CHD depends on its ability to predict
illness outcomes and/or behaviours. Investigation of the SRM has provided some
very interesting findings in samples with CHD. Illness perceptions have been
particularly successful in predicting psychosocial outcomes, such as return to work
and quality of life (Broadbent et al., 2006; French et al., 2005; Petrie, Weinman,
285 Sharpe, & Buckley, 1996). However, results of studies with large sample sizes have
been contradictory, indicating that further work is needed to clarify this area.

Theorists suggest that illness severity should affect illness cognitions, i.e., more
symptoms or more severe symptoms should lead to stronger illness identity (Moss-
Morris et al., 2002; Weinman et al., 1996). However, this may not apply if patients'
290 perceptions of illness are inaccurate and do not match object measures of disease
severity (Frostholm et al., 2005, 2007). One large prospective survey of CHD patients
has shown that the most important correlate of illness perceptions was disease
severity, and disease severity also predicted illness perceptions at one-year follow-up
(Aalto, Heijmans, Weinman, & Aro, 2005). However, this study showed that all
295 cognitive illness perceptions were associated with disease severity, not just illness
identity, which may also have implications for depressive cognitions and disease
severity.

Weinman et al. (1996) demonstrated that the IPQ had predictive validity for a
sample of 143 patients with MI. Three- and six-month outcomes were predicted by
300 various illness perceptions, including self-rated health, self-ratings of risk of further
MI, self-ratings of control of heart problems, and number of doctors' visits. A meta-
analysis of eight studies of 906 patients demonstrated that four illness perceptions
predicted cardiac rehabilitation attendance (French et al., 2006). Patients with MI
who viewed their condition as controllable, as symptomatic, and with severe
consequences, and who felt that they understood their condition were more likely
305 to attend. However, study effect sizes were small ($r = 0.081 - 0.123$).

Weinman, Petrie, Sharpe, and Walker (2000) demonstrated that if patients
believed that lifestyle variables were the causal factors in their MI, then these patients
were more likely to adopt recommended behavioural changes. However, not all

310 studies have shown significant associations between illness perceptions and
behaviours in CHD patients. Byrne, Walsh and Murphy (2005) conducted a large
cross-sectional study of 35 randomly selected general practices, sampling patients
with established CHD ($n=1084$). Illness perceptions and self-report lifestyle factors
315 (e.g., smoking, exercise, diet) were assessed. Illness perceptions appeared to be poor
predictors of these behaviours, accounting for only 2% of the variance. One distinct
aspect of this research was that illness perceptions were assessed during the chronic
phase of illness (mean duration since diagnosis was seven years), whereas most IPQ
research has focused on cardiac patients during acute phases, e.g., post-MI. To date,
320 no study has identified whether illness perceptions are differentially associated with
cardiovascular risk. Prospective studies are needed to determine the relationship
between illness perceptions, depression and cardiovascular risk.

Interventions

325 Although illness perceptions may be inconsistent predictors of health outcomes in
CHD, this area of research has already demonstrated promise regarding interven-
tions to reduce morbidity. A randomised controlled trial has shown that an
intervention based on illness perceptions could modify medical outcomes (angina
frequency) in cardiac rehabilitation patients (Petrie et al., 2002). This study examined
whether a brief hospital intervention designed to alter patients' perceptions about
330 their MI would result in a better recovery and reduced disability. Sixty-five patients
with a first MI were assigned to receive the intervention or usual care from
rehabilitation nurses. At three-month follow-up, the intervention resulted in
significant positive changes in patients' views of their MI. Of importance however,
patients in the intervention group (14.3%) were significantly less likely than control
335 subjects (39.3%) to report angina pain at three months. Furthermore, the difference
was not due to cardiac rehabilitation attendance, as attendance did not differ
significantly between groups. This study provides evidence for the effect of
psychosocial interventions on morbidity outcomes. The results need to be replicated,
and the effects of other psychosocial outcomes (such as the potential interaction with
340 depressive symptoms) need to be assessed in larger trials.

Associations between illness cognitions and depression suggest that there could
be a relationship between poorer coping in depressed CHD patients and specific
illness cognitions. Significant associations between the two may point to potential
interventions, as one mechanism by which depression increases cardiovascular risk
345 may be through poorer adherence to recommendations provided in secondary
prevention (Carney, Freedland, Miller, & Jaffe, 2002; Ziegelstein et al., 2000). Thus,
depression may interact with illness perceptions to further attenuate adherence to
medical advice.

Positive emotion

350 Considering depression as a categorical disorder may be less useful than one which
considers a spectrum of well-being, from depression to positive mental health
(Keyes, 2004, 2005; Seligman & Csikszentmihalyi, 2000; Seligman, Parks, & Steen,
2004). There is now a more general research drive focusing on 'positive psychology':
consideration of positive aspects of human capacity and function and their effects on

355 health and other outcomes, rather than simply addressing those who have
pathological negative emotional states (Keyes, 2004; Seligman & Csikszentmihalyi,
2000; Seligman et al., 2004). In this way, positive psychology attempts to address a
perceived imbalance in research that has hitherto focused largely on understanding
and treating suffering. Research in this area can help identify the skills for coping
well with life, and teach these skills to those who do not cope so well. Positive
360 emotion has been identified as one of the core constituents of happiness (Seligman et
al., 2004). However, a significant limitation in the positive emotion literature is that
there is no widely accepted definition of what constitutes positive emotion, and
several studies confound positive emotion with a related construct, optimism
(Pressman & Cohen, 2005; Russell & Carroll, 1999). Table 2 contains items from
365 the Global Mood Scale (GMS) (Denollet, 1993), which was developed to assess both
positive and negative mood in cardiac populations, and has been shown to be
responsive to change after cardiac rehabilitation (Hevey, McGee, & Horgan, 2004).
The positive items generally assess energy, relaxation and sociability. These seem to
be the opposite of the fatigue items (this is discussed further in the section on vital
370 exhaustion). However, there is some debate in the literature about whether positive
emotion is truly the opposite of negative emotion, or whether it is orthogonal (i.e.,
bivariate). Most researchers favour a bivariate explanation (Pressman & Cohen,
2005; Russell & Carroll, 1999).

375 A recent theory of positive emotions hypothesises an important role for
positive affect in terms of the potential benefit in treating negative emotion.
Fredrickson (1998, 2001, 2002) proposed the 'broaden-and-build' theory, and treats
positive emotions as separate to negative emotions (i.e., as bivariate). She states
that positive emotions broaden an individual's momentary thought-action
380 repertoires, and consequently build the individual's enduring personal resources,
which then lead to better coping with subsequent life events. An example is
interest, which compels us to investigate our surroundings and develop our current
knowledge (Fredrickson, 2002). A possible example of interest in cardiac patients
which could be beneficial would be a patient reading all provided leaflets about
their illness, perhaps even finding more information on the internet, and then
385 closely following the advice for dietary change and increased exercise. Negative
emotions narrow a person's thought-action repertoire (e.g., phobia: a person may
feel very anxious in a given situation or when exposed to an anxiety-inducing
stimulus, and can barely think beyond escaping the situation or stimulus), whereas
positive emotions broaden this array of possible behaviours. Another important
390 aspect of this theory is that personal resources acquired in this way are thought to
have strong durability, and these resources outlast the positive states that induced
them (Fredrickson, 2002). They can subsequently be drawn upon in later
experiences, and through this method, the individual can transform themselves
(i.e., becoming more knowledgeable, sociable, etc.). This then leads to an *upward*
395 *spiral* of positive affect (see Figure 1). An important hypothesis of this theory is
that positive emotions 'undo' the effects of negative emotions (Fredrickson, 2002) –
which may be significant in terms of negative emotions and increased cardiovas-
cular risk.

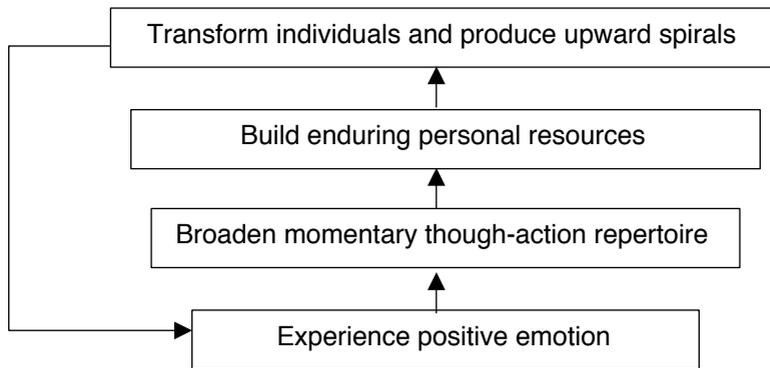


Figure 1. The Broaden-and-Build Theory of positive emotion (Fredrickson, 1998, 2001, 2002).

Associations with depression and CHD

Just as depression has been associated with negative health outcomes, positive emotions are associated with favourable health outcomes in several disease types (Keyes, 2004; Maruta, Colligan, Malinchoc, & Offord, 2000; Ostir, Markides, Black, & Goodwin, 2000; Ostir et al., 2001a; Ostir et al., 2002; Ostir, Ottenbacher, & Markides, 2004). The prevalence of cardiovascular disease was lowest among those who were mentally healthy (defined as flourishing), but highest among those who were depressed and languishing (devoid of well-being but not depressed), in a study of over 3000 adults (Keyes, 2004). This suggests a protective role for positive emotion in medical patients.

This protective role of positive emotion has been demonstrated in aetiological studies and CHD samples (Kubzansky, Sparrow, Vokonas, & Kawachi, 2001; Ostir, Peek, Markides, & Goodwin, 2001b; Scheier et al., 1989, 1999). A 10% reduced risk of incidence of MI per unit increase of positive emotion scores has been demonstrated in older adults, even when controlling for negative affect (Ostir et al., 2001b). Although this study can be criticised for using a depression questionnaire to assess positive affect, previous work has demonstrated that the scale used (Center for Epidemiological Studies – Depression scale: CES-D) subdivides into positive and negative emotion items (e.g., Miller, Markides, & Black, 1997; Sheehan, Fifield, Reisine, & Tennen, 1995). The items of the CES-D can be seen in Table 2, and four positive emotion items that load together are: ‘I felt that I was just as good as other people’, ‘I felt hopeful about the future’, ‘I was happy’, and ‘I enjoyed life’. A 10-year prospective study of optimism and CHD incidence in 1306 men showed that those with high levels of optimism had a 55% reduced risk of cardiovascular morbidity or mortality in adjusted analysis when compared to those with high pessimism levels (Kubzansky et al., 2001). Also of significance was the reported dose–response relationship between optimism and outcomes, which has parallels with similar (but opposing) results with depression outlined previously (Lett et al., 2004; Rugulies, 2002).

Positive and depressive emotion ratings were assessed in 866 cardiac catheterisation patients as predictors of survival over a mean follow-up of 11.4 years (Brummett et al., 2005). Positive and depressive scores were associated with increased and

decreased survival, respectively. Interestingly, however, when positive and depressive scores were analysed together, positive emotions were no longer statistically significant (Table 1). This suggests that although positive emotions are important, they may have lesser significance than depressive symptoms in CHD prognosis during the acute phase. However, although positive emotions may not have an immediate impact, over time it is possible that they could reduce the impact of negative affect. This may be important in cases where increased chronic negative affectivity is present, rather than episodic bouts of negative affect. Further prospective studies are needed to address this possibility, and determine the relative contribution of positive and negative affect to outcomes.

Interventions

Interestingly from a cardiovascular perspective, positive emotions are thought to negate the lingering effects of negative emotions. This is thought to work through the broadening mechanism – an individual's repertoire cannot narrow and broaden at the same time (Fredrickson, 2002). Positive interventions have been shown not only increase happiness, but also to decrease depression (Brown & Ryan, 2003; Fava, Rafanelli, Cazzaro, Conti, & Grandi 1998; Fava & Ruini, 2003; Seligman, Steen, Park, & Peterson, 2005). Positive psychology has already proved valuable in formulating strategies in managing affective disorder in cancer patients (Brown & Ryan, 2003). The efficacy of several brief interventions has been demonstrated previously (Seligman et al., 2005). One intervention that was shown to increase happiness and decrease depressive symptoms for six months involved participants writing down three things that went well each day (and their causes) for one week (reported effect size = 0.5 at six months) (Seligman et al., 2005). Although these interventions have shown efficacy for reducing depression, they have not been tested in the CHD population. The measurement of positive emotion in CHD patients could provide useful models for patient management by helping to define the characteristics of those at lowest risk. Future studies could use both validated positive and negative measures of well-being to capture the spectrum of psychological adaptation. It may be that positive emotions are useful for treating depressive symptoms, but also protective of subsequent cardiovascular morbidity and mortality. If this is the case, then the introduction of positive psychology interventions may provide a promising alternative to an outright focus on negative emotions such as depression.

Vital exhaustion

Some psychological constructs have been specifically developed in the cardiac population. Vital exhaustion is characterised by feelings of excess fatigue, energy loss, irritability and demoralisation, which appear unrelated to (cardiac) illness severity (Appels, 2004; Appels, Hoppener, & Mulder, 1987; Appels & Mulder, 1989). Several of these symptoms are similar to somatic symptoms of depression, and some authors have therefore stated that vital exhaustion has poor content validity (McGowan et al., 2004). Table 2 contains the 23 questions from the Maastricht Interview for Vital Exhaustion. Comparing these with DSM-IV symptoms of depression, 14 questions for vital exhaustion could be argued to overlap with DSM-

IV symptoms. Furthermore, 19 of these questions could overlap with the ICD-10 classification of depression (which differs from the DSM-IV classification in that it includes symptoms of anergia, hopelessness and irritability). The level of overlap between depression and vital exhaustion requires further clarification. One main difference may be that vital exhaustion does not seem to measure anhedonia (apart perhaps from one item on sex). This may be crucial as anhedonia has specifically been linked to increased cardiovascular risk (Doyle et al., 2006). Furthermore, vital exhaustion appears to have substantial overlap with positive and negative emotion (as characterised by the GMS and assuming that positive/negative emotion is a univariate construct), with 14 vital exhaustion items corresponding to 18 GMS items (Table 2). This then leads to the intriguing question – is vital exhaustion a marker for lack of positive affect (or vice versa)? Studies assessing an association between vital exhaustion and depression have shown that the constructs are highly correlated (Kopp, Falger, Appels, & Szedmak, 1998; McGowan et al., 2004; Wojciechowski, Strik, Falger, Lousberg, & Honig, 2000). However, it is unclear whether research in this area measures overlapping symptoms of depression and vital exhaustion, or whether the unique symptoms of exhaustion are more important. Three main factors have been identified – fatigue, depressive symptoms and irritability (Appels, Kop, & Schouten, 2000). Fatigue may therefore have a significant role (but perhaps different role to depression) in outcomes in those with CHD (McGowan et al., 2004), although some researchers report that somatic symptoms of depression are less relevant for risk prediction than depressed affect (Barefoot et al., 2000; Carney & Freedland, 2003).

Associations with depression and CHD

Several studies have shown that vital exhaustion is a precursor for cardiovascular morbidity and mortality in otherwise healthy individuals (Appels, 2004; Appels et al., 2000; Appels & Mulder, 1988, 1989; Bages, Appels, & Falger, 1999; Falger & Schouten, 1992). Appels and Mulder (1988) showed that vital exhaustion increased the risk of MI by 150% in a study of 3877 civil servants followed for four years. McGowan et al. (2004) investigated the association between depression and vital exhaustion in 305 first MI patients. Depression was highly correlated ($r = 0.61$) with vital exhaustion, but the constructs loaded separately in factor analysis. However, these authors used a depression scale which did not measure somatic symptoms of depression (i.e., the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) – see Table 2). Therefore, it is not surprising that the constructs were not found to load onto one factor. Other researchers failed to provide support for separate constructs of vital exhaustion and depression (Wojciechowski et al., 2000).

A study on the impact of depression and fatigue on outcomes following MI showed that either variable was associated with sudden cardiac death, however when both variables were included in analysis, fatigue moderated the influence of depression (Table 1) (Irvine et al., 1999). Fatigue has also been demonstrated to have the strongest predictive power for incident MI, but the predictive power of depression and irritability was eliminated when controlling for fatigue (Appels et al., 2000). Vital exhaustion may therefore be useful to identify those with fatigue, but without depression, or it may also be an alternative marker of disease severity or comorbidity (McGowan et al., 2004). Vital exhaustion prior to MI has also been

shown to be more important for developing subsequent depression, than depression prior to the acute event (Spijkerman, van den Brink, Jansen, Crijns, & Ormel, 2005). Notably, no studies with CHD samples have shown that vital exhaustion predicts outcomes for longer than five years (Table 1). It is possible that exhaustion may be better considered in terms of a short- to medium-term risk prediction, and that differing mechanisms may be involved in shorter-term (e.g., physiological) and longer-term (e.g., behavioural) outcomes. Longer-term assessments of vital exhaustion and depression in CHD samples are needed.

Interventions

A short behavioural intervention (combined stress-management and health education programme during eight weekly 2.5 h sessions) was evaluated in an effort to alter vital exhaustion and depression in patients with CHD (Sebregts, Falger, Appels, Kester, & Bar, 2005). The intervention failed to modify either vital exhaustion or depression during the nine-month follow-up, possibly due to a significant reduction in post-event major depression seen in the control group (Sebregts et al., 2005). A similar outcome for controls was also seen in the ENRICHD trial. Further research showed that although angina symptoms were related to vital exhaustion in patients scheduled for coronary angiography, a reduction in angina frequency did not lead to a reduction in vital exhaustion scores (Pedersen & Middel, 2001). These results suggest that vital exhaustion may not be amenable to intervention to reduce associated morbidity.

However, some results have suggested that a reduction in vital exhaustion may indeed reduce morbidity in patients with CHD (Appels, 2004; Appels, Bar, Lasker, Flamm, & Kop, 1997). A randomised controlled trial of an intervention designed to reduce vital exhaustion and morbidity (EXIT) was conducted on 710 patients undergoing percutaneous coronary intervention (Appels, 2004; Appels et al., 2005). The treatment aimed to identify and reduce stressors and make rest more efficient through group discussions. Recovery was promoted by discussing length of resting time, by doing relaxation exercises and by stimulating physical exercise. After 18 months, the intervention group had a 55% reduced risk of being exhausted and a 53% reduced risk of being depressed. Anginal complaints were reduced by 29%. However, no difference in recurrent coronary events was seen. A post-hoc analysis (eliminating events that occurred before or during the intervention) showed that the risk of a further coronary event was reduced by 55%, but that patients with a history of CHD did not benefit. These results provide some support for the intervention. However, replication is needed due to the methodology (vitaly exhausted patients were selected, therefore generalisability is questionable) and the overlap between depression and vital exhaustion. It is interesting that the patients in this trial also showed a reduction in depression. The concept of vital exhaustion is not theoretically derived, therefore it may be difficult to pinpoint effective interventions and to differentiate these interventions from similar interventions for depression.

Type D personality

One particular area of research focuses on distress in the cardiac population and how individuals deal with this distress. This area has addressed personality in order to

565 explain the tendencies to experience negative emotions and thus heighten cardio-
vascular risk. Type D (distressed) personality is based on the stable traits of negative
affectivity and social inhibition, which reflect tendencies to experience increased
570 negative distress across situations and to then inhibit the expression of these
emotions in social settings (for fear of rejection or disapproval – see Table 1)
(Denollet, 1998a, 2000, 2005; Denollet & Van Heck, 2001; Pedersen & Denollet,
2003). Previously, it has been questioned whether Type D personality really
constituted anything different to negative emotion states such as depression, whether
575 it can account for the risk associated with these states, or whether it simply congests
a complex field of multiple psychological ‘risk factors’ (Carney, 1998; Lesperance &
Frasure-Smith, 1996). The DS-14 questionnaire items listed in Table 2 demonstrates
that although negative affectivity of course overlaps with depressive symptoms, the
social inhibition aspect of Type D is very different. Four items seem to assess trait
580 depressed mood, and another item assesses irritability (which is assessed by ICD-10).
Two negative affectivity items that differentiate Type D from depression (i.e., are not
assessed by either DSM-IV or ICD-10 criteria for depression) are anxiety/worry.
Thus, not only does Type D assess trait (instead of state) negative mood, it also
incorporates some symptoms of anxiety/worry and the unique aspect of social
inhibition. Type D is stable over a period of at least 18 months, and has been shown
585 to be unaffected by changes in depression or anxiety states (Denollet, 2005; Martens,
Kupper, Pedersen, Aquarius, & Denollet, 2007). The developers also propose that
the Type D disposition is grounded in personality theory, and therefore reject
criticism which questions its construct validity in terms of its comparison to
depressive or anxiety states (Denollet & Van Heck, 2001).

Associations with depression and CHD

590 Type D has been associated with increased depressive symptoms in several studies
(Denollet & Brutsaert, 1998; Denollet, Pederson, Ong, et al., 2006; Denollet et al.,
1996), and has also been shown to be associated with other constructs related to
cardiovascular outcomes, such as vital exhaustion (Pedersen & Denollet, 2003;
Pedersen & Middel, 2001). Assessment of vital exhaustion and Type D personality in
595 cardiac patients showed that those with a Type D disposition were likely to have a
high vital exhaustion score (Pedersen & Middel, 2001). These associations are
perhaps unsurprising as some theorists postulate that a general predisposition to
negative affectivity makes individuals more prone to episodic phases of strong
negative emotions, such as depression (Suls & Bunde, 2005).

600 However, the growing importance of Type D personality in the cardiovascular
sphere is highlighted by research which shows it to be an independent risk factor for
cardiovascular morbidity and mortality (Table 1) (Denollet, 1998b; Denollet &
Brutsaert, 1998; Denollet, Sys, & Brutsaert, 1995; Denollet et al., 1996; Denollet,
Vaes, & Brutsaert, 2000; Pedersen & Middel, 2001). For example, Type D was an
605 independent predictor of cardiac morbidity and mortality at five years follow-up of
319 patients with established CHD, even when controlling for standard cardiovas-
cular risk factors (Denollet et al., 2000). A review of ten articles investigating the
Type D construct in cardiac patients showed that those with Type D personality were
at increased risk for fatal and non-fatal cardiovascular events, with odds-ratios
610 (ORs) ranging from 4.1 to 8.9 (Pedersen & Denollet, 2003). These large effect sizes

emphasise the importance of research in this area, and the authors conclude that how individuals cope with negative emotions (inhibition) may be an important mechanism of action that differentiates Type D from studies of negative emotions.

Importantly, several studies have shown that Type D personality contributes to cardiovascular morbidity and mortality over and above the risk associated with other constructs such as depression and anxiety (Denollet & Brutsaert, 1998; Denollet, Pederson, Ong, et al., 2006; Denollet et al., 1996; Pedersen & Denollet, 2003). In studies which measured both depression and Type D, results showed that Type D remained the strongest psychological predictor of outcomes (Denollet & Brutsaert, 1998; Denollet, Pederson, Ong, et al., 2006; Denollet et al., 1996). Depression even became non-significant in some studies (Table 1) (Denollet & Brutsaert, 1998; Denollet, Pederson, Ong, et al., 2006). Social inhibition may in fact be the crucial aspect of risk prediction. Denollet, Pederson, Ong, et al. (2006) analysed major cardiac endpoints (death, MI, coronary artery bypass graft (CABG), or PCI) at nine months in a sample of 875 percutaneous coronary intervention patients. Patients completed the HADS and the DS-14, six months post-PCI. Analysis of the major cardiac events showed that the prevalence of such events was significantly higher in those with high negative affectivity and high inhibition (15%) compared with the group with high negative affectivity and low social inhibition (10%). Furthermore, the moderating effect of inhibition was not explained by either depression or anxiety. From these results, it is clear that Type D is not just another measure of general distress or depression. As Type D is a disposition rather than a state, it may potentially have both short- and long-term effects on risk, over and above that of negative emotional states (which would theoretically benefit from intervention). Aetiological studies are needed to assess whether Type D confers risk for the development of CHD in healthy individuals.

Interventions?

It is questionable whether having a negative personality type lends itself to interventions to reduce negative emotions. Relatively short behavioural interventions designed for mood change may have little efficacy against a stable personality disposition, although it is possible that Type D patients could benefit (but not to the extent that non-Type D patients would benefit) (Pedersen & Denollet, 2003). It may be that Type D personality best accounts for the associated psychological risk factors outlined above. This construct may also explain the failure of some of the interventions outlined previously. Unless intervention studies attempt to control for this personality disposition, the efficacy of interventions may be limited.

However, as social inhibition may be the crucial determinant of increased risk in those displaying the Type D disposition, this may prove a fruitful avenue for intervention. As outlined previously, an increase in positive affect may increase personal resources and social interaction, which then lead to better coping with subsequent life events (Fredrickson & Joiner, 2002). Thus, one could speculate that social inhibition might be gradually eroded by an increase in positive emotions and the upward spiral of positive affect. Of significance here are the previously outlined findings demonstrating that high negative affect was unimportant for cardiovascular risk prediction when social inhibition is low (Denollet, Pederson, Ong, et al., 2006).

Hypothetically, positive emotion interventions deserve consideration for a reduction in social inhibition.

Future directions

660 The present paper provides an overview of constructs related to increased
cardiovascular risk, and assesses their potential for intervention. Based on the
accumulating evidence, it is questionable whether depressive cognitions are as
important as previously considered, as they do not seem to be associated with clinical
outcomes and intervention did not demonstrate risk reduction. Intriguingly, there
665 may be some potential for designing interventions based on distorted illness
cognitions. However, the interaction between illness and depressive cognitions needs
to be formally assessed, and illness cognitions have yet to be established as consistent
predictors of clinical outcomes. Somatic symptoms of depression and fatigue may be
more important aspects for associated risk, however, interventions have also failed to
reduce morbidity. An important confounder for consideration is the Type D
670 personality disposition. Type D may influence research findings via two mechanisms
– a general disposition to negative affectivity, and the modulating aspect of social
inhibition. Measurement of positive affect could provide clues on how to intervene in
negative emotions in this population, and studies from other populations provide
some hope for positive emotion interventions. Thus, despite accumulating evidence
675 over almost 30 years, there remain significant challenges in this field.

The first challenge is one of significant overlap between constructs. For example,
it can be argued that depressed mood is assessed by each of the constructs outlined in
the paper (Table 2). This overlap raises several possibilities. First, one construct (e.g.,
depression) or symptom (e.g., anhedonia) is most pertinent for increased risk, and
680 the positive associations found in other research occurs as a result of high correlation
between this pertinent construct/symptom and the other perhaps less important
aspects. Previously, researchers have stated that depression is a heterogeneous
disorder with several differing symptoms, thus assumptions of the homogeneity of
depression in the cardiac population needs to be considered as one explanation for
685 the failure of interventions (Davidson, Rieckmann, & Rapp, 2005; Frasure-Smith &
Lesperance, 2003; van Melle et al., 2007). A first step in assessing this is discovering
which symptom or cluster of symptoms is most relevant for cardiovascular risk.
Therefore, large studies powered to assess all of these relevant variables are required.
We thus echo the call of other researchers who recommend that researchers re-
690 analyse their datasets and perhaps even pool datasets to allow for more powerful
latent variable analysis (Davidson et al., 2005; Suls & Bunde, 2005), in order to
address the increased risk of individual symptoms. Once the most pertinent
symptom/construct is identified, then interventions could be targeted more
efficiently. Failure to identify the most relevant symptoms or constellation of
695 symptoms that increase (or decrease) cardiovascular risk may reduce the efficacy of
future interventions.

Another alternative is that depression, or other constructs, assessed post-event
are actually a proxy measurement of some (perhaps undefined) psychosocial
construct that does contribute to poorer clinical outcomes. For example, all these
700 constructs may tap a common underlying latent variable, such as a disposition
towards negative affectivity, and this latent variable may be the factor that increases

705 risk (Suls & Bunde, 2005). To date, researchers have assumed that negative emotions are independent, and that the effects of these emotions are also independent (Suls & Bunde, 2005). This hypothesis could explain the heterogeneity of findings of various negative emotion constructs and increased risk. The present review clearly outlines that there is substantial measurement and construct overlap. Thus, more sophisticated analysis of such studies is required.

710 A second challenge concerns the potential for intervention. To date, studies on depression have been largely atheoretical, providing no real insight into how interventions may be derived from their findings. To our knowledge, only one study has demonstrated that several theoretical vulnerabilities to depression are present in the depressed cardiac population. Rieckmann et al. (2006) assessed 314 ACS patients with the BDI within one week of hospital admission and reported that both mildly and moderately/severely depressed patients showed higher levels of cognitive, interpersonal and behavioural vulnerabilities. These vulnerabilities independently contributed to depressive symptoms (although the presence of more than one vulnerability increased the odds of being depressed). What may be interesting in the context of the current review is whether depressive symptoms were qualitatively different across those with different vulnerabilities, i.e., are specific vulnerabilities more important for anergia or anhedonia? Furthermore, it would be important to follow patients demonstrating these vulnerabilities to determine which vulnerability-caused symptoms (or symptom clusters) are most relevant for cardiovascular risk. This could then allow targeting of potentially risk-reducing interventions. When the most relevant symptoms, syndromes or interactions are pinpointed, their mechanisms of action need to be identified. Additionally, it is possible that some or all aspects (cognitions, emotions, fatigue and personality) could combine synergistically to produce higher risk than any single aspect. If this is the case, then interventions addressing individual constructs are doomed to failure until the overall, interacting mechanisms are understood.

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730 While the SADHART, ENRICHD and MIND-IT trials showed that a subset of individuals did not respond to treatment, post hoc analysis has shown that non-responders and those with incident depression were at greater risk of subsequent events (Carney et al., 2004; de Jonge, Honig, et al., 2007; Glassman et al., 2002). This is suggestive of a non-responsive subtype of depression which confers increased cardiovascular risk, but also that the 'depression' is actually triggered by the event itself. This raises questions on the extent that depression existed prior to the event, whether 'depression' is simply a marker of disease severity (for those with incident depression), and to what extent pre-existing depression and incident depression confer increased risk. Previous work has demonstrated that 'depression' in CHD patients is mostly made up of somatic symptoms (Luutonen, Holm, Salminen, Risla, & Salokangas, 2002; Sorensen et al., 2005). There is also the suggestion that somatic symptoms may be cardiotoxic, whereas cognitive-affective symptoms are not (de Jonge, van den Brink, Spijkerman, & Ormel, 2006). Incident depression post-ACS has been shown to be related to more severe CAD (Goodman, Shimbo, Haas, Davidson, & Rieckmann, in press; Spijkerman, de Jonge, et al., 2005), and is more cardiotoxic than other depression subtypes (de Jonge et al., 2006; Grace, Abbey, et al., 2005). This suggests that somatic symptoms such as fatigue may be markers of underlying disease severity or prodromal states. If this is the case, then this could explain the findings regarding vital exhaustion and other somatic symptoms of

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750 depression. However, it would not explain the studies which showed an association
between cognitive/affective symptoms and outcomes (Barefoot et al., 2000; Carney &
755 Freedland, 2003; Lesperance, Frasure-Smith, & Talajic, 1996), or the association
between positive emotion and outcomes. Furthermore, aetiological studies have
established an association between depressive symptoms and incident CHD in
760 initially healthy individuals, as demonstrated in systematic reviews (Rugulies, 2002;
Wulsin & Singal, 2003). The findings of Rieckmann et al. (2006) outlined above,
which show that depressed ACS patients exhibit the theoretical vulnerabilities to
depression, also indicate that depression is present prior to the cardiac event in a
765 substantial number of patients. As these vulnerabilities would have been present
prior to the ACS event, and they are theoretically unrelated to CHD, one could
therefore argue that these patients are depressed independently of CHD severity.
Future studies need to differentiate pre-event depression from incident depression to
determine the relative contribution of different subtypes to increased risk. The
potential for intervention will hinge on the identification of the cardiotoxic subtypes
or symptoms of depression.

Other researchers have highlighted issues of sample size, lack of efficacy of
interventions for depression, under-use of appropriate disease severity indices,
inflation of effect sizes of depression (and consequent overestimation of potential for
770 intervention), heterogeneity of depressive symptoms, common genetic pathways for
depression and CHD, physiological mechanisms and lack of studies pinpointing
precise mechanisms (Carney & Freedland, 2007; Davidson et al., 2005; Frasure-
Smith & Lesperance, 2003; Lane, Carroll, Ring, Beevers, & Lip, 2001; McCaffery et
775 al., 2006; Nicholson et al., 2006; Skala, Freedland, & Carney, 2006; van Melle et al.,
2007). The appropriate assessment of CHD severity indices and the subsequent
inflation of effect sizes for depression require further exploration. Nicholson et al.
(2006) concluded that depression has yet to be established as an independent risk
factor for CHD because of non-adjustment for conventional risk factors and severity
780 of coronary disease. In their systematic review of 34 prognostic studies, only eight
reported left ventricular function, and the inclusion of this variable attenuated the
relative risk associated with depression by 48% (from 2.18 to 1.53). Lane and
colleagues have also criticised prognostic studies for imperfect measurement of CHD
risk factors, which may lead to under-adjustment in multivariate prognostic models,
785 thereby inflating the effects of depression (Lane, Carroll, & Lip, 2003; Lane et al.,
2004). Indeed, several studies have shown a relationship between depression and
measures of disease severity, including left ventricular function. Of interest here is
that Type D personality does not demonstrate a relationship with somatic health (de
Jonge, Denollet, et al., 2007). Their study assessed Type D status in a sample from
MIND-IT, and showed no relationship with Type D status at one year and baseline
790 somatic health (including left ventricular ejection fraction, comorbidity index, prior
MI or coronary interventions during hospitalisation). Depression was associated
with comorbidity and left ventricular ejection fraction, and these results further
highlight the potential importance of the Type D construct. Studies need to control
for appropriate disease severity indices, and determine whether the depressive
795 episode existed pre-event, to ascertain the association between depression and
outcomes.

The present review aimed to summarise the current impasse in moving from
prediction to intervention in the depression and CHD outcomes relationship. A

number of recent, large, randomised trials have failed to demonstrate the cardiovascular benefit from psychological or pharmacological interventions in cardiac patients. There is certainly the danger that the wider medical community decide that this topic is concluded and that research funds will be increasingly difficult to commandeer in the future. In this regard, we see our review as somewhat a 'call to arms' for the Health Psychology community. We need to demonstrate that a more sophisticated understanding of these issues can lead to more precise pinpointing of the 'cardiotoxic' aspects of psychological status and interventions that are more refined to address these. The second aim of this review was to provide a single inclusive report of the psychological concepts relevant to depression and cardiovascular outcomes. There is much construct overlap in the negative emotions literature; positive emotions have been relatively neglected in cardiac populations; there is the challenge of designing interventions without knowledge of precise mechanisms; and there is atheoretical assessment of depression and related constructs in the field. The challenge is for psychological research to provide clarity on these issues – overlapping and priority constructs; the faithful translation of constructs into interventions; and controlling where necessary for psychological constructs that are relatively immutable risk factors. To do this, researchers must seek to integrate across concepts, methods and studies.

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