

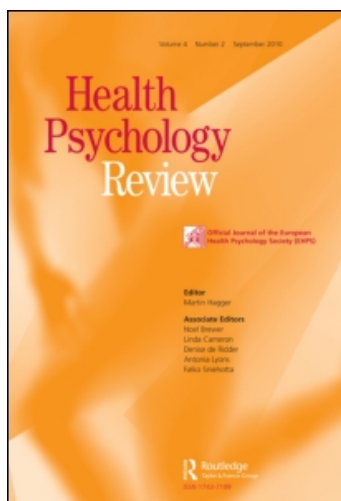
This article was downloaded by: [Glass, David C.]

On: 15 February 2011

Access details: Access Details: [subscription number 933519477]

Publisher Routledge

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Health Psychology Review

Publication details, including instructions for authors and subscription information:

<http://www.informaworld.com/smpp/title~content=t741771149>

Bipolar disorder, Type A behaviour and coronary disease

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First published on: 15 February 2011

To cite this Article Glass, David C. and Contrada, Richard J.(2011) 'Bipolar disorder, Type A behaviour and coronary disease', Health Psychology Review,, First published on: 15 February 2011 (iFirst)

To link to this Article: DOI: 10.1080/17437199.2010.531568

URL: <http://dx.doi.org/10.1080/17437199.2010.531568>

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Bipolar disorder, Type A behaviour and coronary disease

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(Received 10 June 2010; final version received 11 October 2010)

This paper presents a model for integrating two psychological constructs – bipolar disorder and the Type A behaviour pattern – each of which has been associated with enhanced risk of coronary heart disease (CHD). It describes similarities between manic/hypomanic behaviours associated with bipolarity and the behaviours observed in Type A individuals. The proposed model highlights the importance of alternating patterns of coping with challenging and stressful life events. Thus, initial coping efforts are manifested as behavioural *hyper*-reactivity (i.e., Type A behaviours and mania/hypomania), but this gives way to *hypo*-reactivity (including helplessness and depression) after repeated failure to assert control and/or attain relevant goals. This alternation of what originally were regarded as Type A coping patterns resembles the affective and behavioural transitions often seen in bipolar patients. Future research on psychological, epidemiological and pathophysiological issues concerning CHD should document areas of commonality and independence between bipolarity and Type A behaviour. Such studies would benefit from consideration of a model that identifies psychosocial dimensions common to Type A, mania/hypomania and depression.

Keywords: bipolar disorder; Type A behaviour; depression; coronary heart disease

Introduction

This paper is concerned with the systematic use of theory in research designed to identify and understand emotional factors that may promote coronary heart disease (CHD). It describes a framework for integrating two psychological constructs – bipolar disorder (BP) and the Type A behaviour pattern (TABP). Although the subject of two largely separate literatures, the constructs share content reflective of hyper-reactivity (i.e., manic-like symptoms), both include components that involve responsiveness to challenging and stressful events, and each has been linked to cardiovascular problems. Our efforts to suggest an integrative framework for these constructs are based, in part, on a model that uses the dual ideas of hyper-reactivity and hypo-reactivity (i.e., helplessness) to clarify the psychological dimensions of TABP that may account for its association with CHD (Glass, 1977). Helplessness has been identified as a possible precursor of depression (e.g., Peterson, Maier, & Seligman, 1993), and recent research on depression (both minor and major) provides

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strong evidence of its relationship with the onset and progression of CHD (Goldston & Baillie, 2007). Though less extensive, studies of BP and CHD show a similar association. This work implicates mania, as well as depression, as possible CHD risk factors.

The hyper-/hypo-reactivity model, which integrates bipolarity and TABP, proposes that challenging and stressful events will elicit initially vigorous efforts to cope with such events. However, subsequent difficulty in coping will produce depression-like affect and behaviour, including helplessness, and decrements in motivation and efforts to respond to environmental demands. This alternation of Type A modes of coping appears to resemble the shifts between manic and depressive mood states characteristic of many BP patients. Physiological concomitants of such moods and their fluctuations over an extended time period may promote coronary atherosclerosis and CHD events. The precise mechanisms remain unspecified, but they appear to involve autonomic, neuroendocrine and associated physiological changes as discussed later in this paper (e.g., Bekkouche, Holmes, Wittaker, & Krantz, 2011).

The paper begins with a discussion of bipolarity and then moves on to a section that evaluates the evidence for its association with CHD. On balance, the data support a significant positive association. The third section is devoted to describing TABP and its past and current status as a predictor of CHD. A fourth section closely compares the overlap of TABP and BP, ending with a recent study that reports an empirical effort to relate the two constructs. The fifth section presents an integrative model of TABP and BP, in which coping with challenging and stressful life events is hypothesised to trigger initial hyper-reactivity and subsequent hypo-reactivity that may lead to increased risk of CHD. The sixth section discusses ways in which the integrative model provides a unitary framework to guide psychological, epidemiological and pathophysiological research on CHD.

Bipolar disorder

Two principal forms of bipolarity are part of the diagnostic nosology presented in the text revision of the fourth edition of the American Psychiatric Association's (APA) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; APA, 2000). BP I is characterised by the occurrence of one or more manic episodes. There is no requirement for the patient to have experienced depression, although many BP I patients have at least one depressive episode in their history. BP II is characterised by one or more depressive episodes accompanied by at least one hypomanic episode. The distinction and differential diagnosis is explicated in DSM-IV-TR. For purposes of this paper, it is important to emphasise the 'phenotypic' or descriptive characteristics accompanying the two forms of BP because they are shared with TABP. Both BP I and BP II include a distinct period of either elevated or irritable mood, accompanied by three or more additional symptoms such as inflated self-esteem or grandiosity, decreased need for sleep, pressured speech, flight of ideas, distractibility, increased involvement in goal-directed activities, psychomotor agitation and increased involvement in pleasurable activities with high potential for negative consequences (e.g., uncontrolled buying sprees).

Regarding the distinctions between BP I and II, delusions or hallucinations cannot be present in the case of BP II, and this constitutes a major criterion for

differentiating it from BP I. Note, also, that in contrast to a manic episode, a hypomanic episode (usually associated with BP II) cannot be 'severe enough to cause marked impairment of social or occupational functioning' (APA, 2000, p. 365). For some individuals, elevated mood may accompany increased efficiency and accomplishment, although this is not an invariant consequence. While BP I patients need not experience a depressive episode, and some do not (Kessler, Rubinow, Holmes, Abelson, & Zhao, 1997), research has indicated that both types of bipolar patients show a predominance of minor depressive symptoms over major depressive and manic symptoms (e.g., Judd et al., 2003). Depression and mania often occur at levels below that necessary for clinical diagnosis (subsyndromal). Therefore, the concept of a broad bipolar spectrum involving subsyndromal *and* syndromal mood manifestations might be a more appropriate conceptualisation (e.g., Akiskal et al., 2000). Indeed, a dimensional perspective of bipolarity has been endorsed by a leading textbook on manic-depressive illness (Goodwin & Jamison, 2007). This idea of a spectrum of BP illness is compatible with the view that emphasises an overlap of hypomanic symptoms and Type A behaviours. Later in this paper, we examine this overlap and its theoretical implications for treating the two constructs within a unitary framework.

Bipolar disorder and coronary heart disease

This section discusses empirical support for an association between BP and CHD. A PUBMED database search of 'bipolar disorder' and 'cardiovascular disease' over the past 30 years generated more than a dozen clinical and epidemiological studies of CHD *mortality*. Most reported that BP was associated with increased risk of mortality after adjusting for age and/or gender (e.g., Angst, Stassen, Clayton, & Angst, 2002; Fiedorowicz et al., 2009; Garcia-Portilla et al., 2009; Laursen, Munk-Olsen, Nordentoft, & Martensen, 2007; Osby, Brandt, Carreira, Ekblom, & Sparen, 2001; Sharma & Markar, 1994; Weeke & Vaeth, 1986). Two of the studies suggest greater risk of cardiovascular mortality for BP patients compared to those with other mental illnesses, such as unipolar depression (Osby et al., 2001; Weeke & Vaeth, 1986). However, a few studies did not report excess mortality explicitly in bipolar patients (e.g., Black, Winokur, & Nasrallah, 1987; Murphy, Monson, Olivier, Sobol, & Leighton, 1986; Tsuang, Woolson, & Fleming, 1980), or the association was reduced by cardiovascular comorbidity factors which themselves are associated with both BP and CHD mortality (Kilbourne et al., 2009).

There are only two studies that compare the CHD mortality rates of BP I and BP II patients, and the data from both derive from unusually long follow-up periods (Angst et al., 2002; Fiedorowicz et al., 2009). Angst and his colleagues examined overall and cause-specific Standardised Mortality Rates (SMR; observed deaths/expected deaths) in a sample of 186 unipolar and 220 bipolar patients (150 Is and 70 IIs) hospitalised between 1959 and 1963 in Switzerland. Calculations were made by gender, 5-year age classes, and 5-year calendar periods. The results showed that relative to the general population, patients with unipolar and BPs had a significantly higher risk of cardiovascular death. The mortality rate for BP Is was significantly higher than for BP IIs, but this was attributable mainly to cerebrovascular deaths. The bipolar subtype difference in cardiovascular mortality reached statistical significance but was a much weaker effect. Fiedorowicz et al. (2009) report results

of a prospective study also aimed at comparing cardiovascular mortality by bipolar subtype. No effort was made to estimate general population norms for CHD deaths during the study period. Participants were followed for up to 25 years with a median of 20 years. There were 33 deaths during the follow-up period. An initial finding of significantly greater risk of mortality in patients with BP I compared to BP II was no longer observed after an index based on proportion of weeks with clinically significant manic/hypomanic symptoms was introduced into the regression equation. Indeed, the index was itself predictive of CHD mortality.

The foregoing discussion has concentrated on cardiovascular mortality. However, the database search also identified studies reporting higher prevalence of cardiovascular risk factors among BP patients compared to the general population (e.g., Baune, Adrian, Arolt, & Berger, 2006; Goldstein, Fagiolini, Houck, & Kupfer, 2009). The risk factors identified in this body of research included (1) dyslipidemia (e.g., van Winkel et al., 2008), (2) hypertension (e.g., Goldstein et al., 2009; Yates & Wallace, 1987), (3) diabetes mellitus (e.g., Fagiolini, Frank, Scott, Turkin, & Kupfer, 2005), (4) obesity (e.g., Fiedorowicz, Palagummi, Forman-Hoffman, Miller, & Haynes, 2008), (5) smoking (e.g., Kilbourne et al., 2009; Yates & Wallace, 1987) and (6) physical inactivity (e.g., Kilbourne et al., 2007).

To sum up, individuals with BP have more CHD deaths than the general population after adjusting for age and gender. This association became non-significant in one study after controlling for cardiovascular comorbidities. Studies comparing CHD mortality in bipolar subtypes generated equivocal results. Research on CHD risk factors and bipolarity presented a more compelling picture in which BP patients were found to experience a substantial chronic medical burden. One explanation for this observation is that bipolarity may result in a plethora of unhealthy behaviours, such as heavy cigarette smoking, alcohol and/or drug abuse, inadequate physical exercise and poor diets. An equally unhealthy lifestyle entails activities aimed at coping with challenging and stressful events; for example, trying to attain occupational and social goals by means of a pressured and aggressive style. Such behaviours are not unlike Type A behaviours discussed below.

Type A behaviour and coronary heart disease

The TABP consists of a set of overt behaviours that are elicited from susceptible individuals by challenging and stressful environments (Friedman & Rosenman, 1974; Matthews, 1982). The pattern is characterised by competitive drive, impatience and time urgency, hostility and anger. In the Western Collaborative Group Study, for example, men with TABP were found to experience twice the incidence of coronary disease after controlling for traditional risk factors such as cholesterol and cigarette smoking (Rosenman et al., 1975). The idea that global TABP predicted CHD did not remain unchallenged. Reviews of the literature in the 1980's observed that while TABP was reliably related to CHD, the average strength of the correlation had decreased over the past decade, especially in those who already had CHD (Booth-Kewley & Friedman, 1987). A subsequent meta-analysis concluded that the association was, indeed, largely confined to population-based studies with participants free of CHD at the beginning of the study (Matthews, 1988). It also was concluded that the predictive power of the structured interview (SI) technique for assessing TABP was superior to various self-report instruments.

Later commentators advocated abandoning Type A research altogether (Conduit 1992), and a 2002 review of psychosocial factors and CHD made only minimal reference to Type A behaviours (Krantz & McCeney, 2002). The next research trend focussed on associations between CHD and the specific Type A components of hostility and anger. Both of these behaviours are evident in BPs, as elaborated below. The focus on the hostility and anger components produced results indicating that they are the aspects of TABP most predictive of CHD, including atherosclerosis and CHD deaths (Miller, Smith, Turner, Guijaro, & Hallet, 1996). However, an almost exclusive concern with hostility/anger may have been a premature shift in research strategy. Global Type A behaviour has shown, after all, a reliable relationship with CHD in initially healthy people (Matthews, 1988). Moreover, large-scale epidemiologic studies of Type A and CHD usually have not measured stressful life events, certainly not *uncontrollable* stressful events. As we discuss below in greater detail, hyper-reactivity is an initial response to such events, especially in Type A individuals who display increased efforts to assert and maintain control in the face of its possible loss (Glass, 1977). This hyper-reactivity bears a strong resemblance to the manic/hypomanic behaviours associated with bipolar illness. We turn now to an examination of these similarities.

Bipolar disorder and Type A behaviour

Most bipolar individuals show evidence of depression, but the accompanying manic/hypomanic episodes also are core elements in defining the disorder. These episodes can mimic the initial responses of Type A individuals to challenging and stressful life events. Like Type A's, manic and hypomanic patients can be irritable, mistrustful and angry. The elevated and expansive mood that often characterises bipolarity is not usually included as a feature of TABP. However, Type A individuals frequently exhibit enthusiasm for goal-striving activities that are similar to the expansive enthusiasm for occupational and social interactions that typify hypomanic patients (Johnson, 2005a; Rosenman, 1978).

What about other manic/hypomanic features and their presence in TABP? Impatience and time urgent behaviours of Type A individuals have been found to differentiate bipolar II patients from unipolar depressed patients (Oedegaard, Neckelmann, & Fasmer, 2006). The pressured speech pattern of the manic/hypomanic individual may be different from the energetic speech stylistics of Type A's with their propensity to interrupt others. On the other hand, both have a loud, rapid and vigorous voice pattern (Glass, Ross, Contrada, Isecke, & Rosenman, 1982; Rosenman, 1978). Note that TABP speech stylistics generally is accompanied by a hurried 'motor pace' and 'frequent rhythmic movement of hands/feet' (Rosenman, 1978). Such behaviour can be construed as a form of psychomotor agitation not unlike that often observed in mania/hypomania.

Decreased need for sleep is another diagnostic sign of mania/hypomania, especially when triggered by life events that disrupt customary routines and schedules (e.g., Malkoff-Schwartz et al., 2000). A decrement in *need* for sleep usually is not included in the Type A profile, but it is reasonable to infer such a decrement among Type A's as they pursue their customary drive to achieve more and more in less and less time. Indeed, empirical evidence has shown that individuals who sleep fewer hours per night score high on measures of Type A behaviour (Hicks et al.,

1979; McKelvie, 1992). Even more interesting, perhaps, is an early study of these 'habitual short sleepers' (<6 hours per night). It was observed that they were efficient and energetic, but 'non-worriers' who tend to 'avoid problems by keeping busy and by denial which in some cases approached hypomania' (Hartmann, Baekeland, & Zwilling, 1972). The assertive striving of Type A's appears akin to increased involvement in goal-directed activity that typifies many bipolar patients. It may not reflect the grandiosity of an acute manic, but Type A goal-striving has been linked to a propensity to enhance self-worth by gaining approval of others (Johnson, 2002). This conclusion is not meant to imply that A's have an inflated level of self-esteem. While they appear dominant and self-confident and, in fact, may have greater success in attaining career goals (Matthews, Helmreich, Beane, & Lucker, 1980), their feelings of self-worth are contingent upon achieving excessively high and sometimes unattainable goals (Friedman & Ulmer, 1984; Powell, 1992). These features of a Type A individual resemble the self-esteem of a hypomanic patient insofar as both display uncritical self-confidence in occupational and social interactions.

There are empirical data suggesting that people with a history of manic-like characteristics have elevated expectations for success in domains involving social recognition of their achievements (Johnson, Eisner, & Carver, 2009). Still other research implicates goal-striving as a trigger of manic/hypomanic symptoms. Thus, preparation for and completion of final exams have been associated with an increase in manic, but not depressive symptoms in college students with a bipolar spectrum diagnosis (Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2007). Johnson (e.g., Johnson et al., 2008; Lozano & Johnson, 2001) reports similar results following goal-attainment life events in BP I individuals, where such events range from highest attainment (e.g., making law partner or being accepted into graduate school) to little or no attainment. Initial success in attaining goals leads bipolar individuals to develop unrealistic levels of self-confidence, which 'fuel excessive behavioural involvement and . . . ever-higher goals . . .' (Johnson, 2005b, p. 254).

The comparison of TABP and bipolarity – at least its manic/hypomanic components – suggests a distinct overlap, but not isomorphism. Thus, the euphoria that may characterise a manic episode is not a defining feature of Type A behaviour. Indeed, mania may involve certain hedonistic pursuits that contrast with the intense work involvement shown by most Type A individuals. And despite overall similarity, there may be subtle differences between Type A and manic speech stylistics, with the former perhaps being more interruptive and staccato, and the latter more resistant to interruption and overproductive. Nonetheless, the areas of overlap are extensive enough to warrant systematic examination. The important point to bear in mind here is that both TABP and mania/hypomania reflect a general tendency towards behavioural hyper-reactivity. If we acknowledge the dimensional perspective of bipolarity discussed earlier, it is not implausible to suggest that some individuals have a history of BP with hypomania displays sufficiently mild so as to obscure their clinical detection (Barrick, 1999). At that level, we may be dealing with what is tantamount to TABP.

While a definitive test of this hypothesis must await future research, a study by Oedegaard et al. (2006) is highly suggestive. A sample of 42 unipolar and 23 bipolar II patients completed the Jenkins Activity Survey (JAS, Form C), a widely used self-report questionnaire for assessing TABP (Jenkins, Zyzanski, & Rosenman, 1976).

Bipolar IIs had significantly higher global JAS scores than unipolar patients and this difference was due mainly to a 'divergence in factor S' (speed and impatience), one of the three component scores of the JAS. There are obvious limitations of this study, including the very small number of cases, the cross-sectional nature of the design, and, perhaps, the fact that TABP was measured by JAS rather than SI. Nonetheless, the results are consistent with the observation of a BP/TABP overlap. This overlap focusses on the manic side of bipolarity, but there is also evidence that Type A scores show modest positive correlations with traditional self-report measures of depression (e.g., Barefoot, Haney, Simpson, Blumenthal, & Williams, 1990; Byrne & Rosenman, 1986; Francis, 1982). The next section develops this theme in more detail.

Bipolar depression and Type A behaviour

We have just noted that bipolar individuals exhibit a depressive side, and under certain conditions Type A's also show affect and behaviour consistent with depression. Thus, if both types of individuals experience repeated failure, initial self-confidence and striving decline, and depressive symptomatology is a likely outcome (Glass, 1977, pp. 163–173; Johnson, 2005b). Notwithstanding the widely recognised genetic contributions to BP (e.g., Goodwin & Jamison, 2007), recent research has documented an important role for stressful life events in the production of depressive symptomatology (e.g., Johnson et al., 2008; Johnson, Winters, & Meyer, 2006). Serious negative life events (e.g., foreclosure on mortgage/loan) occurring several months before symptom presentation predict the onset and recurrence of bipolar and unipolar depression (Johnson, 2005a). However, most bipolar depression studies do not present data on negative events that reflect the participant's perceptions of the uncontrollability of these events.

The Type A literature, by contrast, contains several studies that focus specifically on uncontrollable events. Thus, it has been found that unexpected negative events (a type of cognitive uncontrollability) were significantly associated with distress in Type A individuals, although perceived lack of responsibility for the events did not show a similar association (Suls, Gastorf, & Witenberg, 1979). More unequivocal results come from a study of Type A coronary patients who reported more *losses* in the last six months than healthy controls, while other negative experiences occurred with equal frequency to patients and controls (Glass, 1977, Chap. 10). Still other research has documented the propensity of Type A individuals to display reduced active coping efforts when confronted by repeated failure to control aversive events (Glass, 1977, Chap. 8). This type of effect is considered a form of learned helplessness and has been associated with depression (Peterson et al., 1993). It is noteworthy, therefore, that feelings of vital exhaustion, prevalent among depressed individuals, were found in Type A coronary patients in association with chronic aversive conditions that could be construed as uncontrollable (Appels, 1990; Falger, 1989; Kop, 1999).

Towards a conceptual model of bipolarity and Type A behaviour

The conceptual model presented in this section is based on the idea that perceptions of challenging and stressful events are likely to increase motivation and elicit effortful coping in most people – at least following initial appraisal of the events (Wortman &

Brehm, 1975). Increased motivation promotes what we have termed hyper-reactivity. It encompasses the display of manic/hypomanic symptoms as well as much of Type A behaviour. Indeed, we suggest this latter behaviour may be characterised on a bipolar spectrum as a form of subsyndromal hypomania. Goal-striving life events appear to trigger perceptions of challenge (the potential for reward) that lead to enhanced self-confidence and elevated efforts to obtain the relevant goal, especially in susceptible individuals such as hypomanics and Type A's (Glass, 1977; Johnson, 2005a). Uncontrollable stressful events (those with the potential for harm/loss) initiate a similar process in Type A's, but there is virtually no empirical evidence bearing on their role as precipitants of mania/hypomania.

There is, however, some research suggesting that if an individual experiences enough failure to attain desired goals, perceptions of uncontrollability ensue and associated declines in motivation and active coping are observed (Brunson & Matthews, 1981; Glass, 1977, pp. 163–169). These consequences may potentiate depressive symptoms in bipolar and Type A individuals. This pattern of responding is what we have termed hypo-reactivity and was conceptualised originally within the learned helplessness framework. The cycling of hyper-/hypo-reactivity need not entail abrupt oscillations. Indeed, the bipolar literature considers the essential feature of 'rapid cycling' to be four or more mood episodes during the past year (Goodwin & Jamison, 2007). While the original model of Type A behaviour did not specify number of alternations of reactivity, nor intervening time intervals, it was specific about the precipitant of the change, namely, perceptions of uncontrollability. Recall that the bipolar literature is not as specific about the role of uncontrollability and only concludes that negative events lead to bipolar depression, just as it does in unipolar depression (e.g., Johnson et al., 2008).

Our model for integrating Type A behaviour and BP is outlined in Figure 1. The core elements are based on the 1977 model, but its current form deviates from an exclusive emphasis on uncontrollable stress. We introduced modifications aimed at taking into account work on goal-striving precipitants of manic/hypomanic episodes. Note, also, that the integrative model assumes that the transition from hyper-reactivity to hypo-reactivity is a recurring process. After successful control or goal attainment in a given instance, the behavioural struggle begins anew with the individual attempting to assert control over another event or to attain yet another goal.

Possible neurobiological substrates

The neurobiological substrates of hyper-/hypo-reactivity have not received extensive examination. However, a recent animal study reports some provocative findings involving behavioural analogues of mania and depression that were triggered by standard laboratory stressors (Maeng et al., 2008). These behaviours involved display of active exploration in a situation that normally produces anxiety-like responses and inhibition, and enhanced spontaneous recovery following induction of helplessness by exposure to uncontrollable aversive stimulation. The observed effects were attributed to activity of glucocorticoid receptors (GR) based on comparisons of transgenic mice bred for overexpression of BAG1 – a protein that attenuates GR activity – with BAG1 heterozygous knockout (+/-) mice and wild type (control) mice.

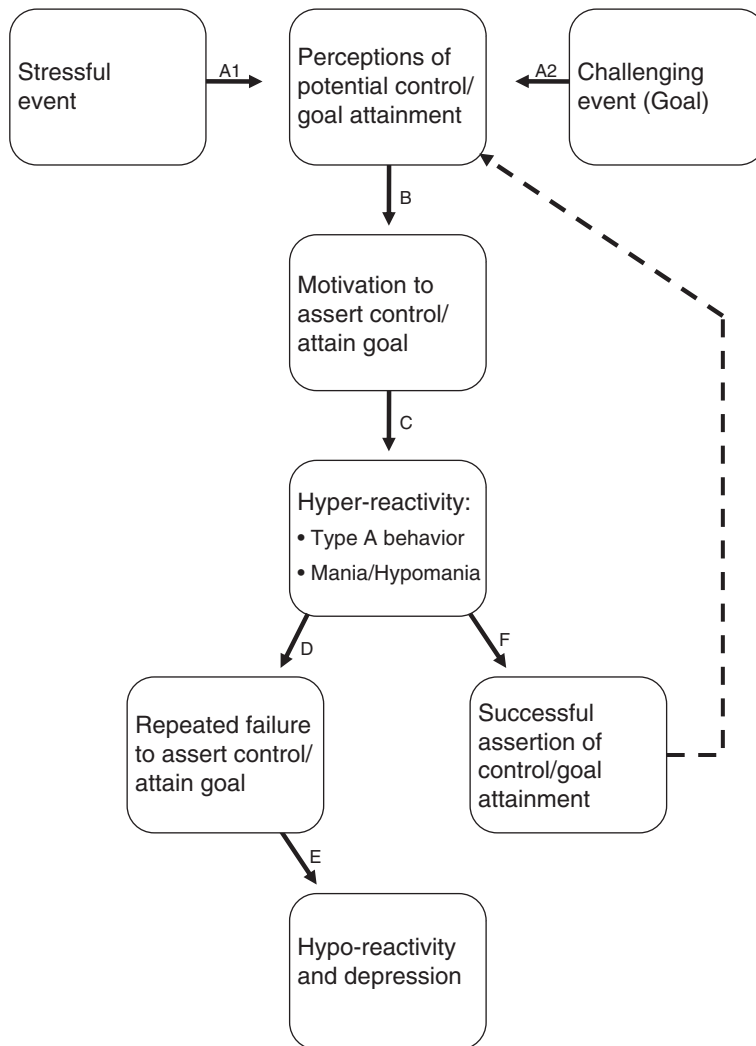


Figure 1. Integrative model of Type A behaviour and bipolar disorder.

Note: Arrows A1 and A2 depict the processes leading to appraisal of an event or goal as potentially controllable and/or attainable. Arrow B indicates that the initial appraisal results in motivation to avoid the stressful event and/or attain the desired goal. Arrow C indicates resultant behavioural hyper-reactivity aimed at asserting control and/or pursuing the goal (i.e., Type A behaviours, manic/hypomanic behaviours). Arrow D reflects an outcome of hyper-reactivity, namely, failure to achieve control and/or attain the goal. Arrow E shows the hypo-reactive behavioural consequences of such failure, including depressive symptoms. Arrow F is the counterpart of Arrow D, insofar as it reflects successful assertion of control and/or goal attainment. The dashed line highlights the recurring nature of the transition from hyper-reactivity to hypo-reactivity following success.

Generalisation of these findings to affective behaviour in humans must await further investigation. Nonetheless, the paradigms of the animal study resemble those used to induce behavioural hyper-reactivity and hypo-reactivity in Type

A individuals. Moreover, there is a sizeable literature on the relationship between stress and activation of the hypothalamic pituitary adrenocortical (HPA) axis including the glucocorticoid, cortisol (Miller, Chen, & Zhou, 2007). It appears that chronic stress produces significant elevations in total daily volume of cortisol. Taken together with the animal research, there is some neurobiological evidence to support the idea of treating both hyper- and hypo-reactivity within a single framework that emphasises the role of challenging events and uncontrollable stressful events in triggering this pattern of behaviour.

Another relevant theoretical analysis suggests that impulsive aggression and lethargic depression in humans may have a common substrate involving low serotonergic levels (Carver, Johnson, & Joorman, 2008). Hostility and aggression are seen in both manic/hypomanic and Type A individuals and, as noted earlier, they appear to enhance coronary risk. Although this neurobiological process differs from the GR activity interpretation of the animal research, Carver et al. do provide support for the idea that hyper-reactive and hypo-reactive states, such as are documented in Type A individuals and bipolar patients, may be conceptualised within a single psychobiological model.

Future directions

The conceptual model presented in this paper has a number of implications for future work on the role of emotional factors in the etiology and pathogenesis of CHD. This section discusses these implications by providing substantive and methodological suggestions for psychological and epidemiological research and mechanism-focussed studies of TABP and bipolarity.

Psychological and epidemiological research

A fundamental premise of the integrative model is that the different emotional factors implicated in CHD should not be studied in isolation. It calls for research aimed at uncovering common and unique attributes that give rise to hyper-reactivity (i.e., Type A behaviour, mania/hypomania) and hypo-reactivity (helplessness, depression). Thus, we might ask about the prevalence and intensity of TABP components in bipolar patients, especially BP IIs, just as we might inquire about the prevalence and intensity of mania/hypomania among Type A individuals. A recent study addressing a related issue found that individual differences in hostile personality were predictive of subsequent elevations in depressive symptoms (Stewart, Fitzgerald, & Kamarck, 2010). In addition to being studied both simultaneously and in sequence, the functional relationships between different emotional factors require examination. The model suggests that episodes of TABP and mania/hypomania are causal antecedents of hypo-reactive, depressive episodes. The precise mechanisms that underlie this transition remain to be determined in future studies. Such research also will permit determination of whether manic/hypomanic symptoms account for increased risk of clinical CHD over and above the depressive symptoms in BPs.

A focus on person-situation interactions is another key premise of the integrative model. Therefore, emotional factors should be studied within a framework that addresses exposure to challenging and stressful events and takes into account

features of those exposures such as controllability and the opportunity for goal attainment. The model also hypothesises that the effects of initial and extended experience with challenging and stressful events may differ in individuals characterised by manic/hypomanic and depressive attributes. Although it may be possible to accomplish tests of this proposition in laboratory analogue studies, as was done in early tests of behavioural responses to uncontrollable stressors in Type A individuals (Glass, 1977), it is likely that future research will require naturalistic settings using both new-generation alternatives to life-events measurement and ambulatory monitoring of relevant physiological processes (Anderson, Wethington, & Kamarck, 2011; Kamarck, Shiffman, & Wethington, 2011). The person–situation perspective has yet to be incorporated adequately in epidemiological research on stress-related emotional factors contributing to CHD risk. The need for well-controlled studies of this sort is palpable.

Research on emotions and CHD should incorporate indices of behavioural manifestations of emotions. Thus, the tendency to rely upon self-report measures in many studies of depressive affect and CHD may leave undetected the manic side of bipolarity and create heterogeneity in groups identified as possessing depressive attributes. This leads to interpretational problems and possibly obscures associations with coronary disease (Barrick, 1999; Goldston & Baillie, 2007). To examine the integrative model, it will be necessary to distinguish between interview and questionnaire assessments for identifying individuals with syndromal or subsyndromal mania and depression. Such efforts also should include *real-time* assessments of hyper-reactive and hypo-reactive responses to challenging and stressful events and related cognitive processes, such as perceptions of control.

Pathophysiological research

The integrative model is behavioural. It does not provide a detailed account of specific mechanisms whereby coping with challenging and stressful events contribute to CHD outcomes. However, the model does provide some general guidance for their study. It points to mechanisms that are plausibly associated with hyper-reactive and hypo-reactive responses, and also with shifts between the two coping patterns. Broadly speaking, there are three principal routes by which behavioural responses can promote CHD: (1) indirect pathways that involve initiation and maintenance of health-impairing habits, some of which were discussed earlier in this paper; (2) direct pathways that involve neuroendocrine, autonomic, cardiovascular and immunological changes precipitated by exposure to challenge and stress; and (3) psychological and behavioural reactions to illness that may delay healthcare seeking and undermine adherence to medication regimens. Although all three are potential routes linking TABP and bipolarity to CHD, the integrative model is most germane to research concerning direct physiologic mechanisms. A review of the sizeable literature on this topic is beyond the scope of this paper. However, some pathophysiological processes appear to be particularly promising mediators of the risk-enhancing effects of hyper-reactivity and hypo-reactivity.

There is growing evidence that hyper-reactive (i.e., Type A, hostile and manic/hypomanic) responses may promote coronary artery disease (CAD) through increased sympathetic adrenomedullary activity (SAM) manifested as elevations in circulating catecholamines, increased stimulation of the heart and blood vessels,

abrupt fluctuations in heart rate and blood pressure and metabolic changes including heightened serum lipid levels (Bekkouche et al., 2011; Chida & Hamer, 2008; Glass & Contrada, 1984; Kop, 1999). The association of vigorous coping with SAM activity (e.g., Frankenhaeuser, 1971; Glass, 1977; Obrist, 1981), and their role in the initiation and progression of CAD, has been recognised for several decades (e.g., Krantz & McCeney, 2002). More recently, subclinical markers of CAD progression related to SAM activity have been identified, including impaired endothelial function and immunological/inflammatory activity. Whereas these markers have been correlated with *hyper-reactive* characteristics such as hostility (Matthews, 2005), other evidence indicates they also may be associated with *hypo-reactive* responses, including indices of lack of control and depression (Kop, 1999; Maeng et al., 2008).

The latter finding takes on added significance in the light of research on the immune system and CAD. It has been reported that men who are hostile and angry, and also experience depression, show higher cytokine levels and related immune activity believed to contribute to cardiovascular disease (e.g., Boyle, Jackson, & Suarez, 2007; Suarez, 2003). In this connection, Kop (2003) has noted that activation of pro-inflammatory immune factors, such as may be reflected in measures of cytokines (e.g., interleukin-6) and C-reactive protein, is a probable candidate accounting for why depression predicts CAD. Moreover, it has been recognised that increased levels of inflammation also are associated with HPA axis function, and evidence suggests that activity of HPA is triggered by active efforts to cope with challenging and stressful life events (Weinstein et al., 2010; see Miller et al., 2007).

The findings outlined above are illustrative of work that has implicated hyper-reactivity (the hostility/anger components of TABP), hypo-reactivity (depression) and, in some cases, their combination, as correlates of pathophysiologic markers believed to contribute to cardiovascular disease (see, also, Betensky & Contrada, 2010). Note, however, that the available evidence does not address the specific effects of repeated alternations between the two modes of reactivity. We do not gainsay the possibility that each mode relates to CHD for different reasons, but the model does imply that both are involved in one process. For this reason, more work is needed to determine pathophysiologic concomitants of transitions from mania to helplessness and, perhaps, the reverse sequence.

Of some relevance to these issues is Kop's (1999) proposal that there are different pathological effects for stressful and challenging events of varying durations (i.e., chronic events, episodic events and acute events). The idea of differential impact of stress duration on CHD is not entirely new. It is reminiscent of early work by Engel (1971) and Greene, Goldstein, and Moss (1972). They suggest that ineffective coping with negative events, over time, results in feelings of helplessness at a point preceding, but close to illness onset. These feelings are accompanied by shifts in autonomic function inimical to cardiovascular health, especially sudden cardiac death (Kamarck & Jennings, 1991; Krantz & Glass, 1984). Indeed, Kop (1999) includes shifts of sympatho-vagal balance as possible mediators of cardiovascular risk.

The Engel/Greene emphasis on helplessness is generally consistent with our model of sequential hyper- and hypo-reactivity to uncontrollable stressful and challenging events. It is also consistent with the observation that vital exhaustion (viz. depression, although the constructs are not isomorphic) is the end-stage of prolonged uncontrollable stress (Kop, 1999). Therefore, we may speculate that hypo-reactivity is likely to be prevalent among initially hyper-reactive individuals who

experience the effects of uncontrollable stress and/or frustrated goal-striving. There are few studies that provide direct empirical support for our speculations. Future research needs to address this issue with special attention to the pathophysiological implications for CHD morbidity and mortality.

Conclusions

A fundamental premise of this paper is the importance of using psychological theory in research on emotions and CHD. Studies of Type A behaviours, bipolarity and CHD would benefit from work guided by a common conceptual model. That model should incorporate the effects of stressful events, challenging events (e.g., goal-striving), perceptions of uncontrollability and the occurrence and alternations of hyper-reactive and hypo-reactive behaviours. It also should include systematic efforts to represent these features of the integrative model in studies of pathophysiological mechanisms mediating the association between psychosocial factors and CHD.

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