

Type A behaviour and coronary heart disease: When will the jury return?

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The nature of the controversy

The Type A construct and its purported links with coronary heart disease (CHD) is one of the better known research topics within health psychology. Awareness has spread even beyond the speciality of health psychology and the Type A construct has penetrated well into the health-conscious media. When controversial debate overtakes such a topic, it is useful for the issues to be put clearly before a general psychology readership. While there have been recent reviews specifically dealing with prospective studies of Type A and CHD risk (Booth-Kewley & Friedman, 1987; Matthews, 1988), the construct validity of Type A, as a whole, is a wider issue. This article attempts to address the question which may best be put: where are we now?

The controversy is easily seen from two recent quotations. Reviewing a new book on the subject, Johnston (1989) writes that the area of 'Type A behaviour pattern and its link with coronary heart disease is currently in a state of some disarray and near crisis'. In contrast, Friedman & Booth-Kewley (1988) conclude: 'By our reckoning, this [body of research] adds up to convincing evidence that we should be asking how, why, and for whom, *not whether* Type A behaviour is an important element in heart disease' (italics added). What are we to make of such apparently diverging opinions?

Defining Type A

The Type A construct was invented by two cardiologists Friedman and Rosenman to describe a certain kind of individual who, they believed, tended to be over-represented in their clinical practice (Friedman & Rosenman, 1974). Type A persons were seen as having a competitive craving for achievement and recognition, a tendency towards hostility and aggression, and a sense of extreme time urgency and impatience. The Type A individual sees goals and challenges everywhere, wants to win every 'game' in life, speaks fast, acts fast, interrupts or manifests impatient gestures when faced with slower mortals, cannot abide queues, is only superficially interested in the aesthetic aspects of life and tends to measure success in terms of material gains, and number rather than quality of goals achieved.

One of the difficulties with the Type A construct is that it is a broad pen portrait which includes or suggests the presence of a number of different but perhaps interacting personality traits. Psychologists who have spent many years refining and

measuring traits are quite understandably prone to regret that the research in this area has not developed around more tried and tested measures, which avoid the artificiality inherent in dividing people into broad types – a practice which is bound to ignore the dimensional nature of real traits (Eysenck, 1985). However, that is water under several bridges. It is Type A which has, for better or worse, been measured and it is Type A which has been supposedly linked to CHD. The issue of Type A measurement is now briefly addressed.

Measurement of Type A

Friedman and Rosenman originally assessed Type A using a structured interview (SI) method (Friedman & Rosenman, 1974). The interviewer in this procedure not only asks subjects about their behaviour but also observes and elicits behaviour in the actual interview. Thus, the subject's style of speaking, how fast or explosive it is, the subject's reactions to pauses by the interviewer, and other behavioural characteristics, are all noted and recorded as part of the assessment. The structured nature of the procedure means that respectable reliability is usually achieved in terms of inter-rater classification agreement (Byrne, Rosenman, Schiller & Chesney, 1985).

Raters have traditionally favoured the use of four categories. Type A1 and A2 simply differentiate degree of Type A and are often collapsed into a single category. Type B indicates a notable absence of Type A characteristics. Finally, Type X is an 'unsure' middling category where Type A characteristics are not sufficiently in evidence to justify a Type A judgement but not so totally absent as to indicate a Type B judgement.

Although the SI method of Type A assessment remains a sort of 'gold standard' against which other measures are judged, reliable self-report measures have been used by researchers. As we shall see, their validity has proven in some cases to be more problematic. The most widely used self-report instrument has been the Jenkins Activity Survey (JAS) (Jenkins, Rosenman & Zyzanski, 1974), but others such as the Framingham Type A Scale (FTAS) have been developed (Haynes, Levine, Scotch, Feinleib & Kannel, 1978).

The predictive validity of Type A

The authoritative view of a review panel, made up of distinguished American scientists gathered together in 1981 under the auspices of the National Heart Lung and Blood Institute, was that Type A could be duly added to the official list of traditional coronary risk factors, such as high blood pressure, high serum cholesterol level and smoking (Review Panel on Coronary-Prone Behavior, 1981). Their conclusions relied heavily on the results from the first major prospective study, the Western Collaborative Group Study (WCGS), in which a sample of over 3000 Californian males, initially free of CHD and between the ages of 39 and 59, were followed up over a period of eight and a half years. It was found that the subsequent incidence of CHD was twice as great among Type As (assessed by SI) than among Type Bs (Rosenman, Brand, Jenkins, Friedman, Straus & Wurm, 1975). To put this relatively greater risk into some absolute perspective it can be noted that 7 per cent

of the entire sample developed some signs of CHD and two-thirds of these were Type A. This degree of risk is comparable with that of traditional physical risk factors. Moreover, statistical analysis revealed that the risk associated with Type A was independent of other risk factors.

Further major support for the role of Type A as a coronary risk factor soon came from the opposite side of the United States, in Framingham, Massachusetts, where a large-scale investigation of CHD was underway (Haynes, Feinleib & Kannel, 1980). These researchers had required their subjects to fill in several psychosocial rating scales at the beginning of the study and certain key ratings were grouped to form a Framingham Type A measure. The FTAS succeeded to a similar extent in predicting CHD over a period virtually identical to that of the Californian study (Haynes *et al.*, 1980). The Framingham study recruited both male and female subjects, and it was therefore the first major study to show Type A as a risk factor for women. However, its predictive power with respect to CHD was better in regard to angina than myocardial infarction.

Since these first major studies, the results of many further prospective investigations have been reported. Most have been considered in recent reviews (Booth-Kewley & Friedman, 1987; Matthews, 1988). Since several investigators have now reported negative or even contradictory results in relation to Type A, a major task is to try to determine what features distinguish supportive and non-supportive studies. Two such features seem to stand out. Firstly, studies which have used the SI method of assessing Type A have tended to indicate that it is a genuine risk factor, whereas studies that have assessed Type A using the JAS have been particularly prone to negative conclusions. Since the classificatory agreement of the JAS with the SI is known to be little better than 60 per cent (one would expect 50 per cent agreement by chance), we might expect research using the JAS measure to be problematic. From a reasonably wide but not exhaustive review of relevant reports, this author has found that estimates of the variance shared by the SI and the JAS seldom much exceed 10 per cent and many researchers have explicitly cautioned that they should not be seen as substitute measures (Mayes, Sime & Ganster, 1984). In at least one report, JAS mean scores did not even relate linearly to SI categorization (Byrne *et al.*, 1985). The FTAS fares somewhat better in its agreement with the SI. Little wonder then that Friedman & Booth-Kewley (1988) have now called for the virtual abandonment of the JAS as a research instrument. It is also worth mentioning that the JAS has very few items pertaining directly to anger/hostility. These particular components, as outlined later, have received considerable attention in recent studies of Type A and CHD (e.g. Dembroski, MacDougall, Williams, Haney & Blumenthal, 1985).

The second distinguishing feature of the studies which have failed to show Type A as a risk factor for CHD is that they tend to be so-called 'high risk' studies. Such studies typically select subjects who are already known to be at risk for CHD and many of these studies have taken subjects who have already suffered one episode of CHD (e.g. Case, Heller, Case, Moss *et al.*, 1985; Ragland & Brand, 1988). Other studies have selected subjects who are at greater risk of CHD by reason of another risk factor. One such British study reporting negative findings (Mann & Brennan, 1987) used subjects exhibiting mild hypertension.

High risk studies lead to several interpretative difficulties. For the most part Type A measures are taken from subjects who are already aware of their greater risk and this may affect their response to assessment, particularly self-report measures. Some studies have also indicated that they had a higher prevalence of Type As in them, thus perhaps limiting Type A differences to a degree where association with CHD is difficult to demonstrate. Type A subjects who have survived an initial episode of CHD may actually represent a subsample of Type A persons different in important respects from those who did not survive. One factor which has received surprisingly little attention is that survivor Type As may be more likely to seek help for early warning symptoms. Survivor Type As may generally be better health monitors, or even complainers. This may be a particularly salient consideration when mortality rate is used as a key variable. Survivor Type As may also be precisely those who have suffered less atherosclerosis and therefore are unlikely to differ from Type Bs in regard to future CHD indices.

One high risk study (Ragland & Brand, 1988) is worthy of special mention since it reports an apparently contradictory finding of greater risk for Type B subjects. It is particularly interesting because the group followed up were the survivors from the original WCGS. Although this group included more Type A persons, by virtue of the original findings, it was nevertheless found that over a further follow-up period mortality from CHD incidence was actually higher among the Type B persons who had survived their original incident. How can we account for this result? Any suggested answer must remain speculative, but at least two possibilities, not mutually exclusive, can be considered.

The first takes up the point that Type A survivors may be crucially different from Type As having fatal first incidents. Let us assume that Type A has two endangering effects in regard to CHD: firstly, a chronic effect which aids the process of atherosclerosis; secondly, an effect which makes the eventuality of an obstruction or blood clot more likely. The second effect may only be of major significance if atherosclerosis has advanced to a certain degree. If we suppose that the Type A persons who survived their first incident were those with less atherosclerosis and, perhaps for a variety of reasons, were less prone to develop atherosclerosis, and if we further suppose that the Type Bs who had an initial incident, despite their low risk status, were those who had and were prone to atherosclerosis, then we have invented a scenario in which the results of Ragland & Brand become more understandable.

The second line of reasoning supposes that the psychological impact of a heart attack may be different for Type As than it is for Type Bs. Type A persons may be more prone to reassess their life-style and modify their values and behaviour. At the very least it can be argued that they have scope to do so, certainly more scope than their Type B opposites who seem to have suffered despite their slower pace of life! Although this may seem unduly speculative, it is worth noting that detailed examination of the figures provided by Ragland & Brand indicates that their reported effect is apparent only for cases where an original myocardial infarction was 'overt', that is to say consciously registered at the time. The figures for so-called 'silent' myocardial infarctions, where damage only comes to light at some later time, seem to show no difference between Type A and B subjects with respect to mortality. Given that anecdotal reports from persons who have consciously suffered a heart

attack often indicate a great sense of 'life-endangerment' it is not beyond the bounds of possibility that Type A persons resolve to reassess their life-style to a greater extent than Type B persons following a significant coronary event.

What then should be the conclusion regarding the status of Type A as a risk factor for CHD? It might be the case that in population studies, as opposed to high risk studies, and using Type A measures other than the JAS – but preferably the SI – the evidence is strongly supportive. However, it would then have to be recognized that the database for such a conclusion is considerably reduced in terms of sample size and also the provenance of evidence. This means that, after several years of research, further prospective studies are necessary to diminish the need for speculative interpretations. Referring to the title of this article, the 'jury is still out', but perhaps for good reason. However, the validity of the Type A construct should not be assessed only in terms of CHD risk studies. It has a potentially wider construct validity in that it not only seems to relate to CHD but to mechanisms which may explain the link with CHD.

Type A, psychophysiological response and CHD

It has been thought for some time that psychophysiological response to stressors, particularly excessive neuroendocrine activity, may be implicated as a mechanism promoting premature CHD (Williams, 1978). Reviews of such evidence (e.g. Krantz & Manuck, 1984), suggest on the one hand that psychophysiological response may indeed be an important factor, but on the other hand they caution against accepting as meaningful any simple construct such as 'reactivity to stress' because different patterns of physiological response exist. Such caution ought to be exercised especially before making the overly simplistic leap to the assertion that Type A persons are at risk because they are physiologically over-reactive to stress. Nevertheless, suspicion has attached itself to neuroendocrine response for good reason, and enough is known at a more 'molecular' level, e.g. effects of catecholamines on platelet aggregation (Ardlie, Glew & Schwartz, 1966), to draw plausible links with pathological processes such as atherosclerosis. If a final synthesis is to be achieved in this area, it must involve differentiation of all of the three constructs which have variously been linked: Type A, reactivity and coronary heart disease. But such dissection will be more profitable if it proceeds in an organized manner. Flawed positions with some apparent truth in them may indeed need refinement, but at least they are temporary structures.

The numerous studies which have now been conducted using SI or JAS assessment of Type A form the focus of earlier reviews (Houston, 1983; Matthews, 1982). Most, but by no means all, have tended to differentiate Type As from Type Bs in the predicted direction. Measures have included heart rate (MacDougall, Dembroski & Krantz, 1981; Mayes *et al.*, 1984), blood pressure (Dembroski, MacDougall, Shields, Petitto & Lushene, 1978; Manuck, Craft & Gold, 1978), skin conductance (Lovallo & Pishkin, 1980; VanDoornen, Orlegeke & Somsen, 1980) and catecholamine response (Contrada, Glass, Krakoft, Krantz, Kehoe, Isecke, Collins & Elting, 1982; Frankenhaeuser, 1980). The most consistent results have

come from measures of systolic blood pressure (Holmes, 1983). More recently, positive results have been found using the FTAS. A/B differences have been found on systolic blood pressure (Smith, Houston & Zurawski, 1985) and heart rate (Evans & Fearn, 1985; Evans & Moran, 1987*a*).

An important question, given that positive findings have not been universal, is whether there are particular kinds of situation which most favour the emergence of Type A/B differences. Broadly speaking, reviewers have emphasized the need for the task or situation to challenge the subjects sufficiently. However, unless we have a theory as to what constitutes effective challenge for Type A subjects, there is a temptation simply to 'see' common denominators in just those studies which have produced positive results. It is therefore necessary to consider theories of what it is to be Type A.

A clue to what may be theoretically relevant is provided perhaps by the very nature of the recommended SI method of assessing Type A. The interview itself often constitutes a challenge to subjects and is likely to arouse anger, especially in hostile persons who may consider their authority is under attack. We shall see that a heightened need to be in control and to bolster self-esteem have both been suggested as core elements of Type A.

Type A and the need to be in control

That an overdeveloped need to control events lies at the core of the Type A behaviour pattern is a theory associated with Glass (1977). He suggests that in situations of challenge, but where control is lacking or ambiguous, the Type A person exhibits relentless striving leading to frustration and exhaustion. Type As will then decline into greater 'helplessness' than Type Bs. Thus he predicts that Type As will tend to be the victims of cycles of *hyper-responsiveness* and *hypo-responsiveness* which are both associated with patterns of physiological response that can be linked to processes which would favour the development of CHD.

Glass (1977) presents considerable evidence from laboratory studies of induced 'helplessness' to support this theory. Brunson & Matthews (1981) similarly report that Type As exposed to repeated failure tend to exhibit significant helplessness effects. It has also been shown that Type As more than Type Bs will choose to 'monitor' for a warning stimulus of a low probability shock even when objectively they have little or no control over it and even though such monitoring is associated with higher cardiovascular arousal (Evans & Fearn, 1985; Evans & Moran, 1987*a*). Also, in a further report, Evans & Moran (1987*b*) found that slow decline in heart rate at the end of a challenging trial (slow 'unwinding') was particularly characteristic of those high on Type A and high on internal locus of control.

However, in practice, it is not always easy to be precise as to what constitutes 'control' (Thompson, 1981). This was apparent for some male subjects in the 'monitoring' experiments just described who behaved counter-intuitively: they proved more likely to reject control the more of it that was offered. This meant that predictions about Type A and control seeking were effectively confirmed only for the female subjects in the sample. Comparable seemingly irrational behaviour by male subjects has been reported before in a very similar experimental situation (Averill,

O'Brien & DeWitt, 1977). The possibility arises that such subjects may have been trying to show superordinate control over the situation and the experimenter by not doing what was expected of them. This in turn may indicate, in regard to Type A hypotheses, that what subjects may at root be most concerned with controlling is the 'image' that they present and that control-seeking theories of Type A are perhaps secondary to theories which emphasize the importance of self-concepts.

Type A and self-esteem

Price (1982) suggested that the competitive relentless striving of Type A persons, their hostile emotions and so forth, stem from a belief that self-esteem is to be measured exclusively by accomplishments. Beneath the superficial achievement striving, there lies a more profound sense of inadequacy and perhaps low self-esteem, although predictions in regard to such measures have to contend with the fact that Type A persons may be highly motivated not to reveal such weakness to others.

There is certainly evidence, direct and indirect, to support the theory outlined by Price. In a study which actually used threat to self-esteem, Pittner & Houston (1980) showed that Type A subjects showed more denial responses than Type B subjects. Furnham & Linfoot (1987) report that Type As reveal a stronger need than Type Bs to 'prove themselves'. Henley & Furnham (1989) asked subjects to rate their 'actual' and their 'ideal' selves on a list of 40 trait-like adjectives. Results showed that Type A persons showed greater discrepancies between their ratings of actual and ideal selves than did Type B persons. Interestingly, however, the same study also suggested that low self-esteem is not necessarily synonymous with negative self-evaluation. It was found, for example, that Type A subjects were more likely than Type B subjects to rate their ideal selves as 'dominating', 'demanding' and 'conceited'.

Analysis of the components of Type A Behaviour Pattern

Both the theories addressed above have arisen from attempts to view the Type A person as a whole and underpin the description with an underlying core element. Another approach has been to recognize the limited predictive validity of global Type A in relation to CHD and to seek to identify fractional components which may give better prediction. Although no firm conclusions are yet justified, some highly suggestive findings have emerged. This essentially atheoretical approach seems, as we shall see, to lend support to some of the notions implicit in the theories already considered.

Eysenck & Fulker (1983) identify Type A individuals as high on neuroticism. If Type A persons are more anxiety-prone, this does not of itself seem to be related to risk of myocardial infarction, although anxiety measures do seem to predict angina (see Eysenck, 1985, for a review of relevant studies). Interestingly, the large Framingham study, not cited by Eysenck, could be interpreted as supporting his case. The overall significant predictive relationship between the FTAS and CHD relies heavily on incidence of angina. Moreover, the FTAS is more strongly and more consistently correlated with anxiety measures than any other Type A scale (Byrne *et*

al., 1985; Evans & Moran, 1987*a*). Unfortunately, the diagnosis of angina is far more 'subjective' than myocardial infarction and may be influenced by how much the patient complains. Since anxiety-proneness measures such as Eysenck's Neuroticism scale are also measures of 'complainers' syndrome', the interpretation of findings in this area is fraught with problems. Eysenck (1985) even cites one study (Elias, Robbins, Rice & Edgecombe, 1982) which reports a negative correlation between anxiety and the degree of arterial stenosis ('narrowing') assessed by angiography.

Angiographic studies have, however, shed light on the role of other components of the Type A Behaviour Pattern, in particular anger and hostility (Dembroski *et al.*, 1985). The background to such studies is similar to that in relation to CHD itself as an end-point measure. About half the angiographic studies have found that global Type A measures do relate to degree of atherosclerosis (e.g. Frank, Heller, Kornfeld, Sporn & Weiss, 1978) and about half have found no significant relationship (e.g. Dimsdale, Hacket, Hutter, Block & Catanzano, 1979).

In an important angiographic study using audiotapes of SI assessments of Type A, Demboski *et al.* (1985) rated their subjects on 12 distinct components of the global profile. Of these only two significantly predicted degree of atherosclerosis: 'potential for hostility' and 'anger-in'. Moreover, the two interacted such that atherosclerosis was particularly pronounced in subjects high on both measures: someone with a lot of potential for hostility but uncomfortable about expressing angry emotions openly. This study is important for two reasons. Firstly, it does seem to offer some resolution of the ambiguities posed by angiographic findings as a whole. Secondly, it is in broad agreement with reanalyses of WCGS data concerning the relationship between Type A and CHD.

Matthews, Glass, Rosenman & Bortner (1977) report that CHD cases in the original WCGS were primarily distinguished from controls on the basis of hostility, anger, irritation, competitiveness and vigorous voice stylistics. Prospective studies have also implicated a quite separate (MMPI) measure of hostility in CHD (Barefoot, Dahlstrom & Williams, 1983; Shekelle, Gale, Ostfield & Paul, 1983). It is therefore not surprising that most recent writers on the subject of Type A have tended to mention hostility as the most promising 'active' component of the Type A global pattern, although there are some dissenting voices (Friedman & Booth-Kewley, 1988). Most recently, Williams (1989) has suggested the possible importance of 'cynical' hostility as a critical coronary-prone component of Type A behaviour.

If further research continues to implicate hostility and anger expression, the Type A literature will begin to look more ordered and not only in relation to prediction of end-points such as CHD and coronary stenosis. Theories of Type A which have stressed self-esteem, or control (in the service of self-esteem), would themselves predict that hostility and difficulties expressing anger flow naturally from the 'pathological' belief system which is posited.

Conclusions and suggestions

A certain subjectivity is inevitable when trying to sum up a literature as vast as that supplied by the Type A construct. However, the following list of conclusions and suggestions seems warranted.

(1) Type A as a global measure has yet to show itself as an indisputable risk factor for CHD. On the one hand, the evidence from population-based studies is strong that somewhere within the measure is a 'kernel' of risk. On the other hand studies based on subgroups already established as being at risk for CHD are likely to face considerable interpretative difficulties.

(2) The wider construct validity of Type A remains strong. Predictions concerning physiological and behavioural responding have not always been supported but there is a coherent pattern in the evidence which suggests that Type A/B differences are particularly likely to emerge when challenge is perceived, when control over that challenge is uncertain, and when failure to control the challenge is seen as threatening to self-esteem.

(3) The core element of Type A in predicting CHD may well involve hostility potential and anger-in. These emotional qualities would also be predicted by cognitive theories of Type A.

(4) More attention needs to be paid to the nature of the disease in assessing psychological risk. Angina and myocardial infarction should be routinely differentiated in reports. Attention should also be routinely paid to end-point measures (morbidity vs. mortality).

(5) While Type A research will undoubtedly continue, researchers and editors should perhaps seek to protect readers and reviewers from overload by not reporting what sometimes seem trivial findings. Type A only has importance because it might well be a coronary risk factor. We do not necessarily need to know every discovered difference between Type As and Type Bs, especially if the difference seems as if it may well have been 'discovered' *post hoc*. The existing 'bank' of studies is already sufficiently vast to allow meta-analyses to be conducted to confirm or disconfirm key hypotheses.

(6) Finally, the jury is still out; it should remain out; it should not, however, be sent home.

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