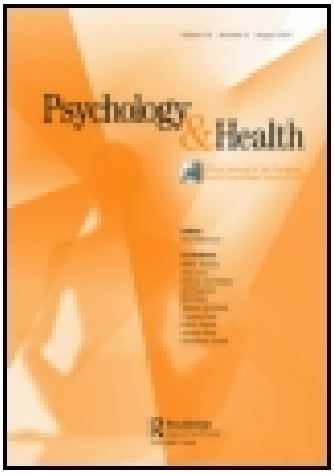

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Type a behaviour, social contact and coronary death

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TYPE A BEHAVIOUR, SOCIAL CONTACT AND CORONARY DEATH

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The risk of coronary death associated with Type A behaviour (Framingham definition) and social contact was examined using a retrospective case-control design. Logistic regression analysis of 134 male cases and 339 controls showed that increased risk was associated with Type B behaviour, and independently with limited social contact. Discussion focuses on issues surrounding the use of retrospective, proxy data; and on the speculation that Type A behaviour may increase risk of non-fatal coronary events, but protect against coronary death.

KEY WORDS: Type A behaviour, social contact, coronary death.

INTRODUCTION

Many studies have been published on the association between Type A behaviour (TAB) and the non-fatal manifestations of coronary heart disease (CHD): myocardial infarction (MI) and angina. The findings are mixed and controversial, but are generally interpreted as showing that at least some aspects of the pattern, notably hostility and hard-driving competitiveness, increase non-fatal CHD risk in healthy populations (e.g. Booth-Kewley and Friedman, 1987; Matthews, 1988; Houston *et al.*, 1992).

In contrast, the available evidence on the relationship between TAB and coronary death (CD) is sparse but striking, in that it suggests either no relationship, or the possibility of an inverse association. In a prospective study of 257 men with a history of CHD, Ragland and Brand (1988a) found that the CD rate for Type As, assessed using the Structured Interview (SI), was 58% of that found in men without Type A characteristics (Type Bs). Two other studies of MI survivors, whose behaviour pattern was assessed with the Jenkins Activity Survey, also found this inverse association, though in neither case was it statistically significant (Case *et al.*, 1985; Shekelle, Gale and Norusis, 1985). Ragland and Brand (1988b) have also reported a prospective analysis of coronary deaths in the Western Collaborative Group Study cohort, where they found no overall difference between Type As and Bs, classified using the SI 22 years earlier. Intriguingly, they did find a significantly increased risk for Type Bs during the interval 11–15 years after assessment. Finally, Orth-Gomer and Unden (1990) have reported no difference in coronary mortality rates for SI-classfied As and Bs over a 10 year period.

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Interpretation of these findings is complicated by the possibility of change in TAB over time, either in response to the experience of CHD (Ragland and Brand, 1988a), and/or as a function of age (Sparacino, 1979). Some clarification may be gained by focusing on deaths from first coronary events, as Gallacher (1989) has suggested. Ragland and Brand (1988a) did provide an analysis of deaths within 24 hours of a first coronary event, and reported no A/B difference. However, their sample contained only 26 cases. Similarly, Orth-Gomer and Unden's (1990) sample produced only 7 coronary deaths in men who were initially free of CHD.

Some clarification may also be achieved by assessing Type A behaviour shortly before coronary death, rather than only 10 or 22 years earlier. Clearly, large-scale prospective studies which include repeated assessment of TAB are the ideal strategy to achieve these aims, but they are prohibitively expensive given the relative infrequency of coronary death. The case-control design, despite its well-known limitations, provides a way of acquiring large samples of coronary death cases with no CHD history, and estimates of the characteristics they exhibited shortly before death (Appels, 1992). One objective of the present study was to assess the relationship between TAB and coronary death using such a design. For reasons given in the methods section, the definition and measure of TAB used in the present study were those developed in the Framingham study (Haynes *et al.*, 1978). The specific TAB elements assessed are shown below in Table 4.

Risk of coronary death has also been found to be prospectively associated with limited social contact, integration or support (House, Landis and Umberson, 1988; Schwarzer and Leppin, 1989). Virtually all of this research has been pursued independently of that on TAB, despite theoretical and empirical grounds for combining them. Good theoretical arguments for studying TAB in its social and ecological context have been articulated (Smith and Anderson, 1986; Margolis *et al.*, 1983), though little heeded. Empirically, associations between TAB and social activity (e.g. Spicer, Jackson and Scragg, 1993) raise the possibility of confounding and mediation processes. Moreover, Orth-Gomer and Lunden (1990) have provided evidence of an interaction effect whereby social isolation increases risk of coronary death in Type As, but not in Type Bs.

The general objective of the present study was to examine the independent and joint effects of TAB and social contact on the risk of coronary death. In the light of the available evidence we hypothesised that social contact would be inversely related to risk of coronary death, but left the direction of the TAB and interaction effect unspecified. We were particularly interested in comparing the results of the present analysis with those from our case-control study of non-fatal MI in men, where we found that higher risk was associated with Type A status, but not with low social contact (Spicer, Jackson and Scragg, 1993).

METHODS

Design

The present data were gathered as part of a large case-control study of non-fatal MI and CD in Auckland, New Zealand during 1986–88. In this article we report analyses of psychological differences between CD cases and controls using data gathered from proxy respondents. Psychological findings from the MI section of the study have been

reported elsewhere (Spicer, Jackson and Scragg, 1993); whilst a full account of the design can be found in Jackson, Scragg and Beaglehole (1991). Issues of bias generated by the use of retrospective, proxy data are addressed in the discussion section below.

Subjects

An overview of how the final samples of cases and controls were formed appears in Table 1. All coronary deaths in the Auckland statistical area aged 25–64 years during the study period were identified using the ARCOS CHD register (Bonita, Beaglehole and North, 1983). This register, which incorporates the diagnostic criteria for CD developed for the multinational MONICA project (WHO, 1988), has been shown to capture over 99% of CD cases (Jackson *et al.*, 1988). Only males were considered for the following analyses, since there were too few female cases to test the hypotheses of interest. The next of kin or a close friend of each case was approached usually 4–6 weeks after the death, and asked if he or she would provide information on the deceased. For the present analyses, Maori and Pacific Island cases were excluded since the proxy response rate was low; and since the cross-cultural appropriateness of the psychological measures was not established. Of those asked to report on white, male cases, 83% agreed to participate. Finally, first event cases were selected in order to control for the psychological effects of having suffered from MI or angina prior to the fatal coronary event. This last criterion excluded 46% of the remaining cases, and resulted in a study sample of 134 cases.

The CD controls were derived from the MI control sample. This latter sample had been randomly selected from the Auckland electoral rolls, group matched by age and sex with the MI cases. To increase the power of the design, controls were over-sampled, with a planned case-control ratio of approximately 1 : 1.5 in males. Of those men selected, 80% provided data about themselves for comparison with that from MI cases. A random subsample of these MI controls was then asked if their next of kin could be approached in order to provide proxy data on the MI control. Thus the CD case and control data were matched in the sense that both sets were furnished by knowledgeable others, rather than by the target individuals themselves. From the sample of white, male controls' next of kin, 85% agreed to participate. Applying the same exclusion criteria as for the CD cases resulted in 339 controls for the present analyses.

Procedure

All providers of information about a CD case or control were visited at home by one of three trained interviewers. These interviews took place 6–8 weeks after the case's death or the control's own interview. The structured interview lasted 30–50 minutes and covered a range of topics besides the psychological ones, including demographic

Table 1 Formation of case and control samples

	Cases	*Controls*
Number of proxy respondents approached	440	460
Number agreeing to participate	298	366
Number of participants excluding Maori and Pacific Islanders	249	355
Number of participants without history of coronary heart disease	134	339

characteristics, physical activity, alcohol consumption, smoking and medical history. Interviewers were blind to the study hypotheses, but it was not practicable to blind them to the case-control status of the target person.

Measures

The design of the present study placed various constraints on the choice of instruments for assessing Type A behaviour and social contact. A proxy respondent cannot be assumed to possess detailed knowledge of the target person's mental life. Moreover, the usual retrospective problems of accurate recall of transient or distant phenomena are compounded when proxy respondents are used. From a practical perspective, only a small portion of the interview time was available for psychological assessment. Accordingly, the chosen measures were brief, and focused mainly on behaviour which is observable and relatively stable over time. To aid memory further, respondents were instructed to consider only the preceding year.

The most highly recommended measure of TAB, the Structured Interview, was clearly not usable for proxy respondents. The Framingham measure (FTAS) (Haynes *et al.*, 1978) was chosen since, of the various self-report measures available, it came closest to meeting the above criteria, whilst demonstrating adequate validity and reliability. Its ten items, shown in Table 4, address various aspects of the Type A pattern as originally defined, the most notable omission being the anger/hostility component. Scores on the FTAS have been associated prospectively with CHD in men and women in the general population (Haynes, Feinleib and Kannel, 1980). We have recently reported a positive correlation between FTAS score and MI incidence in New Zealand men and women (Spicer, Jackson and Scragg, 1993). Gallacher, Yarnell and Butland (1988), using a short form of the FTAS, have also found a positive association with MI incidence in Welsh men. The criterion validity of the FTAS has therefore received some support in three different countries. The measure is adequately reliable (Haynes *et al.*, 1978). and had a Cronbach's alpha coefficient of 0.68 in the present study. In the following analyses the Type A variable was dichotomised at the median of 0.4. This follows the procedure used in the Framingham study, and in our earlier MI analyses.

To assess social contact participants were asked to record the: marital status; church membership; number of house residents; number of group memberships; number of social acquaintances and; number of close friends of the target person. These items in various combinations have been found to predict CHD (Orth-Gomer and Unden, 1987). We have also found them to be associated with MI incidence in New Zealand women (Spicer, Jackson and Scragg, 1993). The details of the scoring sysyem for obtaining the social contact score can be found in Spicer and Hong (1991). No Cronbach's alpha is provided for the social contact measure since the score can legitimately be derived from independent subsets of items. For the following analyses the social contact score was trichotomised to form low, medium and high contact groups of approximately equal size. This allowed us to explore non-linear relationships between social contact and risk of coronary death.

Since age and social class are related to CHD and to Type A behaviour and social contact, they were included in the analyses as covariates. Socioeconomic status (SES) was assessed using a modified version of the British Registrar-General's classification

of social class, which is based on occupation (Pearce *et al.*, 1983). In the following analyses this six-fold classification was collapsed into a white-collar/blue-collar dichotomy by combining classes I, II and IIIN, and classes IIIM, IV and V.

Statistical analysis

All statistical analyses were carried out using SPSSPC programs (Norusis, 1992). In the main analysis multiple logistic regression was used to examine the individual and joint relationships of TAB and social contact with coronary death, whilst controlling for age and SES.

Type A/B, SES and the dependent case-control status were coded as 1/0 variables with Type A, white collar and CD cases each assigned a value of 1. The social contact trichotomy was transformed into two dummy variables using ordinal coding. Thus the first variable represented the contrast low vs medium and high contact; and the second represented low and medium vs high contact. These contrasts are of particular interest where a threshold effect, such as that due to social isolation, is possible (Walter, Feinstein and Wells, 1987). Age was analysed in its original form as a continuous variable. The main analysis followed a two step hierarchical strategy. At the first step, Type A behaviour, the two social contact variables, age and SES were entered into the logistic model. At the second step the two variables representing the interaction of Type A behaviour and social contact were entered. The logistic regression was followed by exploratory analyses of the correlation of FTAS and social contact items with case-control status. For these analyses, point-biserial coefficients were used where the item had been rated on a continuous scale, and phi coefficients where the item was a dichotomy.

RESULTS

The distributions of the study variables for cases and controls are shown in Table 2. Whereas nearly half of the controls were classified as Type A, less than a third of the cases fell into this category (Chi square = 10.99, $p < 0.001$). Cases were reported as having less social contact than controls (Chi square = 29.22, $p < 0.00001$). This difference was most striking in the extreme categories, where cases were more than

Table 2 Distributions of Type A behaviour, social contact, age and socioeconomic status for cases and controls

	Cases	*Controls*
Type A	40(31%)	161(48%)
Type B	88(69%)	172(52%) [a]
Low social contact	62(48%)	76(23%)
Medium social contact	45(34%)	144(43%)
High social contact	23(18%)	112(34%) [b]
White collar	71(53%)	206(61%)
Blue collar	63(47%)	132(39%)
Age in years	M = 55.61	M = 51.16
	SD = 7.24	SD = 8.26 [a]

[a]: $p < 0.001$ [b]: $p < 0.00001$

twice as likely as controls to be socially isolated. The sample of cases contained fewer white-collar workers, but this difference was not statistically significant (Chi square = 2.51, $p > 0.1$). However, the cases were more than 4 years older than the controls on average ($t = -5.47$, $p < 0.001$).

In the control sample, which provides the best estimates of population characteristics, Type As were more likely than Type Bs to be younger ($t = 3.04$, $p < 0.001$) and to be white collar (Chi square = 8.7, $p < 0.01$); but had similar levels of social contact (Chi square = 4.51, $p > 0.1$). Patterns of social contact did not differ by SES (Chi square = 0.31, $p > 0.8$). However, older men were reported as having notably less contact than their younger counterparts ($r = -.27$, $p < 0.001$).

These various relationships raise the possibility of confounding, and the need for multivariate estimates of the associations between the psychological variables and case-control status. Table 3 shows the results of the logistic regression analysis. The statistics provided are: the regression coefficient for each variable; the Wald statistic which uses the chi square distribution to test that a regression coefficient is zero; the odds ratio for each variable with its 95% confidence interval; and an overall chi square test of the relationship between all of the variables and case-control status. The results for the Type A X social contact interaction are not shown since they were clearly insignificant. This was evident from the minuscule change in the –2 log likelihood measure of fit from 490.1 at the first step to 487.6 at the second step.

The results show clearly that Type B behaviour and low social contact are both independently related to increased risk of coronary death. The odds of Type A men being in the CD sample are 0.59 times that for Type Bs, independent of their age, social class and degree of social contact. For men in the bottom third of the social contact frequency distribution the odds of being in the CD sample are approximately 2.22 times that for men with greater contact. Since the other contrast was clearly insignificant, the risk of CD appears to be associated with social isolation, rather than being a linear function of social contact. The analysis provided no evidence of any joint effects of Type A behaviour and social contact on CD risk. Further exploratory regressions using a variety of linear and non-linear ways of scaling the variables and forming the interaction terms also produced no significant effects.

Having found the Type B and social contact effects, we conducted exploratory analyses of the differences between cases and controls within each measure. Since the overall effects did not appear to be confounded with each other, or with age and SES, no statistical control was exercised. However, the number of analyses involved necessitates a cautious interpretation of the results shown in Table 4.

Table 3 Multiple logistic regression of case-control status on Type A behaviour, social contact, age and socioeconomic status

	Coefficient	*Wald*	*Odds ratio**
Age (increase per year)	0.05	10.81[b]	1.05 (1.02 – 1.09)
White/Blue collar (1/0)	–0.21	0.84	0.82 (0.53 – 1.26)
Type A/B (1/0)	–0.52	5.04[a]	0.59 (0.38 – 0.94)
Low/more contact (1/0)	0.80	9.97[b]	2.22 (1.35 – 3.65)
High/less contact (1/0)	–0.33	1.22	0.72 (0.40 – 1.29)
		Model chi square = 51.97[c]	

* This shows the risk of being a CD case for men in the category coded 1 relative to those coded 0, followed by the 95% confidence interval in brackets

[a]: $p < 0.05$ [b]: $p < 0.001$ [c]: $p < 0.0001$

Table 4 Relationship of case-control status with Type A and social contact characteristics

Type A characteristic	*Correlation coefficient**
Competitive and hard driving	−.05
Usually pressed for time	−.25[c]
Bossy or dominating	−.13[b]
Need to excel	−.14[b]
Eating too quickly	−.03
Get upset when waiting	−.09[a]
Pressed for time at day's end	−.16[c]
Concerned with work after hours	.05
Stretched by work to limits	.13[b]
Dissatisfied with work performance	.02
Social contact characteristic	
Number of personal friends	−.17[c]
Number of social acquaintances	−.19[c]
Number of household residents	−.07
Number of group memberships	−.08
Married/permanent relationship	−.10[a]
Church attendance	.02

* A negative coefficient indicates that cases were less likely than controls to exhibit a characteristic. The numbers shown are point-biserial coefficients, or phi coefficients where the characteristic is a dichotomy.
[a]: $p < 0.05$ [b]: $p < 0.01$ [c]: $p < 0.001$

With respect to the FTAS items, cases were less likely than controls to be seen as pressed for time, bossy or dominating, or having a strong need to excel in most things. In contrast, they were seen as more likely than controls to be stretched by work to the limits of their energy and capacity: an item which would have increased their score in the Type A direction. In terms of social contact, cases were less likely than controls to be married, and were reported as having fewer personal friends and social acquaintances.

DISCUSSION

The results suggest that CD risk is associated with Type B behaviour, as measured by the FTAS, and with low social contact. These associations appear to be independent of each other, and uncomplicated by any interaction between the two factors. Before we speculate on the meaning of these findings, it is necessary to ask to what extent they are explicable in terms of design artifacts. The main causes of concern in the present design are the possibilities of selection, recall or proxy bias, and of confounding from unmeasured variables. Selection bias seems unlikely, given the sampling procedures adopted, and the relatively high response rates. The exclusion of various subgroups may have introduced some bias, but arguably their inclusion would have generated even more serious problems.

With respect to recall bias, it is probable that case informants had spent time trying to identify the causes of death, and were especially sensitised to those attributes which are popularly believed to be CHD risk factors. Conversely, control informants would have had no such experience, and may actually have under-reported control characteristics. This type of concern is commonly and rightly raised in the context of retrospective

studies of psychological risk factors for CHD. However, it is notable that the concern is based more on presumptions than on data about informants' responses. Booth-Kewley and Friedman (1987) have found in their meta-analyses of TAB and CHD that prospective studies report weaker associations than do retrospective studies. In contrast, Appels (1992), using comparative studies of the link between vital exhaustion and MI, has shown that retrospective and prospective studies produce similar estimates of the magnitude of association, if the time interval between assessment and the coronary event is taken into account. In the absence of more data on recall bias in this context, it is only possible to provide circumstantial arguments to counter the claim that it has occurred in a particular setting.

Although the case and control informants may have begun their interviews with different histories of speculation, nothing in the interview process should have augmented any such differences. Thus, the interviewers were blind to the study hypotheses, and followed a pre-determined schedule which moved steadily from topic to topic. More importantly. the pattern of results exhibited several reassuring characteristics. For example, the mean Type A score and its correlations with other variables were similar to those found in the prospective Framingham study (Haynes *et al.*, 1978). With the exception of the TAB X social isolation interaction, reported from only one study, the associations between the psychological variables and CD were similar in direction and magnitude to those found by other investigators, as discussed in the introduction.

It seems likely that considerable publicity has forged a link in the public mind between Type A behaviour and CHD. Yet the present results show that the 'opposite' Type B behaviour is associated with CD risk. Moreover, in the same case-control study we have demonstrated that Type A behaviour is related to male risk of MI, but social contact is not (Spicer, Jackson and Scragg, 1993). Further, the configuration of MI results is different for males and females. Accordingly, any attempt to explain away the results in terms of recall bias would have to argue for patterns of bias which differ by disease endpoint and by sex, and which run counter in some cases to popular stereotypes. Such explanations are obviously possible, but they do seem to strain parsimony, if not credulity.

It could be argued that the MI and CD results differ because they are based on self-report and proxy data respectively, thereby raising the general issue of proxy bias. The design of the present study allowed us to shed a little light on this issue since self-report and proxy data were available for nearly 400 individuals who served as both MI and CD controls. These analyses showed that the means and standard deviations were strikingly similar for the self-report and proxy measures of Type A and social contact. At the individual level the self/proxy FTAS scores were correlated 0.47, and those for social contact 0.48. (Details of these analyses are available from the authors.) Although these figures suggest that proxy scores were not a source of bias in the controls, we were unable to conduct a parallel analysis for the cases, and so can only marshal a partial defence against the criticism of proxy bias.

Of all possible, unmeasured confounding variables, the traditional physical risk factors: blood pressure, cholesterol and smoking, are most notable by their absence. These could not be included as covariates since reliable measures of the first two were not available for the CD cases. We have also defended their absence from this study elsewhere (Spicer, Jackson and Scragg, 1993). The main argument is that controlling these covariates typically makes little difference to the estimates of psychosocial

effects since the two sets of risk factors tend to be unrelated (Booth-Kewley and Friedman, 1987; House, Landis and Umberson, 1988; Ragland and Brand, 1988a). Certainly, within the control sample we have found no correlations between self or proxy reported psychological variables and physical risk factors.

As noted above, all of the preceding arguments are circumstantial and certainly do not provide grounds for claiming that the results are uncontaminated by threats to validity. Retrospective, proxy data are problematic, and require validation in other more powerful designs. Meanwhile we believe that the above arguments, and the similarities of our results to others', give us a limited licence for speculating as to what the results might mean.

The association we found between low social contact and CD risk replicates that reported in a number of prospective studies, not only in general, but also in two particular respects. The magnitude of the effect is in the middle of the range reported in House, Landis and Umberson's (1988) review. Moreover, the location of risk specifically in the socially isolated group echoes the findings of several studies they discuss. It is also worth reiterating that we have found no association between social contact and MI in men (Spicer, Jackson and Scragg, 1993), again following the reported pattern of a reliable link between social isolation and coronary mortality but not morbidity.

Our TAB results give further weight to the possibility that, whereas Type A behaviour increases risk of non-fatal MI, Type B behaviour is associated with coronary death. Interpretation of relevant findings is made doubly difficult by the use of various TAB measures which are moderately correlated at best; and by the definition of Type B behaviour purely in terms of the absence of Type A characteristics. For these reasons, it is helpful to explore the results at the item level as well as in terms of A/B classifications.

All but one of the FTAS items which differentiated cases from controls indicated an over-representation of Type Bs in the cases. The exception was the item which described respondents as " stretched by work to the very limits of energy and capacity at the end of an average day of work". Those who admitted this description, and thereby increased their Type A score, were more likely to be cases. The item bears a striking resemblance to one from the Reeder stress scale: "I am exhausted mentally and physically at the end of the day". This is of interest because the Reeder item has been shown to predict coronary death in the Kaunas-Rotterdam Intervention Study: a finding which has since been replicated using more extensive assessment of what Appels (1992) calls vital exhaustion. Thus, regardless of whether or not this item belongs in a TAB measure, we appear to have found supporting evidence for Appels' contention that vital exhaustion is a risk factor for CD.

It might be argued that the association between Type B behaviour and CD is an artifact somehow produced by the presence of the vital exhaustion item in the FTAS. However, the scoring procedure means that the item would dilute the Type B association. The question remains then, why should CD risk be higher in men who are seen as less likely to be pressed for time, to be dominating and to feel the need to excel, when the opposite obtains for MI risk?

It could be argued that the higher mortality of Type Bs was due to the absence of help at the onset of the coronary event. However, the relationship between the behaviour pattern and case-control status was independent of that for social contact, and there was no interaction effect. That said, the social contact measure was no more

than a crude index of complex social processes, so this possibility cannot be entirely discounted. A further possibility is that Type Bs, regardless of their social context, were slower than Type As to seek medical help. No information on this process was gathered in the present study. The proposal does conflict though with data which suggest that Type Bs are more likely than Type As to report symptoms of physical distress (Weidner and Matthews, 1978).

In interpreting their similar pattern of results, Ragland and Brand (1988a) consider the hypothesis that Type As may be protected by more effective psychological coping strategies, but find little supporting evidence. In contrast, Sparacino (1979) notes striking similarities between Type A characteristics and those of elderly survivors in general. These characteristics include being aggressive, demanding, taking control of one's life and engaging in multiple, highly organised activities. He discusses this evidence in the context of his general hypothesis that the health impact of TAB may reverse in old age. We could not test this hypothesis since our oldest participants were only aged 64. However, it is possible that Type A coping strategies provide protection against coronary death at any age. Thus, whereas Type As appear to increase their risk of non-fatal CHD, their capacity to cope with mental and physical adversity may increase their chance of survival. In contrast, although Type Bs may be less likely to develop CHD, they may cope less well with the crisis of a coronary event and be less likely to survive it.

Dienstbier (1989) has recently drawn attention to the "toughening" potential of a history of stress-induced arousal, which may provide a physiological substrate for Type A survival strategies. It is possible that the distinctive psychological stress histories of Type As, and their equally distinctive physiological reactions, may subsequently provide a degree of protection against coronary death. Dienstbier does provide some discussion of Type A behaviour, but does not make this particular suggestion. Clearly it is a highly speculative proposal, and one which contains many assumptions which require testing, especially the paradoxical notion that cumulative physiological processes may simultaneously increase MI risk, but decrease CD risk. Nevertheless, the idea of physiological toughness does provide a starting point from which an explanation of the difference in Type A risk of fatal and non-fatal CHD might be developed.

As we noted at the outset, there is little information available on the association between TAB and coronary death, both in absolute terms and relative to the evidence on non-fatal CHD. More studies are needed which avoid the methodological problems we have discussed above. Meanwhile, the trend in the limited evidence is sufficiently striking for us to note the advice of Ragland and Brand (1988a), who suggest the need for care in distinguishing CHD endpoints in TAB research, and in considering the implications of post-MI modification of Type A characteristics.

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