Evaluation of Type A personality

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Summary: When we look for the criteria for causation of Type A behaviour for coronary heart disease, we lack the reproducibility, the predictability to a certain extent, a pathogenic mechanism and an animal model, all four of the eight recognized criteria for causal inference. The debate therefore is still wide open.

Techniques of behaviour pattern classification

The first method used to determine Type A behaviour was a structured interview (SI), and sound tapes used for training interviewers (Friedman & Rosenman, 1959). Today video tapes and diagnostic indicators of Type A behaviour which show a time urgency and hostility score are also used (Friedman & Powell, 1984). The original personality score used was divided into a full blown Type A (Type A1), a largely Type A (A2), a full blown Type B (B4) and a largely Type B (B3) with an intermediate type (AB or X). Two other behavioural scores have been developed. One is widely used is the Jenkins Activity Survey (JAS) (Jenkins et al., 1967), which is a 61 item questionnaire; and the Bortner Scale composed of 14 items (Bortner, 1969). Both these methods fail to include the total psychomotor behavioural dimension which includes the voice. Nevertheless they were both found to agree satisfactorily with the SI as used in the original collaborative group study and in our own Belgian Heart Disease Prevention Project (Kittel, 1984).

Relationship of Type A behaviour pattern to coronary risk factors

As both ourselves (Kittel et al., 1982) and others have found, there was no relationship between Type A and the classical risk factors such as age, cholesterol, blood pressure, smoking habits, body mass index and the coronary risk profile, at least when social class was taken into account.

Some other coronary risk factors have been less studied in relation to Type A behaviour and merit attention. Physical fitness, defined as the workload reached on a bicycle ergometer for a heart rate of 150 beats per minute showed no relationship with Type A using the JAS. Leisure time activity was found to have a direct relationship with Type A behaviour; the more activity the more intense the Type A behaviour. The same was found for heavy leisure time activity. Thus if physical activity conveys a relative protection against coronary heart disease (CHD), Type A would be more protective than Type B (Kittel et al., 1983). With alcohol consumption, however, heavy drinkers are more Type A both amongst blue and white collar middle aged males (Kornitzer, 1985).

We have also looked at the relationship between Type A and stress, more precisely job stress (Kornitzer & Kittel, 1986). In our prospective physical fitness study Type A assessed by the JAS was strongly correlated with stress at work, even after controlling for socio-professional class and study level (Kittel et al., 1983).

Comparison of Type A studies

Table I summarizes all the studies of Type A behaviour to date. Four show Type A to be a significant independent predictor for CHD, namely the Western Collaborative Group Study (Rosenman et al., 1975); the Framingham Study (Haynes et al., 1980); the French-Belgian Pooling Project (Kornitzer & Lellouch, 1984) and the control group of the Belgian Heart Disease Prevention Project (Kittel, 1984).

In the control group of the Belgian Heart Disease Prevention Project no hard CHD events occurred in B4 subjects while the highest incidence of hard CHD events occurred in A1 subjects. When the events in Types B and AB were added and compared with events in Type A, the risk of CHD in the latter was doubled. Moreover in this sub-group a positive gradient of hard events appeared with increasing quartiles of Type A scores, both by JAS and Bortner techniques. In the intervention group, on the contrary, no relationship

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Table I Type ‘A’ behaviour and incidence of CHD

<table>
<thead>
<tr>
<th>First author</th>
<th>Sex</th>
<th>Age</th>
<th>Study</th>
<th>Technique</th>
<th>Place</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosenman (1975)</td>
<td>M</td>
<td>39–59</td>
<td>WCGS</td>
<td>SI</td>
<td>California</td>
<td>+</td>
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<td>Jenkins (1974)</td>
<td></td>
<td></td>
<td></td>
<td>JAS</td>
<td></td>
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<tr>
<td>Haynes (1980)</td>
<td>M &amp; F</td>
<td>45–64</td>
<td>Framingham</td>
<td>Framingham Scale</td>
<td>Massachusetts</td>
<td>+</td>
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<tr>
<td>Jouve (1980)</td>
<td>M</td>
<td>40–59</td>
<td>EDF</td>
<td>Bortner Scale</td>
<td>Marseilles</td>
<td>0</td>
</tr>
<tr>
<td>Reed (1982)</td>
<td>M</td>
<td>50–65</td>
<td>HHP</td>
<td>JAS</td>
<td>Honolulu</td>
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<tr>
<td>Shekelle (1983)</td>
<td>M</td>
<td>35–57</td>
<td>MRFIT</td>
<td>SI</td>
<td>USA</td>
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</tr>
<tr>
<td>Koskenuo (1983)</td>
<td>M</td>
<td>35–64</td>
<td>FTCS</td>
<td>Bortner Scale</td>
<td>Finland</td>
<td>0</td>
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<tr>
<td>Appels (1984)</td>
<td>M</td>
<td>45–59</td>
<td>KIRS</td>
<td>SI</td>
<td>Netherlands</td>
<td>0</td>
</tr>
<tr>
<td>Kittel (1984)</td>
<td>M</td>
<td>40–59</td>
<td>BHDPP (Intervention)</td>
<td>SI</td>
<td>Belgium</td>
<td>(+)</td>
</tr>
<tr>
<td>Kittel (1985)</td>
<td>M</td>
<td>40–55</td>
<td>PFS</td>
<td>JAS</td>
<td>Belgium</td>
<td>0</td>
</tr>
</tbody>
</table>

SI, Structured Interview; JAS, Jenkins Activity Survey; WCGS, Western Collaborative Group Study; EDF, Electricité de France Study; HHP, Honolulu Heart Project; MRFIT, Multiple Risk Factor Intervention Trial; FTCS, Finnish Twin Cohort Study; KIRS, Kaunas-Rotterdam Intervention Study; FBPP, French Belgian Pooling Project; BHDPP, Belgian Heart Disease Prevention Project; PFS, Physical Fitness Study.

was found between Type A by the questionnaire and the incidence of hard events. Multilogistic function analysis using the classical risk factors, socio-professional class and cultural background (Dutch/French) confirmed the presence of a predictive power of Type A in terms of CHD events in the control group and its absence in the intervention group.

However, seven other studies failed to show any independent prospective relationship between type A and CHD incidence (Table I). In the Physical Fitness Study, multivariate discriminant analysis showed that smoking and serum cholesterol are predictors of the incidence of CHD whereas age, body mass index, linguistic culture and Type A behaviour according to JAS are not (Kittel, 1985).

These contradictory results demonstrate that the one essential criterion for causality is missing, namely reproducibility. What is the cause of these differences? The specific features of the American culture of Type A cannot be invoked since negative results are observed in the Honolulu Heart Project (Reed & Cohen, 1982) and MRFIT (Shekelle et al., 1983) studies and positive ones in the French-Belgian Pooling Project (Kornitzer & Lellouch, 1984) and the Control Group of the Belgian Heart Disease Prevention Project (BHDPP) (Kittel et al., 1982). Social class cannot be the main factor, as in the intervention group of the BHDPP Type A was not a predictor even in the white collar group. The effect of intervention programmes modifying Type A and thereby interfering with the results cannot be an explanation since in a prospective study without intervention no predictive power of Type A was found. Finally, the methods of assessment of Type A could be incriminated, but in our control group of the BHDPP, Type A by the JAS was an even better predictor than by the SI.

Our present conviction is that the psycho-social coronary risk profile is multifactorial. Apart from Type A, other risk factors such as the hostility dimension and other protective factors such as social supports have to be taken into consideration.

Pathogenesis of relationship of Type A behaviour pattern and CHD

Finally, there is the problem of pathogenesis. Some studies, essentially those using the SI, have found a correlation between Type A behaviour and the degree of coronary narrowing on angiography (Franck et al., 1978). Others, like ourselves, have not observed this relationship (Kornitzer et al., 1982). Friedman et al. (1960) observed a relation of Type A behaviour with urinary catecholamine excretion comparing 10 Type A1 and 10 Type B4s. We compared 30 Type A2 and 30 Type B and observed no difference either in urinary catecholamine excretion or in arrhythmias on 24 hr Holter recording in the two groups (De Backer et al., 1979).
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References


