TYPE A Behavior

Edited by
Michael J. Strube

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Foreword

After some 20 years of intensive research, Type A behavior was designated a risk factor for coronary heart disease (CHD) in 1981 by a review panel sponsored by the National Heart, Lung and Blood Institute (NHLBI). The designation was based primarily on the prospective Western Collaborative Group Study, a reanalysis of the Framingham data, and evidence from coronary angiographic studies. Recent evidence has compromised the seeming unequivocality of these data. The Type A area is, in my judgment, an exemplar of research into biobehavioral mechanisms whereby social and psychological events culminate in physical disease, or at least potentiate disease. Nevertheless, the evidence in support of Type A as a risk factor remains sufficiently compelling, in my judgment, to suggest that something important is out there. Continuing efforts should, therefore, be expended on the study of Type A behavior and its component elements.

Over eight years have elapsed since the NHLBI designation and research on Type A has, indeed, continued unabated. Many of the papers in this volume typify the nature of this research and reflect directions being taken by contemporary investigators. Thus, we see efforts to (1) isolate the so-called "toxic" elements in the Type A behavior pattern such as cynical hostility and anger, (2) delineate gender differences and similarities in Type A behavior and its association with CHD, (3) determine the heritability, if any, of the behavior pattern and its principal components, (4) specify developmental antecedents of the behavior pattern, as well as the reliability of assessing the pattern in children, and (5) develop and evaluate programs aimed at altering Type A behavior.

Of greater importance, perhaps, are the studies based upon theoretical models of the behavior pattern. Such research is a refreshing breath of fresh air. These studies attempt to answer such fundamental questions as what gives rise to Type A behavior and what factors appear to sustain it. Little attention, unfortunately, has been given to conceptualizing Type A behavior over the past two decades. We now see a change that promises to provide an important foundation for understanding the psychological mechanisms underlying the behavior pattern.

Several papers in this volume are noteworthy in this regard, including Strube's self-appraisal model, my own control theory as extended and amplified in, for example, the work of Lawler and her colleagues, and Rhodewalt's conceptual approach to medical noncompliance. We also see in this volume some interesting efforts to look at the role of attentional style in Type A children, and I applaud the paper by Fontana et al. for its systematic approach to Scherwitz's self-involvement hy-

pothesis as an explanation for differences in the evidence suggesting greater physiological reactivity in As compared to Bs.

These research developments are most gratifying to someone who has repeatedly pointed to the relative absence of conceptual models and theoretically-based research in the Type A area. I would hope that this emphasis on conceptualization will be extended to the question of mediating physiological mechanisms. My own hunch is that catecholaminergic and related sympathetic nervous system activity are prime candidates for the mediator role.

I am also struck by the pervasiveness of research indicating a central role for the control variable in cardiovascular disease and, for that matter, in the pathogenesis of a variety of diseases. Taken together with accumulating data on the effects of uncontrollability on catecholamine changes, we may have the beginnings of important insights into the physiological mechanisms underlying the association between psychological variables and Type A behavior. This is, therefore, a nice illustration of exciting theory construction.

It is essential that this area develop coherent and empirically tested models that will stand up to the most rigorous scientific scrutiny. I believe progress is being made in this direction. Such theoretical development, coupled with a solid data base, will go a long way towards addressing the concerns of clinicians by providing them with a foundation for interventions aimed at altering the behavior pattern. In the final analysis, treatment and prevention procedures based upon systematic scientific knowledge are the best rationale for public health policy.

David C. Glass
State University of New York at Stony Brook

PREFACE

From its simple beginnings as a descriptive label characterizing individuals at apparent risk for coronary problems, the Type A pattern has matured into a theory-based variable with applications extending beyond the prediction of coronary endpoints. This diversity is reflected in current research which varies considerably in scope and level of analysis. For example, intense efforts are underway to refine the prediction of coronary disease and to specify the pathophysiological processes. Currently in vogue are attempts to dissemble the Type A pattern in the hopes of identifying the "toxic" subcomponents. Likewise, investigations with special populations (e.g., women, children) have attempted to identify the generality of risk conferred by the Type A pattern, and to identify its developmental antecedents. The Type A pattern also has been found to be useful in understanding noncoronary medical problems, and numerous extensions beyond the medical realm to ordinary social behavior exist. These latter extensions have been justified by recent attempts to understand the psychological underpinnings of Type A behavior. Theory development remains the most critical issue in the Type A area, although the recent emergence of compelling conceptual models augurs well for the future health of this research area. Only through sound theory will investigators be able to place the pattern in a broader, integrative context, embark on more programmatic efforts, and develop a common language for the many different levels of analysis.

This collection of articles brings together a representative cross-section of leading scholars and research programs devoted to the study of Type A behavior. The works attest to the vitality of the area, and the many advances that are being made. The research reported here spans levels of analysis from the physiological to the socio-historical, and covers topics that range widely across the medical and psychological terrains. The discourse varies from the solidly empirical to the conceptually speculative. In short, there is something here for everyone, and the sum total provides an exciting glimpse at the current state of affairs in Type A research.

Michael J Strube
Washington University, St. Louis, MO

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Ray H. Rosenman, M.D.
Director of Cardiovascular Research, Health Sciences Program
SRI International, Menlo Park, CA 94025

Dr. Strube, Guest Editor for this issue of the journal, has kindly asked me to write an overview-type of lead article and I accepted his flattering invitation. Some of the following is by way of review, and some is a personal overview.

Although the concept of risk factors for coronary heart disease (CHD) is well established, there are many reasons (Rosenman, 1986) to indicate that they only partially explain historical changes of CHD incidences, gender differences, geographical heterogeneity among population groups, or the varying rate of progression of coronary atherosclerosis. Moreover, it is not clear that interventions on these risk factors have significantly reduced either primary or secondary rates of CHD.

Considerable evidence (Rosenman, 1986) links the CHD incidence with urbanization, population densification, and industrialization. Thus, the reported association between CHD and risk factors are strongly mediated by the ambient situations in which individuals live and work, as well as by other psychosocial factors that distinguish one individual from another. Since the relationships of risk factors to CHD incidence are based on statistical findings, they do not provide absolute levels of risk or explain the individual specificity of associated risk. Pathogenicity for CHD is not given solely by the level of one or combination of risk factors, since it is the individual with the risk factor who suffers CHD, but at highly variable levels of such factors.

Development of Type A

Such considerations, along with direct observation of patients with CHD, led to the formulation of the Type A behavior pattern (TABP) and its component behaviors (Rosenman, 1986; Rosenman, Swan & Carmelli, 1988). Initial studies appeared to link TABP with the preva-

lence of CHD in both sexes (Friedman & Rosenman, 1959; Rosenman & Friedman, 1961). However, these studies were not epidemiologically sound and it was therefore believed necessary to do a carefully controlled, prospective study in order to test the hypothesis that TABP is associated with the incidence of CHD. This was done in the Western Collaborative Group Study (WCGS) (Rosenman, Friedman, Straus, et al., 1964). The large number of subjects and its design necessitated a new method for assessment of TABP. Self-report biases were found to make the use of questionnaires inadequate for this purpose and a structured interview (SI) was developed (Rosenman et al., 1964; Rosenman, 1978a).

The TABP was defined as an action-emotion complex involving behavioral dispositions such as ambitiousness, aggressiveness, competitiveness, and impatience; specific behaviors such as muscle tenseness, alertness, rapid and emphatic vocal stylistics, and accelerated pace of activities; and emotional responses such as irritation, hostility, and increased potential for anger. The SI was designed to allow observation of these behaviors during the interview, and to provide challenges to evoke their manifestations. The converse Type B behavior pattern was conceived as the relative absence of Type A behaviors in individuals who exhibit a different coping style that is characterized by a relative lack of time urgency, impatience, and hostile responses. TABP was not considered to be either a stressor situation or distressed response, and, therefore, not synonymous with stress, or to equate with anxiety, worry, fear, depression, or neurosis.

The WCGS provided strong evidence that TABP is associated with the incidence of CHD (Rosenman, Brand, Sholtz, & Friedman, 1976) and with the severity of coronary atherosclerosis (Friedman, Rosenman, Straus, et al., 1968). Jenkins, Rosenman, and Friedman (1968) found evidence for replicability of the SI-assessment of TABP, as well as for its stability over short periods of time. A search was made for possible biological mechanisms that might explain the CHD risk associated with TABP (Rosenman & Friedman, 1974). It was found that male subjects with well-defined TABP exhibited greater noradrenergic responses than did paired Type B subjects, both during a competitive, cognitive task (Friedman, Byers, Diamant, & Rosenman, 1975), and in their daily occupational milieus (Friedman, St. George, Byers, & Rosenman, 1960). Explorations were made for interventions on TABP, using both psychological (Rosenman & Friedman, 1977) and pharmacological methods (Rosenman, 1978b).

During the course of the WCGS, several psychologists became interested in TABP. Caffrey (1968) studied relationships of TABP with various measures of personality and behavior. Jenkins, Zyzanski, and

Rosenman (1979) developed a questionnaire for self-report assessment of TABP, the Jenkins Activity Survey (JAS), and Bortner and Rosenman (1967) developed a different type of scale, later shortened by Bortner (1969). The Framingham Type A Scale was developed much later (Haynes, Feinleib, & Kannel, 1980). A scale from the Adjective Checklist (ACL) and the Activity Scale of the Thurstone Temperament Schedule were found to correlate with TABP (Rosenman, Rahe, Borhani, & Feinleib, 1976) and this was subsequently confirmed (Rosenman, 1986).

The above only highlights some early history of TABP, described elsewhere in greater detail (Rosenman, 1986; Rosenman, Swan, & Carmelli, 1988). In the 15 years since these early studies, a large literature has appeared, dealing both with psychological implications of TABP as will as its associations with cardiovascular disorders.

Management Issues

Byrne, Rosenman, Schiller, and Chesney (1985), and others found that self-report questionnaires developed for TABP assessment appear largely to measure attitudes rather than the behaviors that are observed and assessd by the SI. These self-report scales fail to assess some of the most important elements of TABP and exhibit only weak correlations among themselves or with the SI. By including judgments of actual Type A behaviors observed during its administration, the SI extends the breadth and scope of assessments to fit the construct of TABP as a set of overt behaviors that occur in association with, and in response to, relevant situational stressors (Matthews, 1982; Byrne, et al. 1985). As reviewed elsewhere (Rosenman, 1986), various methods have been developed to assess TABP in adult females, college students, and in children (Matthews & Siegel, 1982). TABP also has been assessed from a videotaped, modified SI in order to provide quantization of its component behaviors (Friedman & Powell, 1984). However, this method has not been validated by other investigators, nor has its interrater agreement, test-retest reliability, or usefulness for prediction of CHD yet been demonstrated. Different methodologies also have been developed to assess certain components of TABP from the SI (Dembroski, MacDougall, Shields, et al., 1978; Hecker, Chesney, Black, & Frautschi, 1988).

Construct validation of the TABP concept proceeded along several lines. Under appropriate stimulus conditions, Type A subjects have generally been found (Rosenman et al, 1988) to be more likely than Type B counterparts to exhibit competitiveness, impatience, aggressiveness, irritation, and hostility. Regardless of the problems with self-report measures for TABP assessment, there is remarkable consistency with the TABP construct in the cluster of traits that are measured by such scales (Rosenman, 1986; Rosenman,et al., 1988). Moreover, this consistency in

the pattern of interrelationships among Type A behaviors and self-reports prevails in different samples that vary by age, sex, geographical distribution, and by different interviewers, raters, and sites of administration (Matthews, 1982; Matthews, Krantz, Dembroski, & MacDougall, 1982).

During exposure to a wide variety of physical and mental stressors in the laboratory setting, Type As also tend to exhibit greater autonomic neural and associated cardiovascular responses compared to Type B counterparts (Krantz & Manuck, 1984; Manuck & Krantz, 1986). Taken together, the results of a large number of studies (Rosenman, et al., 1986) lend strong construct validation for the TABP concept. These and other studies also have confirmed that TABP does not equate with anxiety, neuroticism, stress, or psychopathology. Moreover, recent studies (Kahn, Gully, Cooper, et al., 1987; Schneider, Julius, Moss, et al., 1987; de Quattro, 1988) confirmed earlier findings (Friedman et al., 1960, 1975) that Type A subjects exhibit enhanced noradrenergic responses in their daily milieus.

Type A and Heart Disease

The relationship of TABP to CHD and to severity of coronary artery disease has been assessed in a number of studies since the WCGS. Matthews and Haynes (1986) and Haynes and Matthews (1988) found inconsistent results in their elegant reviews. However, they point out that, on balance, population-cohort studies have found that TABP is indeed a risk factor for CHD, while intervention studies on high-risk persons are less consistent with regard to a relationship of TABP either to primary or recurrent rates of CHD. This is somewhat paradoxical since they noted that the relationship of TABP to CHD in the Framingham Heart Study was strongest when levels of other risk factors were elevated.

Considerable attention has been given to a recent finding in a 22-year follow-up of WCGS subjects accomplished from a mailed questionnaire and death certificate data. In this study, Ragland and Brand (1988) found that TABP did not indicate an adverse prognosis for CHD mortality among patients with CHD. These conclusions solely pertained to mortality from recurring CHD events during the 12 year follow-up experience after CHD already was manifest. During this time it is highly probable that major changes occurred in many risk factors, including TABP. The study findings were based on intake data and on a difference of 13 deaths between Type A and Type B patients over a 22 year period.

Booth-Kewley and H.S. Friedman (1987) used meta-analysis to organize findings in a superb review of the relationships between psychosocial variables and CHD. They found the strongest associations to be with TABP, particularly in cross-sectional studies. Haynes and Matthews (1988) noted that the later studies were performed during years in which

a decline of CHD mortality was occurring and during which there was widespread use of possibly protective beta-adrenergic blocking drugs for treatment of hypertension and post-infarction subjects. The negative Type A-CHD associations found in recurrent infarction studies, such as the Aspirin Myocardial Infarction Study (Shekelle, Gale, & Norusis, 1985) and that by Case, Heller, Case, & Moss (1985), are particularly open to question because both studies used the JAS for assessment of TABP, now clearly demonstrated to have major inaccuracies for this purpose (Matthews et al., 1982; Byrne et al., 1985). The study by Case et al. (1985) gave the questionnaire to subjects shortly after suffering acute myocardial infarction, obviously at a time when TABP might be least manifest. Moreover, the JAS notably fails to assess the hostility component of TABP (Abbott, Peters, & Vogel, 1988), which has important relevance for the association of TABP with CHD (Rosenman, 1985).

Haynes and Matthews (1988) considered other factors that might be related to reported inconsistencies, including small sample sizes, the distribution of Type A and B subjects in the samples, low incidence of CHD, as well as drift changes in the administration and assessment of the SI, when this was used. The importance of the latter is emphasized in a series of arduous studies by Scherwitz and Canick (1989). They carefully auditioned the audiotaped intake SIs from CHD cases and control subjects in the WCGS and MRFIT studies and compared interviewer styles, finding marked differences in this regard. It should be recalled that the interviewers in the WCGS had been trained over a long period of time and had ongoing regular follow-up throughout the intake period, while those in the MRFIT Study had only a brief period of training and almost no follow-up (Rosenman, 1986).

In more recent studies, Scherwitz and Brand (unpublished paper) found significant differences in the TABP-CHD risk ratios for various interviewers in the WCGS. These findings strongly suggest that the proficiency of SI interviewers may have confounding effects on the association of TABP with CHD.

Several other factors not considered in the cited reviews might be of even greater importance in the negative results of studies such as MTFIT (Shekelle, Billings, Neaton, et al., 1985). The first concerns the type of subject who is apt to volunteer for multifactorial intervention to prevent primary or recurrent CHD. Study populations are highly selected. Thus, Werko (1976) found that, in a city population, cardiovascular mortality is several times higher among those not answering an invitation for health examination than in participants, presumed to indicate the latter's interest in health problems. It is not likely that time-urgent, competitive, hostile, coronary-prone Type A males often volunteer for intervention studies in

which regular and relatively frequent clinic visits are required of participants, and which seek full compliance with major life-style changes of diet and other risk factors. It has further been shown that subjects who volunteer for such studies are often the "worried well," i.e. those with risk factors and no disease (Criqui, Austin, & Barrett-Connor, 1979). The markedly lower than predicted CHD incidence that occurred during follow-up in the MRFIT Study indeed suggests that the participants were biased by health consciousness and other factors that, despite high levels of classic risk factors, were associated with an unexpectedly low rate of CHD.

Another factor of major relevance concerns rates of intervention study dropouts. There is strong evidence that the CHD incidence may be much higher in subjects who drop out of a study compared to those who remain during many years of follow-up (Bruce, Frederick, Bruce, & Fisher, 1979). Almost half of the subjects in the Ontario Rehabilitation study dropped out during follow-up. The consistent predictors of dropout were smoking and blue collar occupation (Oldridge, Donner, Buck, et al., 1982). The MRFIT Study may exemplify these problems. Thus, the observed incidence of CHD during long follow-up in this large group of subjects at highest risk by reason of the three classical risk factors was markedly lower than that predicted by the Framingham Heart Study experience. Certainly the MRFIT experience confirms the point made by Werko (1976) that the results of the Framingham Heart Study are applicable only to those who took part in it, and cannot be used as representative of other populations in the U.S.A. or elsewhere.

The same factors doubtless relate to inconsistencies in studies of the relationship of TABP to angiographic severity of coronary atherosclerosis (Booth-Kewley & Friedman, 1987; Haynes & Matthews, 1988). Pickering (1985) also reviewed such studies and pointed out that they differed in methodologies, lacked normal control of subjects, showed poor correlation of coronary artery disease with the risk factors, often had small sample sizes, and lacked appropriate endpoints. The role of selection bias in angiography studies is well shown by the finding that those suspected of having CAD not confirmed by angiography have a higher prevalence of risk factors than do subjects without suspected CAD (Pearson, Gordis, Achuff, et al., 1982).

Some confusion about an association of TABP with severity of coronary artery disease (CAD) was introduced by an emphasis on studies with negative findings (Williams, Barefoot, & Shekelle, 1985). However, using the *SI* method of TABP assessment, Blumenthal, Williams, Konig, et al. (1978) and Williams, Haney, Lee, et al. (1980) had observed positive relationships. In their recent analysis of 2,289 patients referred by diagnostic angiography, they again confirmed this relationship

(Williams, Barefoot, Haney, et al., 1988), however, finding it to be stronger in relatively younger subjects. They emphasized that prospective studies generally found that most risk factors exhibit a greatly diminished predictive relationship for both severity of CAD and incidence of CHD with increasing age. Finally, in their reviews of prior studies of the association of TABP with CAD, they point out that those with negative findings had small sample size, failed to consider an age-interaction, and had used self-report questionnaires for assessment of TABP.

Other Data

Aside from relationships to severity of CAD and incidence of CHD, a large literature has developed on other aspects of TABP. Its moderate stability over time has been confirmed (Carmelli, Rosenman, & Chesney, 1987; Abbott et al., 1988; Rosenman et al., 1988). Global TABP does not appear to exhibit significant heritability, although some of its components may have small heritable aspects (Rosenman et al., 1976; Carmelli, Rosenman, Chesney, et al., 1987). The concept of TABP appears to be valid in children (Matthews & Woodall, 1988). It particularly emerges when relevant milieu conditions elicit Type A behaviors in susceptible persons, implying an important role of sociological and socioeconomic factors in an interaction with a subject's personality (Rosenman, 1986). It is therefore not surprising that childhood antecedents of TABP are mainly ascribed to parental and environmental influences that engender learned behaviors (Matthews & Siegel, 1982; Matthews & Woodall, 1988). However, gender and individual differences in active and passive behaviors are apparent even in infancy. This suggests that there are genetic predispositions for TABP that may reside in the distribution and density of hypothalamic nuclei and in other unknown factors that are genetically determined (Rosenman, 1985), but to which little attention has been given.

Self-referencing in the SI was found to be related to TABP, anger, hostility, blood pressure reactivity, severity of CAD, and the primary and secondary rates of CHD in some, if not all studies (Scherwitz, Graham, Grandits, et al., 1986), it was not similarly found by these researchers in the WCGS (Graham, Scherwitz, & Brand, unpublished parer).

Interventions

Levenkron and Moore (1988) recently reviewed issues associated with interventions on TABP. Pharmacological intervention with beta-adrenergic-blocking agents (Rosenman, 1978b, 1983) has been pursued by Schmieder, Friedrich, Neus, et al., (1985) and Krantz, Contrada, La Riccia, et al. (1987). Cognitive methodologies have been developed (Rosenman & Friedman, 1977; Roskies, 1987; Price, 1988), but the