CHAPTER 11

CURRENT ISSUES IN TYPE A BEHAVIOR, CORONARY PRONESS, AND CORONARY HEART DISEASE

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It has been almost 30 years since cardiologists Meyer Friedman and Ray Rosenman first introduced their description of the Type A behavior pattern (TABP; Friedman & Rosenman, 1959). Based on observations gleaned from their medical practice, Friedman and Rosenman suggested that the tendency to exhibit ambition, time urgency, extreme competitiveness, easily evoked hostility, explosive vocal stylistics, and rapid motric mannerisms placed one at risk for premature coronary heart disease (CHD). Specifically, they defined the TABP as "an action emotion complex that can be observed in any person aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and if required to do so, against the opposing efforts of other things and other persons" (Friedman & Rosenman, 1974, p. 84). According to Friedman and Rosenman, the Type A action-emotion complex is best thought of as a person-environment interaction in that the behaviors are observed only among predisposed persons (i.e., Type A) in challenging environments.

In the ensuing three decades, the TABP has been the focal point of a massive research effort to understand biobehaviorally mediated risk for CHD. In general, this research has contributed much to our broadened understanding of biopsychosocial models of personality and disease. However, enthusiasm for the TABP has waxed and waned with the accumulation of additional epidemiological evidence. As of 1981, the available evidence led the American Heart Association to conclude that the TABP was an independent risk factor for CHD (Review Panel, 1981). Since that time there have been several failures to replicate the earlier Type A–CHD association (e.g., Case, Heller, Case, Moss, & Multicenter Postinfarction Research Group, 1985; Shekelle, Gale, & Norusis, 1985; Shekelle, Hulley, et al., 1985). Recent reviews, both qualitative (Haynes & Matthews, 1988; Matthews & Haynes, 1986) and quantitative (Booth-Kewley & Friedman, 1987; Matthews, 1988), support the conclusion that Type A behavior as indexed by the Structured Interview (SI; Rosenman, 1978) is a reliable predictor of CHD, but that Type A behavior as measured by the Jenkins Activity Survey (JAS; Jenkins, Zyzanski,
& Rosenman, 1971), the most frequently used self-report measure of Type A, is not. Moreover, this conclusion is qualified by the fact that the SI does not predict CHD risk in populations with elevated levels of CHD risk factors (i.e., plasma cholesterol, hypertension, smoking) or previous myocardial infarction (MI; Matthews, 1988).

The above findings have led some to suggest that Type A behavior should be abandoned in favor of a search for the more “toxic” components of coronary-prone behavior such as hostility (Dembrofski & Costa, 1988). However, acceptance of this recommendation should be tempered, perhaps, by recent evidence indicating that treatments intended to modify global Type A behavior lead to significant reductions in recurrent Mls among CHD patients (Friedman et al., 1986). Although the Type A modification findings suggest that some sort of TABP-CHD link is still viable, the recent spate of negative epidemiological findings raise fundamental questions about Friedman and Rosenman’s original Type A hypothesis and have forced researchers to reconsider and revise their views of behavioral risk for CHD.

The present chapter will survey the central issues facing investigators in the Type A area. Research on TABP is far too vast to cover thoroughly in one chapter. Thus, we will briefly summarize each area and direct the reader to the most up-to-date reviews on the topic. We begin by discussing issues around assessment of TABP, the potential linkages of behavior to CHD, and the related issue of the search for the toxic behavioral element. After this survey we will turn to the main topic of the chapter. Because Type A intervention appears to hold promise in the treatment of CHD, we will focus at some length on psychological processes and motives thought to underlie Type A behavior and relate these models to attempts at Type A modification. The chapter concludes with a discussion of the lessons provided by research on Type A and CHD for general models of personality and disease.

ASSESSMENT OF TYPE A AND CORONARY PRONENESS

The reliable and valid assessment of the TABP has been a major obstacle limiting the study of individual differences in behavioral risk for CHD. At present, the failure to obtain consistent patterns of association between the various measures of the TABP and physiological mediators and clinical endpoints of CHD indicates that the measurement issue is far from resolved. Three Type A assessment devices, the SI, the JAS, and the Framingham Type A Scale (FTAS; Haynes, Feinleib, & Kannel, 1980) have received the preponderance of research attention and are considered here.

The Structured Interview

Believing that Type A behavior lacks insight into their behavior, Friedman and Rosenman developed the SI to assess the behavior pattern (Rosenman, 1978). The SI is an orally administered interview in which respondents are asked a series of questions about their style of responding to situations that elicit impatience, competitiveness, and hostility. The questions are asked in such a manner as to elicit speech characteristics and behaviors thought to be indicative of Type A. In scoring the SI, particular attention is given to speech stylistics, behavioral mannerisms, and, to a lesser extent, self-reports of behavior. The scoring scheme results in individuals being categorized as either A1 (fully developed Type A), A2 (incompletely developed Type A), X (a combination of Type A and Type B characteristics), B3 (incompletely developed Type B), or B4 (fully developed Type B).

As noted previously, the SI appears to be the measure of TABP most clearly related to the development of CHD in population-based studies (Haynes & Matthews, 1988; Matthews, 1988; Matthews & Haynes, 1986). The SI is also the most reliable Type A predictor of cardiovascular reactivity, a presumed mediator of the association between behavior and CHD (Houston, 1988). In contrast, the findings have been mixed in studies examining coronary artery disease (CAD) via angiography. While there are approximately a half dozen investigations finding a positive association, roughly as many studies find no relationship between SI Type A and extent of artery disease (i.e., Matthews, 1988). These contradictory findings may reflect limitations of cross-sectional angiographic studies rather than the invalidity of the SI (Haynes & Matthews, 1988; Matthews, 1988).

Nonetheless, there are several reasons for concern about the reliability and validity of the SI. Although the SI was initially thought to possess acceptable reliability (Dembrofski, 1978), Schwitz (1989) has questioned this assumption with evidence that SI administration procedures have evolved over the years such that earlier tests of
reliability and validity may no longer be meaningful. This contention is supported by the fact that although SI Type A was normally distributed in the Western Collaborative Group Study (WCGS; Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975), the population on which it was first validated, the proportion of Type A is higher in many of the more recent epidemiological studies. This suggests that the proportion of Type A in the population is increasing, changes in the administration of SI over time have artifactualy produced increased estimates of the prevalence of Type A, or some other selection factor such as differential referral of Type As and Type Bs leads to overrepresentation of Type As in research populations. Any of these possibilities make epidemiological data difficult to interpret.

Interpretive ambiguities are all the more a problem in studies of at risk populations. The high proportion of Type As may restrict the range of scores and, thus, the study's sensitivity to detect relationships between TABP and CHD endpoints. Or, Type As' and Bs' admission into any study may be based on different decisions that are, in turn, differentially related to CHD. For example, Type As and Bs might differ in the criterion used for referral into the studies and thus be evaluated at systematically different stages in the disease process (Matthews & Haynes, 1986). In brief, the shifting proportions of Type As in different epidemiological investigations raise concerns about the SI as a measure of the TABP and the resulting ability to estimate TABP-CHD relationships accurately.

A recently recognized concern with the SI is that the manner in which it is administered appears to be a critical determinant of its predictive validity. Scherwitz (1988) reanalyzed the Multiple Risk Factor Intervention Trial data (MRFIT; Shekelle, Hulley et al., 1985) that initially found no relationship between SI Type A and CHD mortality. In his reanalysis, Scherwitz compared Type A assessments made by interviewers who employed a more relaxed, slower paced interview style with those who used a challenging, faster paced interview style. Type A classifications generated by the former were more predictive of CHD than classifications generated by the latter (Scherwitz, Graham, Grandits, & Billings, 1987).

Finally, the SI may not measure the global Type A versus B ratings from the SI do not reflect several characteristics contained in the definition of Type A behavior. As Scherwitz (1989) noted, the validity of the SI as an assessment tool rests on the conceptual clarity of the hypothesized underlying construct. Thus, as psychometric development of TABP assessment instruments continues, greater attention should be paid to the psychosocial and behavioral processes hypothesized to underlie CHD risk. This important trend is already apparent in discussions of assessments of hostility (Costa, McCrae, & Dembroski, 1989; Siegman, 1989).

The Jenkins Activity Survey

The JAS was developed midway through the WCGS as a self-report measure of the TABP (Jenkins et al., 1971). The JAS (Form C) contains 52 questions similar to the SI. A discriminant function analysis was conducted using JAS item responses to predict SI classifications in the WCGS. The Type A subscale of the JAS is comprised of 21 items and is scored using optimal discriminant function weights. However, given that they are intended to be alternative assessments of a single construct, concordance rates between the JAS and simple SI A-B categories are in the alarmingly low 60% to 70% range (chance being 50% [MacDougall, Dembroski, & Musante, 1979; Matthews, Krantz, Dembroski, & MacDougall, 1982]). Thus, it appears that the SI and JAS are measuring different things. This observation is borne out by epidemiological and laboratory findings. With the notable exception of the WCGS, the JAS has not been reliably associated with CHD or angiographic findings (Matthews & Haynes, 1986). Further, the JAS is not related to cardiovascular reactivity as consistently as the SI (Houston, 1988). At the same time, however, the JAS has been found to be related to many behavioral and cognitive features of earlier definitions of the TABP (Glass, 1977; Matthews, 1982).

It is noteworthy that many of the epidemiological investigations that produce null findings with the JAS are troubled by methodological and interpretive flaws. In addition, it is likely that because the JAS scoring procedure was derived to predict SI classifications and not CHD outcomes, it has obscured the ability of the JAS to predict CHD. That is, the SI is a significant but imperfect correlate of CHD and the JAS is an imperfect correlate of the SI. Nonetheless, while a reexamination of
the JAS’s predictive validity might be warranted, at this point the JAS should not be considered a valid measure of CHD risk (Dembroski & Costa, 1988).

The Framingham Type A Scale

The FTAS is a 10-item scale derived from a 300-item questionnaire administered in the Framingham Heart Study (Haynes & Feinleib, 1982). The predictive validity of the FTAS is largely limited to the Framingham Heart Study in which it predicted CHD endpoints associated with angina. For example, the FTAS did not predict MI in the absence of angina. This unique pattern of CHD endpoints, when considered with information about the construct validity of the scale, raises important questions about the interpretation of the FTAS as a measure of the TABP. The FTAS is unique among TABP assessments in that it is associated with neuroticism and somatic complaints in healthy samples (r = .26 to .42 [Smith, Houston, & Zurawski, 1983; Smith & O’Keefe, 1985; Smith, O’Keefe, & Allred, 1989]). Measures of neuroticism also predict development of angina but not MI (French-Belgian Collaborative Group, Wilhelmson, Wedel, & Pennert, 1982; Hagman, 1987; Medalie & Goldbourt, 1976; Otsfeld, Lebovitis, Shekelle, & Paul, 1964). Further, neuroticism is associated with persistent angina-like chest pain complaints in the absence of underlying CAD (Bass & Wade, 1984; Costa, Fleg, McCrae, & Lakatta, 1982; Roll & Theorell, 1984; Schocken, Greene, Worden, Harrison, & Spielberger, 1987; Wielgosz, Fletcher, McCants, McKinnis, Haney, & Williams, 1984). Thus, it is possible that the results from the Framingham study reflect an association between neuroticism and the tendency to complain of anginalike chest pain, rather than an association between TABP and CHD (Costa, 1986; Smith et al., 1989). At the very least, it is clear that the FTAS assesses a different dimension than that tapped by either the JAS or SI.

Current Developments in the Assessment of Coronary Proneness

Given the apparent difficulties with global Type A as a predictor of CHD, researchers have turned their attention to the components of TABP in an attempt to identify the toxic element of the behavior pattern. This strategy acknowledges that TABP is a multidimensional individual difference and that, perhaps, only some components are associated with risk for CHD (see Chesney, Hecker, and Black, 1988, and Dembroski & Czanjkowski, 1989, for reviews). In the first such attempt, Matthews, Glass, Rosenman, and Bortner (1977) compared new CHD cases to disease-free, matched control participants from the WCGS on a variety of characteristics derived from the SI. Among the characteristics that discriminated cases from controls were the potential for hostility, outward expression of anger, competitiveness, the frequent experience of anger, vigorous response style, and explosiveness of speech. In another reanalysis of the WCGS data, Chesney, Hecker, and Black (1988) rated 12 separate components of the original global TABP. Hostility was the strongest of five significant predictors of CHD in univariate analyses and the only significant predictor in multivariate analyses considering all TABP characteristics simultaneously.

Dembroski (1978) has developed a coding system for rescoring the SI for what he terms potential for hostility. The scoring protocol considers separately (a) self-reports of displays of anger, irritation, annoyance, and resentment; (b) the intensity of response; and (c) the degree of antagonism in the interview interaction. Dembroski and his colleagues have demonstrated positive associations between overall potential for hostility or its elements and both CAD and CHD, even in reanalyses of SIs from studies in which SI-global Type A failed to predict such relationships (Dembroski & Costa, 1987; Dembroski & Czanjkowski, 1989).

Independent evidence for the nomination of hostility as the toxic element in behavioral risk for CHD comes from studies employing the Cook-Medley Hostility (Ho) Scale (1954), a self-report measure of hostility derived from the MMPI. Not only does the Cook-Medley predict CAD (Williams, Haney, Lee, Kong, Blumenthal, & Whalen, 1980) and CHD (Barefoot, Dahlstrom, & Williams, 1983), but it appears to predict mortality from all causes (Barefoot, Dodge, Dahlstrom, & Williams, 1989; Shekelle, Gale, Ostfeld, & Paul, 1983). Three failures to replicate this association between Ho scores and CHD risk have been reported (Hearn, Murray, & Luepke, 1988; Leon, Finn, Murray, & Bailey, 1988; McCranie, Watkins, Brandsima, & Sisson, 1986). However, methodological factors may account for some of the inconsistency (Williams, 1987), and a similar self-report scale has been found to predict mortality in two other prospective studies (Barefoot, Siegler,
Nowlin, Peterson, Haney, & Williams, 1987; Ostfeld et al., 1964). Although the Ho scale has been found to be related to self-reported and behaviorally rated overt hostility (Barefoot et al., 1989; Smith & Frohm, 1985; Smith, Sanders, & Alexander, 1988), it is more closely associated with measures of anger proneness, resentment, mistrust, and suspicion (Smith & Frohm, 1985; Smith et al., 1989; Pope, Smith, & Rhodewalt, 1989).

The Cook-Medley scale and Potential for Hostility are correlated (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1983), but the former primarily reflects covert or experiential aspects of hostility while the latter primarily reflects the overt expression of hostility. Recently, Siegman (1989) argued that these two aspects of hostility may not be equally related to CHD. At least some evidence suggests that more overt, experiential aspects of hostility are better predictors of CAD severity in angiography populations. However, both forms are conceivably linked to CHD. If, as Dembroski and Czanjkowski (1989) suggest, it is the overt expression of hostility and its attendant interpersonal difficulties that increase the risk for CHD, then potential for hostility is, in all likelihood, assessing a toxic aspect of hostility. However, it is also plausible that mistrust and resentment of others leads one to be chronically hypervigilant, a cardiologically taxing condition (Williams, 1989). In addition, the individual who harbors negative beliefs and expectancies about others also should experience a higher frequency of conflictive interactions with an associated increase in physiological arousal (Smith & Pope, 1990).

Work is also in progress that examines the relation of more general dimensions of personality to CHD outcomes. For example, Costa et al. (1989) argue that hostility toward others can be viewed as an aspect of the more basic personality dimension of agreeableness-antagonism. According to their reasoning, it is chronically irritable, impatient, antagonistic individuals who experience repeated episodes of autonomic arousal and, consequently, are at risk for CHD. This research is preliminary but offers promise for behavioral risk for CHD from a broad individual difference perspective.

In sum, 30 years of research indicates that attributes of certain individuals place them at risk for CHD. However, at present it is not clear precisely which characteristic or set of characteristics is the causal agent. The SI is capturing some attribute, perhaps a potential for hostility or even antagonism, that predicts CHD. It is equally clear that the JAS does not reliably predict CHD. Yet certain aspects of JAS Type A would appear to reflect stress-exacerbating appraisal processes and stressful coping responses. Perhaps, as we note elsewhere (Smith, 1989; Smith & Anderson, 1986; Smith & Rhodewalt, 1986), the JAS may indirectly increase CHD risk by its association with the tendency to create stressful environments. Perhaps it is JAS in combination with an additional individual difference (i.e., a hyperreactive cardiovascular system [Musante, MacDougall, Dembroski, & Van Horen, 1983]) or hypervigilance (Rhodewalt & O'Keeffe, 1986; Williams, 1979) that synergistically elevates CHD risk. In this regard, we endorse Costa, McCrae, and Dembroski's (1989) suggestion that the search for coronary-prone behavior might do well to move beyond the Type A construct.

**MEDIATING LINK BETWEEN TYPE A BEHAVIOR AND CORONARY HEART DISEASE**

**The TABP → Reactivity Hypothesis**

The clinical manifestations of CHD result from coronary artery disease (CAD), the thickening of the walls of the coronary arteries known as atherosclerosis. CAD leads to reduced or blocked blood supply to the heart, resulting in angina pectoris (chest pain stemming from insufficient blood flow to the heart), myocardial infarction (death of heart muscle tissue), and sudden coronary death.

A central issue in research on the TABP has been specification of the mechanisms through which behavior contributes to CHD. The presumed culprits are thought to be Type As' excessive cardiovascular and neuroendocrine responses to appropriately challenging or stressful situations. It is the magnitude of change from baseline, or reactivity, and not resting or chronic differences between Type As and Bs that is thought to accelerate CAD. Accumulating evidence indicates that activity of the sympathetic-adrenergic-vascular system and its concomitant increases in blood pressure, heart rate, myocardial oxygen demand, and elevations in circulating epinephrine, norepinephrine, and free fatty acids hastens CAD and probably precipitates acute CHD events (see Krantz, Glass, Schaeffer, & Davia, 1982, for a review), although this conclusion is still controver-
sial (Krantz & Manuck, 1984). There is also evidence, albeit sparse, that Type As, compared with Type Bs, display greater pituitary-adrenocortical reactivity, another mechanism implicated in the etiology of CHD (Williams, Lane, Kuhn, Melosh, White, & Schanberg, 1982; Henry, 1983).

Laboratory evidence for the Type A-reactivity hypothesis is fairly consistent for SI-defined Type A but less so for JAS-defined Type A (see Contrada & Krantz, 1988; Houston, 1988; and Wright, Contrada, & Glass, 1985, for reviews). While not differing in resting levels, Type As respond to environmental challenge, social competition, and threats to self-esteem with larger increases in blood pressure than do Type Bs (Dembroski, MacDougall, Herd, & Shields, 1979; Glass et al., 1980; Pittner & Houston, 1980; van Schijndel, De Mey, & Naring, 1984). For example, Glass et al. (1980) demonstrated both elevated cardiovascular (systolic blood pressure, [SBP]) and neuroendocrine (plasma epinephrine) reactivity in Type As who were playing a competitive game while being harassed by their opponent compared with harassed Type Bs and nonharassed Type As and Bs.

In sum, researchers concluded that Type As are most physiologically reactive in situations that elicit active, effortful coping or anger arousal (Contrada & Krantz, 1988). This conclusion is complemented by Obrists' (1981) work that indicates that active coping is accompanied by the greatest sympathetically mediated cardiovascular reactivity. It is noteworthy that Type A-B differences in reactivity are most pronounced in situations that are moderately as opposed to extremely challenging, difficult, or of moderate incentive value. Type As and Bs appear to be equally reactive to highly challenging or stressful situations. It is also of interest that Type As experience heightened cardiovascular arousal in anticipation of difficult or challenging tasks (Gastorf, 1981).

An important test of the Type A-challenge-reactivity-CHD hypothesis would be provided by examinations of Type A reactivity in naturalistic settings. Unfortunately, few such tests have been conducted and those that do exist have produced somewhat inconsistent findings (Contrada & Krantz, 1988). Interpretive problems with these field studies have been compounded by their failure to translate models of Type A-reactivity into meaningful experimental designs (Matthews, 1982; Rhodewalt, Hayes, Chemers, & Wysocki, 1984; Smith & Rhodewalt, 1986; Smith, 1989). Although conceptual models of the TABP empha-

size the interaction between the individual difference dimension and appropriate situational variables such as challenge or demand, many investigations simply report the presence or absence of Type A-B main effects on cardiovascular or biochemical responses. Epidemiological studies linking TABP to CHD endpoints are clear examples of this “main effect” approach to TABP.

A faithful translation of the laboratory TABP by challenge interactions on reactivity would involve assessment of level of challenge in the field, TABP, and degree of reactivity. Studies meeting these requirements do not exist (Ganster, Syme, & Mayes, 1989); however, there is evidence that such an interactive approach might be useful. For example, studies do show that Type As respond to high levels of perceived job stress with greater reports of psychological and physical symptoms than do Type Bs (Rhodewalt et al., 1984). Perceptions of challenge in the workplace (i.e., reduced control [Rhodewalt, Sansone, Hill, Chemers, & Wysocki, 1988], role ambiguity [Howard, Cunningham, Rechnitzer, 1986], or low autonomy and peer cohesiveness [Chesney, Sevelius, Black, Ward, Swan, & Rosenman, 1981]) lead to reports of higher job stress (Rhodewalt, Strube, Hill, & Sansone, 1988) and job dissatisfaction (Howard et al., 1986) in Type As than in Type Bs. Moreover, perceived job demand by TABP interactions have been found on cardiovascular outcomes such as systolic and diastolic blood pressure (Chesney, Eagleston, & Rosenman, 1981; Howard et al., 1986).

The studies described above are quite limited in the conclusions they permit. Studies are needed that examine Type A-B differences in cardiovascular reactivity to objective job demands. Taken together, however, the research described above suggests that such field studies would further our understanding of behavioral risk for CHD.

The Reactivity→TABP Hypothesis

An alternative approach to the Type A-reactivity relationship has been proposed by Krantz and his colleagues (Contrada & Krantz, 1988; Krantz, Arabian, Davia, & Parker, 1982; Krantz & Durel, 1983). This model is based on findings from patients undergoing coronary artery bypass surgery. Compared with Type B patients, Type A patients exhibit elevated SBP while under general anesthesia. Additionally, beta-adrenergic blocking agents, medications that dampen cardiovascular
reactivity, also mute Type A speech stylistics and potential for hostility (Krantz, Durel, Davia, Shafer, Arabian, Dembroski, & MacDougall, 1982). Krantz argues that Type A behavior may be a correlate of a biological predisposition to exaggerated cardiovascular reactivity rather than its cause. Thus, Type A learning to become behaviorally reactive to challenge and demand, in part because they experience exaggerated physiological arousal to such events. Although this is a plausible and provocative hypothesis, available data are not completely consistent (Contrada, Krantz, & Hill, 1988). For example, Kornfield, Kahn, Frank, Heller, Freeman, and Keller-Epstein (1985) found that SI-Type A was unrelated to intraoperative SBP among patients undergoing general surgery who had no history of CHD. Internal analyses did reveal, however, that SI-Type A was associated with intraoperative SBP among the small number of patients who had a positive family history of CHD.

Evidence for the heritability of Type A would provide further support for the somatopsychic model of Type A. While a global Type A does not evidence significant heritability (Rahe, Herzig, & Rosenman, 1978), the Type A components of explosive speech stylistics and potential for hostility do appear to reflect significant heritability quotients (Matthews, Rosenman, Dembroski, Harris, & MacDougall, 1984). Moreover, cardiovascular hyperresponsiveness to stress also may be heritable (Rose, Grim, & Miller, 1984; Smith et al., 1987). It appears that the biological contributions to Type A behavior and CHD risk merit further study; however, at present the data are too limited to permit any conclusions.

One implication of the biological approach to TABP might be that the behaviors are little more than epiphenomena with no direct relation to CHD. Such a conclusion is, at minimum, premature and, if accepted, could limit further understanding of biobehavioral pathways to CHD (Smith, 1989; Smith & Anderson, 1986). This point is illustrated by animal research on CAD (Manuck, Muldoon, Kaplan, Adams, & polefrone, 1989). These investigators placed cynomolgus monkeys fed an atherogenic diet into stable or unstable social environments. Two individual difference measures, heart rate reactivity to a capture stressor and social dominance, also were obtained. Autopsy data indicate that socially dominant male animals in an unstable social environment exhibited significantly greater CAD than did socially submissive animals in an unstable environment or both types of animals in a stable environment. Monkeys exhibiting greater heart rate reactivity also had more advanced CAD than their low reactivity counterparts. However, heart rate reactivity was not related to social dominance. Thus, simple individual differences in reactivity could not explain the interactive effect of social dominance and situational stress on CAD, even though subsequent research has indicated that this interactive effect is mediated by beta-adrenergic, sympathetic nervous system activity (Kaplan, Manuck, & Clarkson, 1987).

**Type A → Reactivity: Additional Considerations**

Although research available to date is not definitive, it does support the hypothesis that repeated episodes of cardiovascular and neuroendocrine arousal contribute to CHD (Manuck & Krantz, 1984). Individual differences, particularly TABP and its components, have been at the center of this research effort. Although precisely which element of Type A is related to reactivity and disease is an open question, the individual difference approach to CHD risk is still viable. However, as we have noted elsewhere (Smith, 1989; Smith & Anderson, 1986; Smith & Rhodewalt, 1986), basic conceptual issues guiding the study of individual differences in cardiovascular reactivity may restrict our full understanding of the ways in which individual differences in reactivity link TABP to disease.

Specifically, our concern is the ways in which individual difference variables are examined with regard to cardiovascular reactivity. One of two approaches is typically taken (Manuck & Krantz, 1984). Reactivity is often treated as a trait that is triggered by a pervasive set of daily events and produces a chronically elevated steady state of cardiovascular arousal in Type A individuals, which in turn places them at greater risk than Type Bs for CHD. Alternatively, reactivity is viewed as an attribute of the person who statically interacts with certain eliciting situations to produce greater increases in reactivity in Type As than in Type Bs confronted with the same situation. It is the relative increase in magnitude of reactivity that places the Type A at added risk for CHD. The focus in this latter model is on differences in reactivity between Type As and Bs in specified equivalent situations.
Both the trait and static interaction models of individual difference in cardiovascular reactivity have the potential to obscure the complex relationships between people and their environments conveyed in most recent approaches to personality (Cantor & Kihlstrom, 1987). Rather than statically interacting with situations, person characteristics can actively produce differences in situations. Individuals modify situations through their cognitions and behavior and, in turn, situations selectively elicit and reinforce certain cognitions and behaviors. Rather than simply possessing a set of stressful and cardiovascularly taxing coping responses, Type A may actually create many of the situations to which they are responding with exaggerated reactivity.

A transactional approach such as we are advocating appears to be warranted by an extensive body of recent research that collectively indicates TAPB is related to a variety of stress-engendering social and cognitive processes (reviewed in Smith & Anderson, 1986; Smith & Rhodewalt, 1986). Through their choice of situations (Feather & Volkmer, 1988; Ortega & Pepal, 1984), appraisals of situations (Carver, 1980; Rhodewalt & Comer, 1982; Rhodewalt & Davison, 1983; Smith & Brehm, 1981a), social interactions (Van Egeren, 1979a, b; Van Egeren, Snideman, & Roggelin, 1982), and self-appraisals (Cooney & Zeichner, 1985; O'Keefe & Smith, 1988), Type As operate on their environments in ways that should increase the frequency, duration and intensity of stressors, and, presumably, episodes of cardiovascular reactivity. The stress-engendering aspect of Type A behavior and its relation to cardiovascular reactivity is clearly illustrated in a study by Ortega and Pepal (1984). Compared with Type Bs, Type A subjects selected higher levels of task difficulty and exhibited higher levels of cardiovascular arousal when working on such tasks.

Differences between Type As and Type Bs in coping behaviors are also consistent with the view of TAPB as stress-engendering behavior. Compared with Type Bs, Type As prefer to work alone when under stress (Dembroski & MacDougall, 1978), have difficulty delegating work load (Strube & Werner, 1985), suppress or deny fatigue (Carver, Coleman, & Glass, 1976), and engage in active, problem-focused coping (Rhode- walt & Agustsdottir, 1984; Smith & Brehm, 1981b). All of these responses should increase the duration and, perhaps, intensity of exposure to reactivity-arousing situations.

The transactional model of TAPB and reactivity, although as yet untested, has several implications for future research. First, as noted previously, the SI is a better predictor of cardiovascular reactivity and CHD than the JAS. However, the JAS has been most closely associated with stress-engendering behavior. Tests of SI-TAPB as a correlate of a challenge-and-demand-engendering style, for the most part, have not been undertaken. It is possible, however, that different TAPB assessment instruments vary in the degree to which they measure reactivity versus tendencies to create stressful situations. As has been suggested elsewhere, the best prediction of behavioral risk for CHD may be gained from a combination of independent predictors of reactivity and challenge-and-demand-engendering style (Musante et al., 1983; Smith, 1989; Smith & Anderson, 1986; Smith & Rhodewalt, 1986).

Second, if future research confirms that hostility or antagonism and not other components of the TAPB are the sole toxic elements, the transactional model will still be a useful framework in which to study behavior, reactivity, and CHD risk (Smith, 1989; Smith & Frohm, 1985; Smith & Rhodewalt, 1986). As with global Type A, physiological reactivity is the presumed mediator between hostile behavior and disease (Williams, Barefoot, & Shekelle, 1985). Thus, hostile persons, through their cognitions, affective reactions, and behaviors, create more frequent, intense, and longer lasting interpersonal stresses, and experience concomitant episodes of arousal (Smith & Pope, 1990).

Although limited, recent research with the Cook-Medley Scale is consistent with this interpretation. Highly cynical, hostile people report more interpersonal stress and conflict in their families, marriages, and jobs than do low hostile individuals (Smith & Frohm, 1985; Smith, Pope, Sanders, Allred, & O'Keefe, 1988). High scores on the Ho scale are also associated with increased levels of antagonism, anger, and blame during discussions of marital conflicts (Smith et al., 1988). Further, highly hostile individuals, compared with their less hostile counterparts, displayed more aggression, reported more anger, and viewed their interaction partner more negatively when playing a mixed-motive game (Pope et al., 1989). Finally, compared with less hostile individuals, highly cynical, hostile people evidence greater increases in blood pressure during exposure to interpersonal stressors (Hardy & Smith, 1988; Smith & Allred,
1989; Suarez, Williams, & McCrae, 1988). It is equally plausible that the impatience and irritability associated with potential for hostility also has interpersonal consequences in terms of stressful, conflictive interactions. These findings highlight the potential relevance of the transactional approach to the study of hostility and CHD risk (Smith & Pope, 1990).

**TABP→Health Behavior→Disease**

In addition to differences in the creation of, or responses to, demanding situations, Type As and Bs differ on several other health-relevant dimensions that could place them at differential risk for CHD as well as for other health difficulties.

Type As are more likely than Type Bs to use denial and suppression when coping with a stressor (Carver, Coleman, & Glass, 1976; Gentry, Oude-Weme, Musch, & Hall, 1981; Pittner & Houston, 1980; Pittner, Houston, & Spiridigliozzi, 1983). Coping with denial is particularly prevalent among Type As when they are engaged in a task. For example, Type As who expected to continue working on an aversive task reported less fatigue and fewer symptoms than Type As who believed they had completed the task and Type Bs regardless of task involvement (Weidner & Matthews, 1978; see also Stern, Harris, & Elverum, 1981). Similarly, Schlegel, Wellwood, Copps, Gruenich, and Sharratt (1980) asked Type A and B postinfarct patients to report daily levels of challenge and MI-related symptoms such as chest pain. In addition, neuroendocrine measures constituted a physiological index of challenge. For both self-report and physiological measures of challenge, Type As reported fewer symptoms on high challenge days than on low challenge days. In contrast, Type Bs reported more symptoms with greater challenge.

Taken together, these findings indicate that Type As, particularly when they are engaged in an involving or challenging task, deny or fail to notice physical symptoms of fatigue and illness. Such denial can cause delay in seeking treatment and, in the case of MI, delay can lead to a more severe infarction and death (Matthews, Kuller, Siegal, Thompson, & Varat, 1983). More generally, Type As’ tendency to ignore symptoms and signs of distress may prolong their contact with stressful environments and delay or interfere with their seeking care and assistance.

Type As and Bs also differ in the way they respond to medical treatment. Type As are more sensitive to threats to behavioral freedom than are Type Bs (Glass, 1977) and respond to such threats with active and often demand-engendering attempts to reassert control. Studies indicate that, relative to Type Bs, Type As are more likely to perceive coercive intent (Carver, 1980) and respond with “reactance” (Wicklund & Brehm, 1976) or efforts to reacquire the threatened behavior (Rhodewalt & Comer, 1982; Rhodewalt & Davison, 1983; Snyder & Frankel, 1975).

Rhodewalt and his colleagues (Rhodewalt & Fairfield, 1990; Rhodewalt & Marcroft, 1988; Rhodewalt & Strube, 1985) have argued that to the extent that aspects of the health-care setting threaten one’s behavioral freedom, Type As should be likely to display reactance-motivated noncompliance with treatment (see also Taylor, 1979). In two demonstrations, JAS-defined TABP was significantly associated with noncompliance (Rhodewalt & Marcroft, 1988; Rhodewalt & Strube, 1985). Moreover, medically noncompliant Type As reported greater self-blame for the health problem, were more angry about it, and thought of it more as an entity to be fought than did compliant Type As and Bs (all responses thought to be characteristic of reactance).

Because studies also found many Type As to be compliant, the model requires additional refinement. Nonetheless, available data suggest that treatment noncompliance motivated by reactance is an alternative pathway through which Type A behavior affects health status. This observation takes on added significance when one considers the kinds of life-style changes prescribed to MI patients in order to reduce the risk of recurrent MI. Type A patients are more likely to construe such recommendations as threats to their behavioral freedom. They should then be more apt to continue unhealthy behaviors such as smoking or excessive work load and, consequently, increase the risk for recurrent MI. This speculation suggests that those providing cardiac rehabilitation should be sensitive to the control dynamics of the treatment regimen.

To summarize, the TABP-cardiovascular reactivity hypothesis has received the most attention and appears to be the most viable direct path between individual attributes and behaviors and CHD risk. Regardless of what component of the TABP turns out to be most toxic, exaggerated and frequent episodes of reactivity are likely to be the mediating link to accelerated CAD and increased
risk of CHD. Other aspects of the TABP also may contribute to CHD risk. Type As’ selective attention to symptoms and response to illness and treatment may indirectly contribute to increased risk for CHD.

THE PSYCHOLOGY OF TABP

A key question guiding much research on the TABP asks, Why do Type As behave as they do? This question becomes all the more important in the context of recent attempts to modify TABP in order to reduce CHD risk (Friedman et al., 1986; Gill et al., 1985). A thorough understanding of the psychological dynamics underlying TABP will permit the identification of critical cognitions, motivations, and behaviors as targets for intervention.

While Friedman and Rosenman provided a detailed description of the TABP, they were less specific about the developmental antecedents or psychological underpinnings of the behavioral syndrome. Friedman and Rosenman (1974) conjectured that feelings of insecurity and concerns about personal worth were at the core of the TABP. They suggested that these concerns probably went back to the Type A individual’s childhood experiences. Accordingly, the Type A’s excessive strivings and persistent struggles were attempts to acquire symbols of recognition and worth. After 15 years of research on the psychology of the TABP, researchers are just now beginning to substantiate Friedman and Rosenman’s observations.

TABP and Control Threat

David Glass was the first to investigate systematically a psychological account of the TABP. In a seminal monograph, Glass (1977) depicted the TABP as a set of coping responses elicited by perceived threats to control. According to Glass, the Type A behaviors of time urgency, achievement striving, competitiveness, aggressiveness, and hostility could all be considered control mastery behaviors that permit the Type A to reassert or maintain control in challenging situations. Repeated performance of these control mastery behaviors elicits cardiovascular and neuroendocrine arousal, and ultimately places Type As at elevated risk for CHD.

More than a decade of theory and research has elaborated on Glass’ original formulation. While the emerging perspectives to some degree share the assumption that the TABP is an active coping response to threat, they differ in their explanations for why this reactivity occurs. Collectively, the theory and research implicate a variety of self-evaluative and self- regulatory processes as central to the psychology of TABP.

TABP, Self-Involvement, and Identity

Scherwitz and colleagues (Scherwitz, Berton, & Leventhal, 1978; Scherwitz & Canick, 1988) proposed that Type As are more self-involved than Type Bs and that it is self-involvement that underlies CHD risk. This conclusion is based on the observation that SI-defined Type As respond to the SI with frequent self-references (the use of the personal pronouns I, me, my, mine). JAS-defined Type A is also associated with greater self-referencing (Rhodewalt, 1984). More important, Scherwitz, Berton, and Leventhal (1977, 1978) found that highly self-referencing Type As also displayed elevated systolic and diastolic blood pressure during the SI compared with low self-referencing Type As and highly and low self-referencing Type Bs (Scherwitz et al., 1977, 1978). Self-referencing is related to CAD severity (Scherwitz et al., 1983) and prospectively related to CHD, death from CHD (Scherwitz et al., 1987), and recurrent CHD (Powell & Thoresen, 1985). Self-referencing, then, particularly in interaction with extreme Type A behavior, appears to place one at risk for CHD (see Krantz, 1984, for an exception).

Initially, Scherwitz and colleagues (Scherwitz et al., 1978, 1983) suggested that Type A self-referencing reflected a greater degree of objective self-awareness or self-focused attention (Carver & Scheier, 1981). As such, Type As were more likely to compare performance outcomes with internal standards. Because most individuals and Type As in particular tend to hold high internal standards (cf. O’Keefe & Smith, 1988), the comparison process typically results in a large discrepancy between current performance and internal standards and subsequent effortful attempts to reduce the discrepancy. Although quite plausible, the chronic self-awareness hypothesis is not supported by the fact that in males TABP is unrelated to trait self-consciousness as indexed by the Private Self-Consciousness Scale (it is weakly related in women; Smith & Brehm, 1981b). In addition, self-referencing, at least in JAS Type As, appears to be a
function of self-attributional statements and not an indicator of more general self-salience in thought (Rhodewalt, 1984).

More recently, Scherwitz and Canick (1988) proposed that self-referencing is prominent in those with a fragile or vulnerable identity. Perhaps because of a lack of clear identity, Type As are constantly engaged in a chronic struggle to "shore up" their identity images. Self-referencing is thought to be in the service of maintaining and repairing desired, weakly held identities.

Scherwitz and Canick drew the parallels between self-referencers and narcissism and speculated that individuals with easily threatened identities may use hostility as a coping mechanism to ward off such threats. This speculation is supported by the reported association between frequency of self-referencing and potential for hostility (Dembroski, reported in Scherwitz & Canick, 1988). Although as yet untested, pursuing the relations among identity process, TABP, and CHD risk appears to be a potentially fruitful avenue.

**TABP and Cognitive Social Learning**

An alternative perspective was offered by Price (1982), who contended that the TABP is the result of cognitive social learning processes occurring in a context of social and cultural imperatives. At the core of her model is the assumption that Western society promotes the beliefs that one must constantly prove oneself and that resources are in short supply. Related to these beliefs are fears of insufficient self-worth, that self-worth is not constant and must always be demonstrated, and that insufficient resources are available. These beliefs, according to Price, form the cognitive basis of TABP and underpin the psychological and interpersonal characteristics of ambition and competitiveness. TABP, in this view, reflects a chronic striving for approval and material gain in order to prove one's self-worth. At the interpersonal level, attempting to prove oneself while coacting with hard-driving people engenders competitiveness. Competitiveness, in turn, triggers overt aggressiveness, easily aroused impatience, irritability, and hostility. Finally, while noting positive and negative, short- and long-term consequences of TABP, Price argued that, overall, the behavior pattern is sustained because the underlying beliefs guide the interpretation of positive and negative outcomes in a self-confirmatory fashion.

As noted previously, cognitive self-regulatory processes have been tied to TABP. Type As set higher goals for performance (Grimm & Yarnold, 1984; O'Keefe & Smith, 1988), are chronically self-critical (O'Keefe & Smith, 1988), and attend more to negative feedback than do Type Bs (Cooney & Zeichner, 1985). Further, Type A-B differences in satisfaction with performance are greatest when performance standards are ambiguous and disappear when performance standards are clear (O'Keefe & Smith, 1988). As O'Keefe and Smith noted, the pattern of Type A high standards and self-criticism are characteristic of cognitive social learning explanations for competitive, hard-driving, and achievement-oriented behavior (Carver & Scheier, 1981; Mischel, 1973). However, the predicted self-regulatory correlates are much more closely related to the TABP as defined by the JAS than by the SI (O'Keefe & Smith, 1988).

In sum, Price's social cognitive model of Type A is supported by studies of Type A self-regulatory processes. However, evidence of the proposed underlying cognitive set of beliefs and values is lacking. Burke (1984a, 1984b) reported small associations between TABP and measures of the beliefs described by Price, although the meaning of these findings is unclear because Price contended that Type As have little insight into these beliefs.

**TABP and Self-Appraisal**

Strube (1985) provided yet a different perspective on the psychology of the TABP. He suggested that Type As place a high value on the attainment and accurate assessment of success and productivity. In situations that create uncertainty about their ability, Type As, compared with Type Bs, respond with greater attempts to generate diagnostic feedback. In Strube's view, the TABP is a set of coping behaviors designed to acquire uncertainty-reducing, self-evaluative information. Strube reinterpreted much of the laboratory research on the TABP and responses to uncontrollability as evidence for Type As' exaggerated attempts to reduce uncertainty. In direct tests of the self-appraisal model, Strube and his colleagues (Strube & Boland, 1986; Strube, Boland, Manfredo, & Al-Falaij, 1987) demonstrated that compared with Type Bs, JAS-defined Type As made uncertain about an ability will select performance settings and work harder and longer in order to obtain uncertainty-reducing feedback.

Although Strube's model is provocative and ex-
isting data are largely congruent with his conclusions, the basic premise that Type As strive for accurate self-evaluation may be called into question. Rather, it seems that Type As strive for feedback and appraise such feedback in ways that substantiate their sense of self-worth and self-efficacy. For instance, Type As' self-attributions appear to be in the service of enhanced self-efficacy or control motives rather than for the purpose of accurate self-appraisal (Rhodewalt, Strube, Hill, & Sansone, 1988).

It is also noteworthy that although the findings of Strube and Boland (1986) and Strube et al. (1987) are consistent with self-appraisal predictions, they are also open to alternative interpretations. Specifically, in each study, attempts to make subjects uncertain about their abilities also may have threatened their control or challenged their self-worth. Seeking diagnostic feedback or persisting at a task may not have been behaviors aimed at obtaining accurate self-assessments so much as behaviors aimed at reasserting control or self-worth. The studies do not permit one to distinguish clearly which motives were operating. It is quite possible that the Type A-B differences in self-evaluative behaviors, taken as evidence for the self-appraisal model are, in fact, behaviors motivated by concerns about self-worth and identity (e.g., Price, 1982; Scherwitz & Canick, 1988).

**TABP and Antagonism**

Recently, Costa et al. (1989) proposed that the TABP component of potential for hostility is a correlate of the more basic personality dimension of agreeableness-antagonism. At this point the research is more descriptive than explanatory. However, antagonistic people are described as “self-centered, concerned with their own status, gain, or amusement. They are willing to fight for their goals and they view others as either hostile competitors seeking the same selfish ends or as contemptible fools” (Costa et al., 1989, p. 51). It is striking that antagonism also may be a manifestation of underlying concerns about identity and self-worth. Of course this conjecture, as well as the linking of antagonism with CHD outcomes, must await further research.

**TABP and Developmental Perspectives**

A theme that emerges across the theoretical statements about Type A people is that the TABP is motivated by concerns about the self-system. This conclusion is complemented by work addressing the developmental antecedents of adult behavior (Matthews & Siegel, 1982, 1983; Matthews & Woodall, 1988; Thoresen & Pattillo, 1988). Parent-child interactions and cognitive social learning processes are thought to contribute to TABP. For example, Matthews and Siegel (1982) proposed that during their developmental history, Type As' parents taught them to value performance and productivity but failed to provide clear performance standards. The consequence of these child-rearing practices is a child, and eventually an adult, who is chronically striving for poorly defined goals.

In a recent review of the literature on TABP and children, Thoresen and Pattillo (1988) also point to child-rearing practices and the development of self-regulatory behaviors in Type A children. They add to this discussion intriguing speculations about the relation of early attachment behavior and development of TABP. Borrowing from the infant attachment literature (Bowlby, 1982), Thoresen and Pattillo posited that 12- to 18-month-old infants who exhibit avoidant behavior when reunited with their mothers after a brief separation are likely to display TABP later in life. Specifically, children who were avoidant as infants were found 7 years later to be more hostile and easily angered (Sroufe, 1986).

Early infant attachment behavior is thought to reflect inner cognitive representations of reality that serve to guide expectations and explanations for future outcomes. Thus, Thoresen and Pattillo suggested that the underlying social cognitive schema of the Type A develops, in part, from the early experiences of the insecure-avoidant infant.

Like Scherwitz and Canick (1988), Thoresen and Pattillo also drew parallels between TABP and the literature on narcissistic personalities. It is striking that narcissists respond to threats to self-esteem with intense anger and hostility toward others. Thoresen and Pattillo also cited similarities between child-rearing practices implicated in the development of narcissism and the TABP. The parents of narcissists are believed to be inconsistent in meting out affection, highly competitive and critical, quick to anger, and eager to bask in the reflected accomplishments of their children (Miller, 1981).

Before proceeding to evidence pertinent to the development of the TABP, we should note that TABP can be assessed reliably in children as
young as 4 or 5 years of age (Corrigan & Moskowitz, 1983; Matthews & Angulo, 1980; Murray, Bruhn, & Bunce, 1983). However, several assessment instruments have been developed with only moderate convergent validity (for a review, see Thoresen & Pattillo, 1988). Thus, it is a concern that, as in the case of adults, the various modes of Type A assessment for children and adolescents are not measuring identical Type A characteristics. Therefore, additional psychometric development is in order. These assessment difficulties notwithstanding, there are findings indicating that Type A assessments in childhood predict individual differences in the TAPB in adolescence and early adulthood (Steinberg, 1986; Visintainer & Matthews, 1987).

A group of investigations implicate parent behaviors in the development of the TAPB and are consistent with a cognitive social learning interpretation. Mothers of Type A children give fewer positive evaluations to their child, encourage effort, and are more often rejecting than the mothers of Type B children (Matthews, 1977). Parents of Type A children are also more indiscriminate in their administration of both encouragement and criticism (Bracke, reported in Thoresen & Pattillo, 1988).

Perhaps most important is the finding that mothers of Type A children, particularly non-Type A mothers, are more likely to provide unclear or ambiguous performance standards for their children (Matthews, 1977). This practice no doubt contributes to the Type A child's preference to compare socially with superior others and to make negative self-statements and attributions, particularly under conditions of challenge and ambiguous performance standards (Matthews & Volkin, 1981; Murray, Matthews, Blake, Prineas, & Gillum, 1986).

To summarize the theoretical perspectives of TAPB, a picture of the Type A individual is developing that is more firmly tied to current cognitive social learning theories of individual differences (Cantor & Kihlstrom, 1987). Although still undocumented, current researchers are suggesting that either overly rigid or poorly formed self-conceptions make Type As sensitive to self-evaluative threats and motivate them to operate on their social environments to obtain feedback and symbols of their self-worth. The available developmental evidence is consistent with the view that child-rearing practices, particularly but not exclusively those of the mother, foster different self-regulatory strategies that could account for adult TAPB. However, as with other areas of TAPB investigation, issues concerning the valid and reliable assessment of coronary-proneness are limiting progress. It is clear that life-span studies of CHD risk, including multiple means of TAPB assessment and employing transactional or at least interactionist methodologies, are required to understand fully the complex relationships among familial factors, individual differences, social interactions, environment, and CHD. Further, many psychological approaches to the development and maintenance of the TAPB have focused on the global pattern rather than on the more specific and toxic element of hostility. More specific—or perhaps different—approaches may be necessary as the focus of Type A research shifts.

**TAPB, INTERVENTION, AND REDUCED RISK FOR CHD**

A justification for the need to understand any disease risk factor is the presumption that such knowledge will permit the development of risk-reduction interventions. Such has been the case with the TAPB. In fact, interest in the TAPB has been sustained in part because of recent reports of successful TAPB modification and concomitant reductions in CHD risk (Friedman et al., 1986; Gill et al., 1985). These recent, broad-based intervention trials were preceded by a group of small-sample, narrow-focused, relatively brief duration intervention studies. Although these studies suggested that modification of the TAPB might be viable, they were generally burdened with conceptual and methodological shortcomings that made them of limited value. Reviews of this earlier work may be found in Chesney, Eagleston, and Rosenman (1981), Haaga (1987), and Nunes, Frank, and Kornfeld (1987) and will not be discussed here.

The Recurrent Coronary Prevention Project under the direction of Meyer Friedman (RCP; Friedman et al., 1982) was a 5-year clinical intervention trial with 1,035 postinfarct patients. The RCP attempted to modify the TAPB, document its alteration, and evaluate reductions in the TAPB against "hard" clinical endpoints of CHD, specifically recurrent MI and coronary death. RCP participants were individuals who had suffered a documented MI 6 or more months previous to the study. Both self-reports and videotaped structured interviews (VSI) were used to
assess the TAPB and these measures revealed that
approximately 97% of the participants exhibited
moderate to extreme Type A behavior. Participants
were randomly assigned to either a cardiac
counseling group who received information about
diagnosis and treatment of CHD, along with
counseling to enhance adherence to dietary, exer-
cise, and drug regimens, or a cardiac counseling
plus TAPB modification group. There was also a
control group from whom only intake and out-
come measures were obtained.

The TAPB modification group received broad-
based intervention designed to teach them relaxation
and breathing techniques and ways of modifying
their Type A cognitions, behaviors, and affective
responses (see Powell & Thoresen, 1987, for a de-
tailed description of TAPB group therapy). Modifi-
cation of the TAPB included instruction of self-observation and assessment, and restruct-
uring of the environment. Participants were
taught to modify Type A assumptions, attitudes, and
beliefs, and to acquire new realistic values,
beliefs, and internal standards. They were taught
self-management through observation and role-
plays of Type B behavior. Finally, they were en-
couraged to invest in “things worth being” such as
the renewal of old friendships, the acquisition of
new hobbies, and the substitution of assertiveness
for hostility.

Several follow-up reports of the effectiveness of
the modification attempt are now available and
the results are quite striking. At the end of 4.5
years, patients receiving Type A and cardiac coun-
seling had a significantly lower level of recurrent
CHD (12.9%) than did patients receiving only
cardiac counseling (21.2%; Friedman et al.,
1986). In addition, both self-report and VSI data
revealed significantly greater reductions in the
TAPB in the Type A modification group com-
pared with the cardiac counseling group. At the
end of 4.5 years, 35.1% of the TAPB modification
group had shown at least a I standard deviation
[SD] decline in both self-reported and VSI Type A
scores compared with only 9.8% of the cardiac
counseling group (p < .0001; Friedman et al.,
1986). More importantly, regardless of the treat-
ment group, those patients who showed a clear
reduction in the TAPB suffered only one-fourth
the CHD recurrences as those who failed to show
a change. Participants who displayed only some
reduction in Type A scores had CHD recurrence
rates between the clear reduction and no reduction
groups, indicating a nonsignificant trend toward
a dose-response relationship (Powell, Friedman,
Thoresen, Gill, & Ulmer, 1984).

Gill et al. (1985) employed the RCPP’s Type A
modification program with a group of healthy
Type A participants (officer-students at the U.S.
Army War College [USAWC]). At the end of the
9-month treatment program, over 40% of the Type
A modification group showed a significant reduc-
tion in TAPB scores compared with 9% of no-
treatment controls. Moreover, significant reduc-
tions in Type A scores were associated with
significantly lower serum cholesterol levels.

The two intervention studies reported above
provide fairly compelling evidence that the TAPB
can be modified and that such changes are asso-
ciated with reduction in CHD risk. However, as
Price (1988) noted, these studies leave many unan-
swered questions. After successful replication of
these treatment effects, studies are needed to eval-
uate the duration of reductions in Type A behav-
or and the long-term risk reductions associated
with such changes (Price, 1988).

A related concern has to do with the length of
treatment necessary to produce meaningful alter-
ations in the TAPB. Counseling in the RCPP con-
tinued throughout the study; biweekly for the
initial 3 months, monthly for the next 3 months, and
bimonthly for the remainder of the study (a total of
33 sessions). Friedman et al. (1986) noted that
although the effect of Type A treatment on decline
in TAPB is continuous over the 4.5 years, the
greatest decline is observed after the first year.
Treatment in the USAWC study lasted for 9
months (21 counseling sessions). Gill et al. (1985)
reported that 44% of the TAPB counseling sub-
jects displayed marked or some reduction in Type
A scores compared with 8.9% for controls at 8
months.

Collectively, the findings from these interven-
tion trials indicate that statistically reliable reduc-
tions in Type A behavior can be obtained after as
little as 8 months of counseling and maintained
with continued counseling for up to 4.5 years.
They also reveal that for many Type As the inter-
ventions do not produce marked reductions (-1
SD) in Type A behavior (65% in the RCPP, in-
cluding those who dropped out of treatment, and
58% in the USAWC sample). There are two impor-
tant implications in these data. First, 42.7% of the
RCPP TAPB counseling participants dropped out
of treatment. To the extent that treatment is ben-
eficial, then attention should be directed toward
promoting participation and adherence to thera-
apeutic recommendations. Treatment endeavors should be cognizant of the potential threats to control and self-esteem inherent in broad-based life-style interventions, threats to which the Type A is likely to be particularly sensitive and reactant (Rhodewalt & Fairfield, 1990; see also Krantz & Schulz, 1980). Thus, TABP modification procedures are likely to be more effective if they permit the individual to maintain a sense of self-efficacy and worth.

In addition, as Price (1988) suggested, new treatment approaches based on recent developments in the conceptual models of the TABP need to be tested and compared with current treatments. If only certain components of the TABP such as hostility are toxic, then focused interventions targeted at hostility or the self-concept might be more effective than attempting to modify the global TABP. Several interventions have been found effective in reducing anger and hostility (Hazuleus & Deffenbacher, 1986; Moon & Eisler, 1983; Novaco, 1975; for a review see Biaggio, 1987). Narrowly focused interventions may also be less threatening to the Type A individual.

These are only a few of the issues facing those interested in reducing CHD risk through modification of behavioral risk factors. The reader is directed to recent, more extensive reviews of the conceptual, methodological, logistical, and ethical concerns facing intervention researchers (Levenkron & Moore, 1988; Price, 1988). In sum, large-scale, long-duration intervention studies are needed to address the many questions remaining. Future intervention trials will be useful not only for their applied significance, but for the opportunity they provide to test basic theoretical questions. Finally, one need always keep in mind that CHD is a multidetermined outcome and that reduction of other risk factors is desirable, particularly in Type As who are resistant to behavioral change.

**CURRENT STATUS AND REFLECTIONS**

This chapter has attempted the daunting task of summarizing 30 years of theory and research on the TABP as a behavioral risk for CHD. We have sought to evaluate the current status of the Type A hypothesis and clarify unresolved issues for future research. At present, the consensus is that some but not all of the loose constellation of attributes defining the TABP place Type A individuals at elevated risk for CHD. The most likely culprit in the array is hostility. This elevated risk most likely occurs through the contribution of frequent, exaggerated episodes of cardiovascular and neuroendocrine reactivity to CAD. This general conclusion is qualified by the method of assessing the TABP and its components and by the populations studied. Further, research indicates that a set of self-conceptions and self-regulatory behaviors, probably acquired through social learning experiences beginning in early childhood, form the psychological foundation for much of the TABP. Moreover, successful modification of the TABP is associated with reduced risk for recurrent CHD.

The above statement about the current status of the TABP is a global summary based on active areas of research. Thus, current conclusions will be confirmed or revised through continued research on assessment of risk, biobehavioral pathways to disease, underlying developmental, social, and psychological processes associated with behavioral risk, and the efficacy of clinical intervention.

Despite the eventual judgment about the importance of the TABP in the development of CHD, research on the TABP provides valuable lessons for those concerned with models of personality and disease and their clinical applications. That is, 30 years of research on psychosocial and behavioral risks for CHD serve as the prototype for individual difference approaches to health outcomes such as cancer (Greer & Watson, 1985). Investigators in these fields will benefit by examples from research on the TABP.

What are those lessons? First, research on the TABP and CHD illustrates the complexity of studying personality and disease relationships. Epidemiological evidence is often not as informative as it might be. Epidemiological studies as well as intervention trials are costly, labor intensive, and time consuming. Such studies of risk and intervention will be more useful if greater attention is given to the testing of underlying theory. Epidemiological investigations and clinical intervention trials will be most useful if preceded by thoughtful development of conceptual models and careful psychometric development of assessment devices. Then, the conceptual models must be faithfully translated into epidemiological designs. For example, there is a vast discrepancy between models of the TABP-CHD association and epidemiological studies of those models. As noted previously, the models suggest that Type As in certain situations
display exaggerated reactivity and are ultimately at increased risk. Yet, situational factors are rarely assessed, and the conceptually more precise interactional effect cannot be tested. The default test of the Type A main effect is not the most theoretically relevant and may be less sensitive.

An additional interpretive difficulty is present in many studies of the TABP and CHD risk. Most conceptual models of TABP propose that the behavioral risk contributes to CHD by accelerating CAD. Frequent, exaggerated cardiovascular and neuroendocrine responses are thought to mediate the disease process. Often, epidemiological investigators confuse independent and mediating variables and seek to find an independent relationship between TABP and CHD while controlling for serum cholesterol or blood pressure, variables reflecting or at least related to the presumed mediator.

The same concern holds for the TABP and other risk factors as well. To illustrate, if the relationship between TABP and CHD holds only before controlling for cigarette smoking, then the typical conclusion is that TABP is not an independent risk factor for CHD above the risk accrued from smoking. This is true, but it begs the important question of why the TABP might be associated with smoking.

At the same time, we encourage investigators to consider null findings carefully. Early Type A research appears to be limited by a confirmatory bias that prohibited the critical evaluation of the Type A construct, and thus, encouraged investigators to invest research resources into areas with limited potential for advancing our understanding of behavioral risk for CHD (Booth-Kewley & Friedman, 1987).

In sum, there have been many false starts and dead ends encountered along the way and there are many questions as yet unanswered. However, the cumulative result of the vast literature on the TABP is an emerging understanding of the basic mechanisms that define coronary-prone behavior.

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