Type A Behaviour, Physical Fitness, and Physiological Reactivity

by

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To Susan,

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ABSTRACT

The mechanism whereby Type A behaviour (TABP) might influence the development of cardiovascular disease (CHD) is not known. However, alteration in the production of chemicals involved in platelet aggregation have been associated with a predisposition to CHD. Further there is evidence that suggest that these chemicals are influenced by psychological stress as well as exercise. In contrast to the detrimental effects of the TABP, it has been demonstrated that long-term aerobic conditioning is associated with both physiological and psychological benefits, which may be protective against CHD.

This study examined the interaction between the TABP, physical fitness, and physiological reactivity, with a particular focus on platelet aggregation measures. The two prostaglandins that were of interest to this study are thromboxane and prostacyclin. Thromboxane, which is produced by blood platelets, stimulates platelet aggregation and vasoconstriction. Prostacyclin, which is released from the vessel wall, blocks further platelet adhesion and aggregation to an injured vessel, stimulates vasodilation, and has an important role in preventing propagation of clots. Recent evidence suggests that these prostaglandins may be implicated in the development of atherosclerosis and CHD, and that their production may be altered as a result of both emotional and physical stress.

The Structured Interview for university students was

used to assess behaviour type. In addition, questionnaires were used to measure the hypothesized "toxic" components of the TABP (i.e., time urgency, anger expression, and hostility). The physiological concomitants of Type A behaviour included measurement of the blood chemicals thromboxane A_2 and prostacyclin. Heart rate, systolic and diastolic blood pressure were also measured. Fitness level was indexed by VO_2max values (i.e., maximal oxygen uptake), measured through a graded treadmill test. A sample of 97 male university students participated in the study. The experiment consisted of two sessions. Fitness level was determined in the first session, while during the second session subjects were exposed to a psychosocial stressor (i.e., the Stroop color-word test).

The major result of this study was that Type A behaviour and aerobic fitness have an interactive effect on cardiovascular functions and the production of thromboxane. That is, while at rest, unfit Type As were found to have higher levels of thromboxane and higher heart rate than did fit Type As and Type Bs.

In response to the psychosocial stressor, Type As showed greater reactivity than did Type Bs in terms of both heart rate and systolic blood pressure. No individual differences were observed in the production of the prostaglandins during the stressor. However, unfit Type As continued to demonstrate shorter bleeding times at the end of

the stressor, which suggest that an unknown hemostatic variable may have inhibited the production of the prostaglandins during the stressor. Finally, Type As were found to recover from the stressor at a slower rate than Type Bs in terms of heart rate.

These results strongly suggest that at resting state, the unfit Type As present with higher cardiovascular risk status. The results of this study strongly suggest that the benefits associated with aerobic fitness may protect Type A individuals against the adverse effects of their coping style to stress.

OVERVIEW

The Type A behaviour pattern was first introduced by cardiologists Friedman and Rosenman (1974) as a result of their observations of the behavioral characteristics of coronary patients. To use their now popular definition, the Type A pattern is "an action-emotion complex that can be observed in any person who is 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 or other persons" (Friedman & Rosenman, 1974, p. 67). The major features of this behaviour pattern include extremes of aggressiveness, easily aroused hostility, a sense of time urgency, and competitive achievement striving. Furthermore, the Type A pattern is thought to be a continuum of behaviours ranging from extreme Type A to extreme non-Type A or Type B (Matthews, 1982).

Over the past 25 years, psychologists and medical researchers have invested considerable time and effort in an attempt to understand the overt behavioral style known as Type A. The interest generated by this research has made Type A behaviour a common part of lay terminology and a frequent conversation topic at cocktail parties. One of the main reasons for the rapid growth of published work on the Type A pattern is that it has been identified as a coronary heart disease (CHD) risk factor. That is, there is some

evidence that the Type A behaviour pattern (TABP) is associated with increased risk of CHD, and this increased risk is independent of other traditional risk factors such as elevated blood pressure, serum cholesterol level, and smoking (Williams, Haney, Lee, Hong Kong, Blumenthal, & Whalen, 1980).

Many researchers have recently questioned, however, the utility of a global Type A construct, especially in light of findings that indicate that components of the TABP such as hostility and anger are better predictors of CHD than is the global measure of Type A (Barefoot, Dahlstrom, & Williams, 1983; Blumenthal, O'Toole, & Haney, 1984; Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; Shekelle, Gale, Ostfeld, & Oglesby, 1983; Strube, Turner, Cerro, Stevens, & Hinchey, 1984; Williams et al., 1980). It is suggested that hostility in conjunction with anger suppression may lie at the foundation of coronary-prone behaviour.

In the last 10 years, several attempts have been made to alter risk factors for CHD in Type A individuals. Suinn and Bloom (1978) developed an anxiety management training program which was shown to lower scores on the Jenkins Activity Survey scales measuring anxiety, speed and impatience, and the hard driving component. Roskies, Spevack, Surkis, Cohen, and Gilman, (1978), reported

reductions in systolic blood pressure, serum cholesterol, and several psychological symptoms in a group of healthy, middle-aged male volunteers who underwent either brief group psychotherapy or behaviour therapy. Roskies, Seraganian, Oseasohn, Hanley, Collu, Martin, and Smilga (1986) have also recently reported positive findings in reducing behavioral reactivity (as measured by the Structured Interview) among a sample of Type A managers with a 10-week cognitive-behavioral stress management program. In light of estimates which suggest that between 50 and 75% of the general population may be Type A (Rosenman, 1974), the cost-effectiveness of preventative programs such as those developed by Suinn or Roskies may be questionable.

The use of aerobic physical activity as a preventive approach to CHD has received growing attention. Interest in physical activity has risen dramatically since the early 70's, mostly because of the popular belief that physical fitness will increase longevity and protection against CHD, the leading cause of death in the US (Rigotti, Thomas, & Leaf, 1983). Large scale epidemiologic studies have also shown that vigorous exercise is associated with lower mortality due to CHD (Paffenbarger, Hyde, Wing, & Hsieh, 1986; York, Mitchell, & Graybiel, 1986).

The primary purpose of this study is to examine the interaction between physical fitness, the TABP and its toxic

components (i.e., hostility, anger, and time urgency), and physiological reactivity. Few studies have examined whether the risk profile of Type A individuals is affected by fitness level. As discussed in the following sections, it does not appear that hypotheses related to the interaction of physical exercise and psychosocial risk factors for CHD have been adequately tested. However, prior to reviewing the literature on TABP and physical fitness, the techniques for assessment of the Type A pattern will be described since it has been suggested that these measures do not assess the same aspects of the Type A pattern.

ASSESSMENT OF TYPE A

Although a number of measures have been developed to assess the Type A pattern, four were found to be related to CHD: the Structured Interview (SI; Rosenman, 1978), the Jenkins Activity Survey (JAS; Jenkins, Rosenman & Zyzanski, 1974), the Framingham Type A Scale (FAS; Haynes, Feinleb, & Kannel, 1980), and the Bortner Rating Scale (Bortner, 1969).

The Structured Interview (SI). The SI (Rosenman, 1978) was the assessment tool developed and used in the original study demonstrating a relationship between the TABP and CHD, i.e., the Western Collaborative Group Study (WCGS; Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975). It is a provocative interview where the questions are presented in

such a way as to elicit impatience, hostility, and competitiveness from Type A individuals. Both the content of answers and style of speech behaviours are used in the classification of the behaviour pattern. Based on the results of the interview, individuals are classified into one of the following categories: A1, extreme Type A; A2, moderate Type A; X, equal representation of Type A and B behaviour; B3, moderate Type B; B4, extreme Type B. As to the reliability of the SI, the interjudge agreement of classification usually ranges between 75 and 90%, while test-retest reliability of 80% agreement in the dichotomous A-B classification over periods that ranged from 12 to 20 months, have been reported in the WCGS (Dembroski, 1978). As to the validity of the SI, it is based on its association with CHD as measured in the Western Collaborative Group Study (Rosenman et al., 1975).

The Jenkins Activity Survey (JAS). This self-report measure (i.e., questionnaire) was designed to be very similar to the SI. The items were selected on their ability to discriminate between the SI-defined Type As and Bs. It was developed to provide an objectively scored measure of Type A behaviour (Jenkins et al., 1974). In addition to the A/B scale, the JAS also yields scores on three scales, i.e., speed and impatience, hard-driving, and job involvement. The test-retest reliability correlation of the JAS has been reported to be in the 0.60s across one to four years

(Jenkins, 1978). As to the validity of the JAS, it was established by comparing JAS scores to Type A ratings based upon the SI with the WCGS population. Jenkins (1978) reported that the SI ratings and JAS scores corresponded 73% of the time. The validity of JAS has also been established by demonstrating that it is significantly associated with the incidence of CHD (in the WCGS population). However, more recent reports indicate that test-retest reliability (i.e., with a six week interval) can range from 0.43 to 0.72 amongst university student populations (Landy, Thayer, & Colvin, 1988). Among the self-report measures of the TABP, the JAS has been used most frequently (Matthews & Haynes, 1986).

The Framingham Type A Scale (FAS). The FAS was developed to assess the TABP in the Framingham Heart Study (in the town of Framingham, Massachusetts) (Haynes et al., 1980). It contains 10 items that assess an individual's competitive drive, sense of time urgency, and perception of job pressures. The validity of the FAS was established by demonstrating that the scale was predictive of CHD in the Framingham Heart Study.

The Bortner Rating Scale. This scale was developed from the SI and was intended to serve as a screening measure of Type A behaviour (Bortner, 1969). It has been used mostly with European samples. This 14-item scale has been reported to have test-retest reliability correlation of 0.71 with a 4

month interval, to correlate highly with the JAS, and to predict CHD (Bass, 1984). The validity of the scale was demonstrated by its agreement with the SI, i.e., the two scales agreed 75% of the time in the Belgian-French Cooperative Heart Study (see Matthews & Haynes, 1986).

Despite the claim that all these measures assess the Type A pattern, the association between the SI, JAS, and FAS, is much lower than one might expect. The overlap between these measures has been reported to be only 10-20% above the 50% chance level (Matthews, 1982; Matthews, Krantz, Dembroski, & MacDougall, 1982). Myrtek and Greenlee (1984) reported in their review that the average correlation between the SI and JAS is 0.34, which means that the common variance shared by these methods is approximately 11% . As to the FAS, it has been found to correlate weakly with the SI ($r = .20$) and moderately with the JAS ($r = .53$) (Blumenthal & Kamarck, 1987). It is therefore fair to assume that the most commonly used measures assess different aspects of personality and of the TABP.

The SI appears to measure a general reactivity to provocative situations, the latter taking the form of rapid, loud, and explosive speech, and physiological responding, while the JAS is unique in measuring of self-reported time pressure (Matthews et al., 1982). There are indications that the SI-defined Type A behaviour is more closely related to

A/B differences in physiological responding (MacDougall, Dembroski, & Krantz, 1981; Contrada, Wright, & Glass, 1985), and that the JAS is more sensitive to the detection of psychological differences (Musante, MacDougall & Dembroski, 1984).

These differences between the SI and the self-report measures may account for the studies reporting a weak or no association between Type A Behaviour and CHD (this literature will be reviewed in the following section). For instance, Contrada et al. (1985), found in their review of the literature that studies using the SI consistently found a relationship between the pattern A and physiologic reactivity (i.e., systolic blood pressure, diastolic blood pressure, and heart rate), while the studies using the JAS failed to do so. These latter observations are consistent with recent meta-analyses of the Type A literature which reported a strong association between Type A and CHD when TABP was defined by the SI and not by the JAS (Friedman & Booth-Kewley, 1988; Matthews, 1988). Since physiological reactivity is the leading hypothesis concerning the means by which the Type A pattern is related to CHD (Musante et al., 1984), the JAS-defined Type A pattern appears to be a particularly weak predictor of coronary risk. In light of these findings, it has been suggested that the terms Type A and coronary-prone behaviour should not be used interchangeably (Matthews, 1982).

It has also been suggested that only certain components of Type A behaviour are associated with CHD, namely hostility and anger (Matthews & Haynes, 1986) (this literature will be reviewed in the following section). Hence, with respect to Type A assessment measures, it appears that the greater strength of association of CHD with SI-defined TABP as compared to JAS-defined Type A behaviour may be due to the stronger focus of the SI on hostility and anger (Dembroski et al., 1985). This suggests that hostility and anger may be considered toxic elements of the TABP in relation to CHD.

TYPE A BEHAVIOUR AND CORONARY HEART DISEASE

This section will review studies that have investigated the relationship between global Type A behaviour as assessed by the four major assessment techniques, and coronary heart disease. Population-based studies will first be reviewed, followed by studies of high-risk persons.

Population-based studies. The first major prospective study that was specifically designed to examine the association between the TABP and CHD was the Western Collaborative Group Study (WCGS; Rosenman et al., 1975). This large scale study had a sample of over 3000 middle-aged men (35-59) who were free of CHD symptoms at intake into the study. As assessed by the SI, 50.4% of the sample were Type A. At the end of an 8.5 years follow-up, Type A subjects

were over two times more likely to develop CHD than Type Bs. The JAS was also administered to a sub-sample of WCGS, and followed-up over four years (Jenkins et al., 1974). The results were such that (JAS-defined) Type As were 1.8 times more likely to develop CHD over a four-year period than Type Bs.

The Framingham Heart Study was another large scale prospective study that examined the relationship between psychosocial factors and the incidence of coronary heart disease over an eight-year period (Haynes et al., 1980). The subjects (n=1674) included men and women in both white and blue collar jobs who were free of CHD symptoms at the beginning of the follow-up period. At the end of the follow-up period, Type A men (as assessed by the FAS scale) were reported to be 1.8 times more likely to develop CHD than Type Bs. However, it was also found that Type A men employed in white-collar occupations were 2.9 times more likely to develop CHD than Type B men. The Framingham study was also the first to report on the association between TABP and CHD among women. In the overall sample, Type A women had a slightly higher risk (2.2 times) than the Type A men (1.8 times). It was also found that working women did not have an increased risk of CHD as compared to housewives, i.e., Type A working women and Type A housewives had an increased incidence of CHD as compared to their Type B counterparts.

Similar findings were also obtained with European populations. The Belgian-French Cooperative Heart Study, using the Bortner Rating Scale to assess Type A, reported that among a healthy population of 2811 male civil servants and factory workers, Type A was a significant predictor of CHD (i.e., Type As were between 1.5 and 1.8 times more likely than Type Bs to develop CHD) (see Matthews & Haynes, 1986).

Finally, using the JAS to identify Type As among nearly 2000 healthy men, DeBacker et al. (1983) reported in the Belgian Heart Disease Prevention Trial that Type As were 1.9 times more likely to develop CHD than Type Bs. Unfortunately, no multivariate analyses to control for other CHD risk factors were reported.

Studies of high-risk persons. Jenkins, Zyzanski, and Rosenman, (1976), using the WCGS population, followed-up for at least one year 267 men who had had at least one CHD event. Their results showed that the FAS Type A score was a significant predictor of recurrent CHD events, while controlling for other CHD risk factors.

More recent studies have failed to consistently support the TABP and CHD relationship. For example, Dimsdale et al. (1981) studied the predictors of new CHD events in a group of 189 men who had undergone cardiac catheterization because of CHD symptoms. After a follow-up of one year, no association was found between JAS defined Type A and new CHD events. On

the contrary, they reported that Type B was predictive of new CHD events. Similarly, Case, Heller, Case, and Moss (1985) measured JAS-Type A behaviour in over 500 patients within two weeks after an acute myocardial infarction. After a follow-up period of one to three years, they found no relation between the Type A score and total mortality or cardiac mortality.

Perhaps the most ambitious replication attempt (i.e., of earlier findings suggesting that Type A predicts CHD) was the MRFIT intervention study (Shekelle, Hulley, Neaton, et al., 1985). This study included middle-aged men (35-57) who had no evidence of CHD but who had levels of smoking, serum cholesterol, and diastolic blood pressure sufficiently high to place them in the upper 15% of the distribution of risk factors. It was reported that behaviour type as assessed by either the SI or JAS was not significantly associated with risk of first major coronary events after a follow-up of at least seven years.

Similar results were obtained by Shekelle, Gale, and Norusis (1985) who administered the JAS to 2314 participants in the Aspirin Myocardial Infarction Study. All the subjects had a myocardial infarction before entering the study and were followed for at least 3 years. JAS-defined Type A behaviour did not predict recurrence of CHD in either men or women.

In summary, several early large scale prospective studies with population-based samples (i.e., free of CHD), have shown that Type A behaviour is a risk factor for coronary heart disease. These studies were instrumental in the sponsoring of a panel by the National Institutes of Health to review the literature on the association between Type A behaviour and CHD (Review Panel on Coronary-Prone Behaviour, 1981). This panel concluded that Type A behaviour constituted an independent risk factor for CHD.

In contrast, more recent studies of persons already at risk for CHD consistently failed to support the hypothesis that Type A behaviour is a significant contributor to this risk status. Furthermore, these later negative findings were obtained with both the SI and the JAS, suggesting that the failure to find an association between the TABP and CHD in those studies cannot be attributed to the measurement error of one particular assessment tool. As a result, the current state of affairs with respect to the status of coronary-prone behaviour may best be described as one of confusion. The TABP hypothesis appears less empirically robust now than it did at the time when the review panel (1981) concluded that the TABP was an independent risk factor (Williams & Barefoot, 1987).

These negative findings in fact corroborate earlier conclusions which suggested that one of the shortcomings of

previous Type A research has been the failure to appreciate the multidimensional nature of the Type A construct. This is seen in the interchangeable use of Type A measures that have little overlap, and which as global measures lack predictive power (Matthews, 1982). If only certain aspects of the multidimensional nature of TABP are "toxic", then assessment of the global construct would provide a measure that may contain a considerable amount of "noise" in addition to the coronary-prone component(s) (Williams & Barefoot, 1987). This has led researchers to question the utility of a global Type A construct and to subsequently investigate specific components of the Type A pattern that might be better predictors of CHD.

COMPONENTS OF TYPE A AND CHD

The first indications that some aspects of the global TABP are more coronary-prone than others came from a comparison of SI responses of 62 CHD cases among participants of the Western Collaborative Group Study with 124 symptom-free controls matched for age and place of employment (Matthews, Glass, Rosenman, & Bortner, 1977). The SI responses were coded according to the content and style of the responses, and out of 40 different ratings, only eight discriminated between cases and controls. These ratings included: potential for hostility, explosive voice modulation, vigorous answers, frequent experience of anger,

irritation at waiting in lines, competitiveness, and time urgency. Of interest, one should note that most of these items are concerned with hostility and anger. Unfortunately, multivariate analyses were not conducted on these data to evaluate the contribution of CHD risk factors other than the TABP. Hence, it is difficult to say which proportion of the variance was accounted for by these items.

Guided by the findings of Matthews et al. (1977) that suggested that hostility and anger may be strong predictors of CHD, and also by an older psychoanalytic literature that emphasized the role of repressed anger and strong needs to dominate others in the development of CHD, Williams et al. (1980) evaluated the relationship between CHD severity, the SI-defined TABP, and the MMPI-based hostility (Ho) scale scores (Cook & Medley, 1954). Among a sample of 424 men and women patients referred for diagnostic coronary arteriography, they found that SI-defined TABP and Hostility score were independently and significantly related to severity of arteriographically documented coronary atherosclerosis, this relation being as strong in women as it was in men. Furthermore, multivariate analysis revealed that the hostility score was more strongly related to atherosclerosis than was the TABP. These findings led Williams et al. (1980) to conclude that hostility, as measured by the Ho scale, may be the only aspect of the global TABP which contributes to coronary proneness.

Further evidence that hostility as measured by the MMPI-based Ho scale is associated with the development of CHD is also provided by two prospective studies. In a 25-year follow-up of 255 medical students to whom the Ho scale had been administered, Barefoot et al. (1983) found that subjects with high Ho scores (i.e., Ho > 13) were five times more likely to develop CHD than were subjects with low scores. Shekelle et al. (1983), also found in a population of nearly 2000 middle-aged healthy men that high hostility scores (Ho scale) had a significantly higher 10-year incidence of CHD than low hostility scores. This association was found even after multivariate adjustment for age, cholesterol level, number of cigarettes smoked per day, and alcohol consumption. Unfortunately, Type A behaviour was not assessed in these two studies.

To date, only one study using the Ho scale failed to find an association between hostility scores and CHD. This study followed up for 25 years a sample of nearly 500 physicians who had completed the MMPI while in medical school (McCranie, Watkins, Brandsma, & Sisson, 1986). The latter was similar in many respect to the Barefoot et al. (1983) study where the MMPI was completed as a class exercise and the subjects were told that the results would only be used for research purposes. In contrast however, in the McCranie et al. (1986) study, the subjects were told that the results

would play a role in their admission to medical school. In light of these different testing conditions, it is not surprising that the hostility scores were quite low in the McCranie et al. (1986) study, and it is possible that the results were compromised by a social desirability bias. In summary, with the exception of the McCranie et al. (1986) study, higher hostility scores as measured by the MMPI-based Ho scale have been found to be highly correlated with the severity of CHD, and also to predict CHD rates in men over follow-up period ranging from 10 to 25 years.

It should be noted at this point that the Ho scale was developed by Cook and Medley (1954), and was designed to assess a form of hostility that is characterized by cynicism, resentment, and a distrust of others. It appears to be a very stable trait, with test-retest correlations of .84 (over 4 years) in the Shekelle et al. (1983) study and of .85 after 1-year in Barefoot et al. (1983). These coefficients are consistent with Cook and Medley's (1954) earlier report of .86 . Recent evaluation of both convergent and discriminant validity support the assumption that the scale measures hostility, but a relatively specific form of hostility (Smith & Frohm, 1985). That is, it has been suggested that the label "cynical hostility" may be more appropriate for the scale, since high scorers are likely to view others with distrust, resentment, and suspiciousness, rather than being overtly aggressive in their behaviour. This interpretation

of the scale is supported by a recent factor analytic study which identified a major cynicism factor in the Ho scale (Costa, Zonderman, McCrae, & Williams, 1986).

The relationship between hostility and CHD also received some support from studies using the Buss-Durkee Hostility Inventory. This self-report scale is a 75-item true-false scale, designed to measure each of seven subtypes of hostility (i.e., physical assault, indirect hostility, irritability, negativism, resentment, suspicion, and verbal hostility). A global measure of hostility is also obtained with this scale (Buss & Durkee, 1957). It has been reported that two-week test-retest reliability for the global hostility score is .82, while the reliability for the seven subscales ranges from .64 to .78 . Validity data include positive correlations with other self-report hostility scales. For instance, this scale has been reported to be associated with the Ho scale, especially with the Resentment ($r = .70$) and Suspicion ($r = .69$) subscales (Smith & Frohm, 1985). Diamond et al., (1984) also reported an interaction between the Buss-Durkee and the SI, and with the potential for hostility from the SI, and their relationship with systolic blood pressure changes during a challenging laboratory task (i.e., high hostile subjects showed greater reactivity than low hostile subjects).

Further support for the association of hostility with CHD was found in two studies using the SI-based components

scoring technique developed by Dembroski and MacDougall (1985). This technique provides ratings on a number of content and stylistic dimensions, such as potential for hostility (PoHo), the tendency to suppress anger (anger-in), loud, rapid/accelerated, and explosive speech, and verbal competitiveness. The PoHo has been reported to correlate significantly ($r = .37$) with the Ho scale (Dembroski et al., 1985), though clearly the measures are not interchangeable. In the first study, based on the ratings of the SI of patients ($n = 131$) who underwent diagnostic coronary angiography, Dembroski et al. (1985), found that the dimensions PoHo and anger-in were better predictors of CHD than were any of the other components, including the overall Type A ratings. These associations were found even after statistical adjustment for age, sex, and the traditional risk factors. An interesting finding of the Dembroski et al. (1985) study was their observation of a positive relationship between PoHo ratings and CHD indices (i.e., the number of vessels occluded) only in those patients that had the tendency to suppress their anger. PoHo was not related to CHD in patients who reported expressing their anger. In the second study, MacDougall, Dembroski, Dimsdale, & Hackett (1985) also found in a sample of 125 patients referred for angiography, that PoHo and anger-in were the only dimensions (out of 12 rated Type A dimensions) to predict the number of vessels occluded 50% or more.

Thus, it appears that aspects of anger expression (i.e., anger-in) and hostility (i.e., cynical hostility) may be important for coronary risk. In light of the findings presented in this section with both healthy and at-risk samples, hostility and anger appear to be dimensions of the TABP that are strongly related to CHD. A similar conclusion has also been reached by two extensive reviews of the Type A literature (Manuck, Kaplan, & Matthews, 1986; Matthews & Haynes, 1986). Although it has been suggested that the hostility observed in at-risk samples may have developed as a result of CHD (and not vice versa) (Matthews & Haynes, 1986), the similar findings obtained with different types of samples (i.e., healthy and at-risk subjects) and measures suggest otherwise, i.e., that hostility and parameters of anger expression are strongly related to the development of CHD.

Finally, Wright (1988) has recently argued that time urgency and chronic activation are inadequately assessed by the usual TABP measures, and that these components may be important in the prediction of CHD. He defines time urgency as concern not of large amounts of time, but more often over a few seconds, and chronic activation as staying active or "keyed up" for most of the day, every day. Although this hypothesis still needs to be adequately tested, based on clinical observations and theoretical considerations, it is argued that hostility and anger may be related to time

urgency, i.e., because of the frustrations related with this continual time urgency, and/or because of this driven need to achieve (typical of Type A individuals) which is conducive to the experience of anger (Wright, 1988).

PHYSIOLOGICAL MECHANISMS OF CHD

Even though hostility and anger appear to be strongly related to CHD, may be even more than the global TABP, the mechanisms by which these Type A components contribute to the development of CHD is not well understood. Several physiological processes have been hypothesized to occur (Krantz & Manuck, 1984; Matthews & Haynes, 1986; Wright, 1988).

As discussed in a literature review completed by Krantz and Manuck (1984), coronary atherosclerosis is a condition characterized by the narrowing and deterioration of the coronary arteries, the blood vessels that nourish the heart. An excess accumulation of cholesterol and related lipids forms a mound of tissue, or plaque, on the inner wall of the artery. There is evidence suggesting that the initiating event involves injury to the inner wall of the coronary arteries, and that this injury can be produced by either mechanical (hemodynamic) or chemical injury, which in turn can be potentiated by cardiovascular or endocrine correlates of the sympathetic nervous system (Krantz & Manuck, 1984).

More specifically, hypertension and elevated HR are hypothesized to accelerate the atherosclerotic process by creating points of increased stress in the coronary arteries, and also turbulence in the blood flow, which over time can (mechanically) promote injury to the inner wall of the arteries (Krantz & Manuck, 1984).

There is considerable evidence to suggest that serum cholesterol contributes to the fibrous plaques which are characteristic of atherosclerosis (Troxler & Schwertner, 1985). More specifically, it has been shown that by lowering the level of serum cholesterol, there is a corresponding reduction in deaths due to CHD. The reduction of low density lipoprotein cholesterol (LDL) has also been found to reduce even more the mortality rate due to CHD, while levels of high density lipoprotein cholesterol (HDL; which promotes the efflux of cholesterol from the plasma) are in contrast inversely related to the development of CHD (Troxler & Schwertner, 1985).

Although there is evidence to suggest that stress is related to cholesterol elevations, there is also considerable variability in the level of support between studies reporting this relationship, and within individuals in these studies (Troxler & Schwertner, 1985). It has also been observed that the type and duration of the stressors may have a differential effect on either the total cholesterol level or

other lipoprotein fractions such as triglycerides (Van Doornen & Orlebeke, 1982). TABP has also been found to be associated with higher cholesterol level, i.e., several studies have observed that Type A individuals have higher (total) cholesterol levels than Type Bs (see Van Doornen & Orlebeke, 1982, for a review).

Catecholamines circulating in the blood have also been hypothesized to contribute to the lesions in the atherosclerotic arteries by increasing the lipid mobilization which are then deposited in arterial walls (Krantz & Manuck, 1984). Catecholamines (i.e., epinephrine and norepinephrine) are sympathetic nervous system hormones and neurotransmitters. Hence, it is suggested that injuries to the artery walls could be caused by hemodynamic effects such as elevated heart rate and blood pressure, and also by biochemical changes such as increased levels of catecholamines and serum cholesterol, which can be produced under prolonged or severe stress (Krantz & Manuck, 1984). Furthermore, the most popular hypothesis explaining the link between TABP and CHD has been that Type A individuals show more cardiovascular reactivity to various environmental events than Type Bs, and that this chronic heightened reactivity would predispose these individuals to greater coronary risk. As a result, measurement of physiological endpoints in relation to TABP has typically involved heart rate, systolic and diastolic blood pressure, levels of serum

cholesterol, HDL, and catecholamines (i.e., epinephrine and norepinephrine).

HOSTILITY, ANGER, AND PHYSIOLOGICAL REACTIVITY

In this section, an overview of the studies which have evaluated the relationship between hostility, anger, and CHD-physiologically relevant reactions will be presented.

In a study examining the effects of harassment and competition upon physiological reactivity in (SI-defined) Type A and B individuals (Glass, Krakoff, Contrada, Hilton, Kehoe, Manucci, Collins, Snow, & Elting, 1980), found that harassed Type As responded with the largest SBP, HR, and plasma epinephrine elevations, compared to nonharassed As and Bs, and to harassed Bs. In contrast, there were no significant differences in the elevations of DBP or plasma norepinephrine for either As or Bs. Glass et al. (1980) concluded that Type As were more physiologically aroused by competition with a hostile opponent than Type Bs. Dembroski, MacDougall, and Lushene (1979), have also reported findings suggesting that highly hostile and competitive (SI-defined) Type As perceive mildly challenging situations as more challenging than do low hostile Type As and Bs, and therefore respond with greater cardiovascular arousal (i.e., increased HR, SBP and DBP).

Similarly, Diamond, Schneiderman, Schwartz, Smith,

Vorp, and DeCarlo Pasin (1984), found that harassment in the context of competition elicited greater SBP amongst Type As than Bs. Their results indicated interesting differences between high and low hostile subjects (as measured by the Buss-Durkee Hostility Inventory). Amongst high-hostile subjects, overt anger expression, and SI-defined Type A behaviour, were both associated with increased reactivity (i.e., SBP). Furthermore, amongst low-hostile subjects, suppressed anger, Type B behaviour, and low SI-defined PoHo, were even more associated with reactivity. Diamond et al. (1984) concluded that hostility and Type A are both associated with increased physiological reactivity, as is suppressed anger amongst Type B individuals. These findings are consistent with Contrada et al.'s (1985) review which suggested that SBP is often associated with differences among SI-defined Type As and Bs, while DBP and HR bear a weak association with individual differences in physiological reactivity among Type As and Bs. Their review also suggests that the JAS is less sensitive than is the SI to the detection of differences in physiological reactivity.

This possibility that different pathophysiological mechanisms may be associated with divergent patterns of anger and hostility had also been suggested earlier by Diamond (1982). In his review of the literature, anger and hostility have emerged fairly consistently as salient psychological factors in essential hypertension. The literature appears to

support the view that at least a subset of hypertensives are chronically hostile, conflicted about anger expression, and tend to be overtly submissive and compliant while harboring considerable resentment.

Hence, hostility appears to be an important component of Type A behaviour that has been associated with cardiovascular arousal. However, although hostility and Type A appear to be associated with physiological responsiveness, it appears that only a subset of Type A individuals may demonstrate increased reactivity. That is, hostility in conjunction with anger appears to be related to greater reactivity amongst Type As, while suppressed anger has also been related to considerable reactivity amongst Type Bs.

It is important to note, however, that even though the hypothesis of increased (physiological) reactivity to stressors amongst hostile Type A individuals has received considerable support, it has not yet achieved the status of a proven risk factor (Krantz & Manuck, 1984).

TYPE A BEHAVIOUR, PROSTAGLANDINS, AND CHD

Another physiological measure of CHD to test the hypothesis of reactivity has recently been developed. Prostaglandins and associated biochemical processes have been identified as playing an important role in the development of atherosclerosis and CHD (Gerrard & Peterson, 1985).

Prostaglandins are hormone-like substances which are produced in response to cellular stress or injury, or in response to specific hormone action. That is, tissue injury releases arachidonic acid (i.e., a polyunsaturated fatty acid), the oxidation of which leads to prostaglandins. The two prostaglandins that are of interest to this study are thromboxane and prostacyclin. Thromboxane, which is produced by blood platelets, stimulates platelet aggregation and vasoconstriction. Prostacyclin, which is released from the vessel wall, blocks further platelet adhesion and aggregation to an injured vessel, stimulates vasodilation, and has an important role in preventing propagation of clots. During hemostasis, the balance of reactions (i.e., platelet adhesion, aggregation, and processes that limit the extent of the aggregation) is very critical. Failure of the aggregation mechanisms could lead to a bleeding disorder, while overactive clot formation could lead to a thrombotic disorder (Gerrard, 1985).

Recent evidence suggests that a disordered balance of thromboxane and prostacyclin may be implicated in the genesis of cardiovascular disease (Gerrard & Peterson, 1985). For instance, it has been shown that isolated vessel segments from animals with atherosclerosis produced less prostacyclin than similar vessel fragments taken from controls (Dembinska-Kiec, Gryglewska, Zmuda, & Gryglewski, 1977). Individuals

with atherosclerosis have also been found to have increased urinary prostacyclin and thromboxane metabolites than controls (FitzGerald, Smith, Pedersen, & Brash, 1984).

Furthermore, there is also evidence that alterations in the production of thromboxane and prostacyclin may be part of the vascular response to both emotional (Levine, Towel, Suarez, et al., 1985) and physical stressors (Mehta, Mehta, & Horalek, 1983; Neri Serneri, Gensini, Masotti, et al., 1986). Levine et al. (1985) reported a significant increase in circulating platelet aggregates in a sample of healthy medical residents undergoing the emotional stress of public speaking. This increase in platelet activation occurred in conjunction with increases in heart rate and catecholamines, suggesting an association between platelet activation and the cardiovascular events that are related to stress. Increases in thromboxane levels have also been reported following the cold pressor test (Neri Serneri et al., 1986), and as a result of (submaximal) physical exercise (Mehta et al., 1983). In both of these two studies, considerably greater increases of thromboxane were noted among CHD patients, and as a result of exercise stress, patients with CHD also exhibited lower increases of prostacyclin compared to normal subjects. As to the possible mechanisms for changes in platelet function during stress, there is reasonable evidence to suggest that chemicals known to undergo changes during stress (i.e., epinephrine and norepinephrine, cholesterol,

Von Willebrand factor) may mediate platelet activation (Gerrard & Peterson, 1985).

These findings then, provide yet another method of evaluating the reactivity hypothesis. That is, even though the role of these prostaglandins is as yet poorly understood, there is evidence to suggest that they may have a major role in cardiovascular disease. Therefore, it is reasonable to ask whether behaviour patterns influence the susceptibility of an individual to cardiovascular disease by altering the production of thromboxane and prostacyclin.

Preliminary work by our research group (Gerrard, Dyck, & Dion, 1988) corroborated earlier findings regarding the reactivity of prostaglandins to psychosocial stressors. That is, we recently examined the reactivity (as measured by the production of thromboxane A_2 and prostacyclin, BP, and HR) of (university male) subjects to (1) a relaxation session, to give a baseline reading, and (2) to the mild stress of the SI, which was used in addition to determine Type A or B behaviour (Gerrard, Dyck, & Dion, 1988). The results showed that in response to the mild stress of the interview (i.e., the SI), there was a significant difference between the Type As and Bs in their production of prostacyclin. Type A subjects showed a significantly lower production of prostacyclin than did Type B subjects, indicating that they are less protected by the actions of prostacyclin when

subjected to stress, and thus over time could become more at risk to develop atherosclerosis. Other preliminary studies in the laboratory of Dr. Gerrard have shown that these biochemicals are implicated in CHD. For instance, lower levels of prostacyclin have been observed among patients with CHD when compared to controls, providing support to the hypothesis that an imbalance between thromboxane A_2 and prostacyclin is implicated in the development of CHD. Preliminary work has also shown that thromboxane A_2 is sensitive to acute exercise, i.e., subjects showed significantly higher levels of thromboxane immediately after exercise (Carter, Gerrard, & Ready, 1989). It is not known at this point, however, if these changes in thromboxane would be different as a function of fitness level.

For these reasons, it is natural to inquire whether the observed relation between TABP and prostacyclin is altered by fitness level. The literature which bears on the hypothesis that physical fitness has physiological benefits will be reviewed in the following section.

PHYSICAL FITNESS AND PHYSIOLOGICAL BENEFITS

Repetitive endurance aerobic exercise such as walking, jogging, swimming, rowing, or bicycling, is the type of activity hypothesized to have a beneficial cardiovascular effect. Isometric or muscle-building exercises are believed

not to provide such benefits. When aerobic exercise is carried out at sufficient intensity, frequency, and duration, predictable cardiovascular, metabolic, and muscular changes occur that allow an individual to perform more work within the aerobic range (Rigotti, Thomas, & Leaf, 1983). It is these changes that are referred to as the "training effect". This training effect is achieved by aerobic exercise that is carried on for at least 20 min three times a week, at an intensity of 70% of maximal heart rate (i.e., maximal heart rate is estimated by subtracting the individual's age from 220) (Rigotti et al., 1983).

Several prospective studies, in which active and sedentary groups were observed over time for the development of CHD, were conducted since the early 1970s. One of these studies examined the role of job-related physical activity amongst a sample of 3000 San Francisco longshoremen over a period of 22 years (Brand, Paffenbarger, Sholtz, & Kampert, 1979). They reported a significantly lower risk of fatal myocardial infarction (MI) and sudden death in those men whose work activity required a heavy energy expenditure (i.e., over 8500 kilocalories (kcal) per week). This difference persisted after correction for seven CHD risk factors by multivariate analyses, indicating that the protective effect of activity was independent of traditional risk factors.

Paffenbarger and his colleagues also reported recently

a 12 to 16 years follow-up of over 15,000 Harvard alumni, assessing the influence of physical activity and other life-style characteristics on length of life (Paffenbarger, Hyde, Wing, & Hsich, 1986). The alumni reported by questionnaire how much walking, stair climbing, types of sports they participated in and time they spent on them each week. The energy used for these activities was then transformed in kcal per week. While controlling for blood-pressure status (e.g., hypertension), cigarette smoking, and body weight, they found that the risk of death became progressively lower as the physical activity level increased from below 500 to about 3500 kcal per week. Furthermore, men with an index of less than 2000 kcal per week had a 38% greater risk of death than men with a higher index. Or men whose weekly energy output in various activities and sports totaled 2000 or more kcal per week had a 28% lower all-cause death rate than less active men. Hence, an inverse relationship was again observed between physical activity and mortality rate.

Troxler and Schwertner (1985) also reported in their review of CHD risk factors that the literature supports the view that high density lipoprotein (HDL) cholesterol consistently increases during aerobic exercise (but not during anaerobic exercise). This is especially important since high HDL levels have been associated with lower risk of heart disease (Troxler & Schwertner, 1985).

Rigotti et al. (1983) reported in their extensive

review that data from Finland, Norway, England, and the American Cancer Society, also supported the protective role of exercise. Although the evidence discussed so far were derived from nonintervention studies, and as such do not establish a causal relationship, they nevertheless support the hypothesized relationship between physical inactivity and the development of CHD.

Other studies which have examined the effect of exercise training on survival in persons who have survived a first heart attack, were inconclusive (Franklin, Besseghini, & Golden, 1978; Rovario, Holmes, & Holmsten, 1984). However, the beneficial effects of exercise in the rehabilitation of cardiac patients have been demonstrated more clearly. It has been found that exercise training substantially increases the ability of these patients to tolerate physical activity (Franklin et al., 1978; Rovario et al., 1984). That is, following exercise training these patients are able to exercise for longer periods of time, and have lower heart rate and blood pressure during exercise. Hence, although it is not clear at this point that exercise has an effect on the progression of the atherosclerotic process, exercise has been shown to contribute to the cardiac rehabilitation of patients with established CHD (Rigotti et al., 1983).

The relationship between aerobic fitness and cardiovascular reactivity to psychological stress has also

been examined, since prolonged hyper-reactivity (i.e., increased heart rate (HR) and blood pressure) can lead to structural changes typical of hypertension (Steptoe, Melville, & Ross, 1984), which is in turn a CHD risk factor. This relationship was examined by Perkins, Dubbert, Martin, Faulstich, and Harris (1986), who compared the cardiovascular reactivity (as measured by systolic and diastolic blood pressure (SBP, DBP), and HR) to psychological stress (i.e., a video game, and a mental arithmetic task, both with monetary incentives based on performance) amongst three groups (i.e., aerobically trained mild hypertensives, untrained mild hypertensives, and untrained normotensives. Their results showed that the trained subgroup of hypertensives showed reduced cardiovascular reactivity compared to the untrained group of hypertensives, and similar reactivity as shown by the normotensives. Hence, the results indicate that aerobic training can be beneficial to mild hypertensives.

Keller and Seraganian (1984) also reported that physical fitness is associated with autonomic reactivity to psychological stress. In their first experiment, three groups of subjects' (i.e., a very fit group - marathon runners; an inactive group; and a group enrolled in an aerobic exercise program) autonomic reactivity (as measured through electrodermal activity) was compared in response to two (laboratory) stress tasks, i.e., the bolt head maze and the Stroop color-word test. Physical fitness was measured by

the recovery time needed for the HR to return to basal level following an up/down stepping task. Their results showed that the very fit group recovered significantly faster than the two other groups, and that by the end of their training program, the subjects in training recovered significantly faster than the inactive subjects. In regard to the electrodermal activity in response to the stress tasks, the very fit subjects showed significantly less electrodermal changes than did the two other groups, suggesting that they were less reactive to psychological stress. In a second and better controlled experiment, subjects were randomly assigned to one of three treatment conditions: aerobic conditioning, music appreciation, or Yoga meditation. The meditation condition was intended to serve as an expectancy control. The second set of results showed that the exercise group significantly improved in aerobic fitness level while the other two groups did not. Furthermore, once in the last stage of their exercise program, the exercise group responded to the tasks by showing significantly less electrodermal changes, i.e., they recovered faster than the two other groups. Hence, these experiments suggest that aerobic fitness is associated with more rapid autonomic recovery from psychosocial stress, and more generally that fit individuals may be more effective in coping with emotional stress than are less fit individuals (Keller & Seraganian, 1984).

Hull, Young, and Ziegler (1984), also designed an

experiment to determine whether physical fitness is related to lower reactivity to various stressors. Based on the degree of fitness, four groups were formed (aerobic fitness was defined as the length of time the subject could run until their HR was within 90% of their maximum HR); group 1 included basically sedentary subjects while group 4 was composed mostly of very fit runners (and the two other groups were in between with respect to fitness levels). The four stressors used were: 1) a film depicting industrial accidents, 2) performing the Stroop color-word test with conflicting auditory input for 5 minutes, 3) putting a foot in ice water for 1 min, and 4) exercising on the automated treadmill until exhausted. Following the stressors, various measures were taken, including JAS-defined Type A behaviour, a mood checklist, HR, BP, and blood sample. Their results showed that for the entire sample, aerobic fitness was associated with lower baseline HR. In addition, for subjects 40 years of age or older, fitness was associated with lower resting SBP, and lower DBP in response to the film and the Stroop task. Furthermore, only two fitness-related mood differences were noted, i.e., fit subjects were less angry and less depressed after the film than were less fit individuals. The only significant difference between Type A and Type B subjects was that Type As were more responsive (i.e., higher HR) than Type Bs on the Stroop task. Interestingly enough, TABP had no effect on BP. Hull et al.

(1984) suggested that aerobic fitness is not associated with lower JAS scores. Furthermore, in light of the significant bradycardia observed among very fit subjects, the lower norepinephrine (NE) at fixed loads of exercise and the rapid decline in NE levels after cessation of exercise, it is suggested that aerobically fit persons have greater metabolic efficiency.

Cardiovascular reactivity has also been examined amongst Type As and Type Bs, regardless of the physical fitness component. For instance, it has been shown that SI-defined Type A behaviour had increased cardiovascular responding (i.e., SBP) when performing a task, that is in the presence or absence of explicit incentives, while Type Bs only showed increased cardiovascular responding when provided with incentives (Blumenthal, Lane, Williams, McKee, Haney, & White, 1983). Hence, in this study Type As had increased cardiovascular response under all conditions, whereas Type Bs showed similar responding only when the opportunity for reward was presented.

Blumenthal et al.'s (1983) results seemed to be consistent with Contrada, Wright, and Glass' (1985) review paper, which shows that the SI assessment method is more consistently associated with reactivity (i.e., SBP and HR) than the JAS. This may explain the failure of Hull et al. (1984) to observe an association between physical fitness and

lower JAS. Hence, it is possible that if Hull et al. (1984) had used the SI (rather than the JAS) to classify the Type As, an interactive effect between physical fitness and TABP might have been observed.

In summary, prospective studies have shown an inverse relationship between (aerobic) physical activity and mortality rate, i.e., physical activity appears to lower the risk of developing CHD, MI, and sudden death. There is also evidence to suggest that aerobically fit individuals show reduced cardiovascular reactivity to various stressors, compared to sedentary individuals, as well as greater metabolic efficiency. With respect to the effects of aerobic fitness on patients with established CHD, it is unclear whether exercise has an effect on the progression of CHD, although benefits in the rehabilitation of these patients have been observed. Finally, preliminary work has shown that thromboxane is sensitive to acute exercise. It is not known, however, if these changes would differ with varying fitness level (Carter, Gerrard, & Ready, 1989).

In the following section, a growing body of literature documenting the relationship between physical fitness, mental health, and CHD, will be reviewed. This literature appears to be especially relevant to the findings discussed earlier, i.e., the relationship of the toxic components of Type A, namely hostility, anger, and time urgency, in the development

of CHD.

PHYSICAL FITNESS AND PSYCHOLOGICAL BENEFITS

Most individuals who exercise on a regular basis often report that it makes them "feel good" or "feel better". In the last decade, no one would dispute the increased interest in physical exercise. People are running by the thousands, organized (running) races are taking place everywhere, and exercise clubs offering a variety of programs are opening up at an amazing rate. This reflects in part the popular conviction that physical fitness has psychological as well as physiological benefits.

As reported in an extensive and critical review of the literature in this area, a considerable number of studies suggest that physical fitness training leads to improved mood, self-concept, and work behaviour (Folkins & Sime, 1981). However, Folkins and Sime (1981) pointed out that although quite suggestive, most of these studies were poorly designed, i.e., they had either no control group, or nonequivalent control groups which introduces problems such as selection bias. For example, Lobstein, Mosbacher, and Ishmail (1983), reported findings based on a comparison between physically active and sedentary middle-aged men on the depression scale of the MMPI. Even though their results suggested that physically active subjects were less depressed

than the inactive ones, these differences do not establish cause and effect; i.e., these differences might be related more to personality characteristics than to exercise, and/or they could have existed before these individuals were active. Hence, in order to obtain interpretable data, it is important that a control group be included, that it matches the experimental group, and that the expectations of the subjects be similar to the ones of the experimental group (Folkins & Sime, 1981).

In another study which evaluated the relationship between swimming and mood (Berger & Owen, 1983), it was reported that subjects who had participated in a 14 weeks swimming training program were significantly less tense/anxious, less depressed, and less hostile and angry (as measured by the Profile of Mood Survey, POMS), than controls at the end of the training programs. However, problems with the design of this study allow for other interpretations of the data. For instance, since these researchers did not verify that a training effect had occurred in the experimental condition, the mood changes cannot be unambiguously attributed to alterations in fitness level. Furthermore, since the groups were not matched for expectancies (i.e., subjects in the experimental condition possibly expected some benefits from participating in the training program, while the ones in the control group were not part of a condition that could lead to such

expectations). It cannot be argued that the training program of this study caused the improved mood. Other interpretations could include that the changes in mood were more related to expectations of the subjects who chose to enroll in the training program, or that the changes were related to the social context of the training group.

A more recent review of the literature (Simons, McGowan, Epstein, Kupfer, & Robertson, 1985) on the effects of exercise in the treatment of depression also reported methodological problems in most of the studies reviewed. The studies using clinical populations nevertheless all support the hypothesis that depression of clinical proportions can be treated with exercise. Simons et al.'s (1985) review provides grounds for cautious optimism. Some of the major methodological problems identified included studies with no control group, lack of powerful comparison treatment, no assessment of fitness changes, or a questionable method to assess the levels of fitness (i.e., using a maximal effort test where maximal oxygen consumption is estimated may not be appropriate with a clinical population since the results are highly dependent on motivation).

In light of the weak design of the majority of studies in this area, Goldwater and Collis (1985) examined the psychological effects of cardiovascular conditioning. In order to obtain better experimental control, (1) they had the

control group enrolled in an activity program, to control for factors such as emotional benefits resulting from the social support of the groups; (2) the control group exercise program had the appearance of a beneficial program, to control for the possibility that subjects might feel better because of their expectations of the benefits of conditioning programs; (3) and finally, subjects were randomly assigned to either group, in order to control for expectations that might be specific to those people concerned enough to enroll in conditioning programs. The experimental group had a strong emphasis on intense cardiovascular exercise (i.e., running, swimming, soccer), and lasted 6 weeks with 5 X 90 minute sessions per week. The program designed for the control group also lasted 6 weeks but was of much lesser intensity, i.e., two weekly sessions of 60 minutes, and included exercise such as stretching, marching, and volleyball. Their results showed the experimental group had a significantly greater (cardiovascular) training effect (as measured by a multistage continuous test that employs an 18-inch bench and submaximal work loads) than did the controls, and although not significant, the experimental subjects showed a greater decrease in anxiety and a greater improvement in self-reports of psychologic well-being. These results were interpreted as supporting the hypothesis that physical conditioning leads to improvement in psychological functioning. Although the results were in the expected direction, they were not

significant, thus weakening the interpretation. Hence, the basic assumption still awaits stronger evidence.

The mechanism(s) which link psychological benefits to exercise are not well understood. At this point, the literature supports several hypotheses (Morgan, 1985). The first one, the distraction hypothesis, maintains that distraction from stressful stimuli, as opposed to exercise per se, is responsible for the improved affect associated with exercise. This hypothesis is based on the work of Bahrke and Morgan (1978) which compared the effects of different conditions (i.e., exercise, meditation/relaxation, and distraction) on the reduction of anxiety. Their results showed that the three conditions seemed comparable in their anti-anxiety properties. The literature does suggest, however, that anxiety reduction following exercise persists for a longer period of time than that observed following distraction (Morgan, 1985).

The second hypothesis (i.e., the monamine hypothesis), maintains that neurotransmitters such as norepinephrine and serotonin (which are implicated in depression and schizophrenia, respectively) are increased in the brain as a result of chronic exercise, which would lead to improved mood (Morgan, 1985). Although tenable, this hypothesis is based mostly on small animal research, and still needs to be tested with humans.

Another biochemical hypothesis, i.e., the endorphin

Another biochemical hypothesis, i.e., the endorphin hypothesis, maintains that the actions of the various exercise-induced endorphins produced in the brain can be "morphine-like" in that they have the ability to reduce the sensation of pain and produce a state of euphoria (Morgan, 1985). However, this hypothesis is highly equivocal at this time. It is of course possible that these three hypotheses have interrelated mechanisms involved in the association of exercise with improved mood.

Cognitive explanations for the relationship of exercise and mood have also been suggested. For instance, Bandura's self-efficacy theory (1977) maintains that treatments are effective because they create a sense of self-efficacy by arranging self-mastery experiences. On this hypothesis, exercise may achieve an antidepressant effect because it engenders a perception of skill mastery or increased self-efficacy expectations. Although this hypothesis is plausible, it still needs to be directly tested (Simons et al., 1985).

Hence, although the strength of the association between exercise and mood is modest at this point, it has consistently been observed. Furthermore, although the mechanism(s) underlying this association are still not clearly understood, the hypotheses that have been put forward are reasonable and testable.

MODIFYING TYPE A BEHAVIOUR

Several attempts have been made to alter risk factors for CHD in Type A individuals. The effects of psychologically based interventions and those based on physical activity will be reviewed separately.

Psychological Intervention. Suinn and Bloom (1978) developed an anxiety management training program which was shown to lower anxiety as well as scores on the JAS scale. Roskies, Spevack, Surkis, Cohen, and Gilman (1978) reported reductions in SBP, serum cholesterol, and several psychological symptoms in a group of healthy, middle-aged male volunteers who underwent either brief group psychotherapy or group behaviour therapy for 14 weekly sessions. Follow-up data, however, showed superior results for subjects receiving group behaviour therapy, possibly due to their practicing specific tension-reducing exercises (Roskies et al., 1978).

More recently, Gill, Friedman, Ulmer, and Drews (1985), also reported a significant reduction in the TABP (SI-defined) of healthy military officers following the completion of an 8 month group behaviour counseling program. The counseling program focused on 1) cognitive restructuring; 2) instruction in self-recognition of the causes underlying the emotional reactions to various emotional challenges; and 3) advice on how to avoid or modify potentially stressful environmental situations. Interestingly enough, their

control group (composed of Type A individuals) also showed significant reduction in the TABP, but to a lesser extent, suggesting poor experimental control. More specifically, although they reported that the subjects exercised approximately for the same number of hours per week, they failed to investigate the type of exercise that the subjects participated in (i.e., aerobic vs. anaerobic), and/or to directly assess aerobic fitness level. In light of the influence of physical fitness on physiological reactivity, Gill et al. (1985) might have improved their experimental control if such variables would have been controlled.

In light of estimates which suggest that between 50 and 75% of the general population may be Type A (Rosenman, 1974), the cost-effectiveness of preventative programs such as those mentioned above is needless to say questionable.

Physical Activity Intervention. The use of aerobic physical activity as a preventative approach to CHD has received growing attention. To date, few studies have examined whether an exercise program or different levels of aerobic fitness could be effective in modifying the risk profile of Type A individuals.

The first study to examine the effects of exercise on JAS-defined Type A behaviour pattern was conducted by Blumenthal, Williams, Williams, and Wallace (1980). A sample of 46 middle-aged healthy adults (males and females), with a

distribution of 21 Type As and 25 Type Bs, completed a fitness program (i.e., walking/jogging). The program consisted of 10 consecutive weeks, with three sessions of 30-45 min per week. The JAS and several physiological measures were taken before and after the completion of the training program. They reported that their training program significantly reduced the coronary risk profile of Type A individuals. It significantly affected physiologic variables (i.e., HR, SBP, DBP, HDL, and plasminogen activator release in response to venous occlusion), and also reduced the magnitude of the TABP. Hence, this study does appear to demonstrate that (aerobic) physical exercise can reduce the coronary risk profile of Type As. Unfortunately, the design of this study is such that their results are inconclusive. An initial flaw in the design was that subjects were not randomly selected nor randomly assigned to the experimental conditions, i.e., the sample consisted of adults who had registered for a fitness program. Hence, the design did not control for a possible self-selection bias (i.e., differences between individuals who choose to exercise and the ones that didn't). Furthermore, a control group was not included, which makes it impossible to rule out variables such as passage of time, expectancy, and other variables as responsible for the benefits associated with exercise. Finally, in light of recent evidence which suggests that the JAS does not have adequate reliability (Landy et al., 1988),

and that it has a weak association with physiological reactivity (Contrada et al., 1985), one may question the conclusion reached by Blumenthal et al. (1980), regarding the reduction of the magnitude of TABP. The effects of their training program on physiological variables appear, however, to be consistent with the literature reviewed in the preceding section.

A second and more recent study reported on a Type A intervention project (Roskies et al., 1986). A sample of 107 healthy Type A male office managers (identified from a larger sample of 425 volunteers) were randomly assigned to one of three treatment conditions: 1) an aerobic exercise condition which consisted of three sessions of 20-25 min of jogging per week; 2) a cognitive-behavioral stress management condition which consisted of 2 sessions per week where individuals were taught to perceive and respond to potential stressors with newly acquired coping skills; and 3) a weight-training condition which consisted of two training sessions per week. The latter group served as a control group. The three treatment conditions were delivered in a group format and for 10 weeks. Treatment efficacy was measured by changes in behavioral (i.e., as measured by the SI), and physiological reactivity (i.e., in response to a series of laboratory psychosocial stress tasks such as an arithmetic quiz, the Stroop color-word test, etc.). Cardiovascular responses measured were HR, SBP, and DBP. Their results were that the

cognitive-behavioral stress management program was the only condition to significantly reduce behavioral reactivity as measured by the SI (i.e., both a reduction of the global Type A and of all its components). The aerobic and weight-training conditions did not reduce significantly the behavioral reactivity. Furthermore, none of the conditions were successful in producing significant changes in physiological reactivity. As a result, Roskies et al. (1986) concluded that a short-term stress management program delivered in a group format may be the most promising preventative program for Type As in relation to CHD.

In view of the literature which indicates that aerobic exercise reduces the risk factors for CHD, and also reduces psychological symptoms such as depression, anxiety, hostility, and anger, Roskies et al.'s (1986) results on the ineffectiveness of the aerobic exercise condition are surprising. The study reports that a training effect was produced by the aerobic condition but not by the two other conditions (based on a significant increase of the estimated maximal oxygen uptake, and a significant decrease of basal HR). However, the results also indicate that the difference between the decreases of HR for the aerobic and weight-training groups is only 1.9 beats per min, i.e., the difference between the pre-post HR means for the aerobic group was 5.8 beats per min, while the difference for the weight-training group was 3.9. While this was a

statistically reliable difference, the training effect in the aerobic group is not large. Hence, it is possible that Roskies and her colleagues may not have substantially altered physical fitness levels, which may have compromised the effectiveness of the fitness treatment condition on behaviour type.

Finally, another study examined the effects of TABP (SI-defined) and aerobic fitness on HR and BP changes elicited by laboratory challenges (Lake, Suarez, Schneiderman, and Tocci, 1985). Aerobic fitness was indexed by VO_2max (i.e., maximum oxygen uptake) values. VO_2max is considered to be the most reliable index of overall cardiovascular work capacity (Simonson, 1972). Subjects with high VO_2max values are able to utilize more oxygen than subjects with low values, and likewise have more efficient cardiovascular systems (Fox & Matthews, 1981). In this study, VO_2max values were estimated on the basis of the recovery HR scores of a step test. This procedure was used to classify subjects into two groups, i.e., fit versus sedentary. The two groups were significantly different in terms of baseline DBP, HR, and mean arterial BP. The laboratory challenges included a mental arithmetic task, a task of courage (presentation of a live snake), a cold pressor task, and a card game task that involved harassment from the card dealer. Their results showed that in general Type A subjects responded with greater reactivity (i.e., in

SBP and DBP) than did the Type Bs. Furthermore, sedentary Type As reacted with greater BP reactivity than either Type As or Bs or sedentary Bs during the SI. During the challenging verbal exchange of the card game, the reactivity of fit Type As did not differ from those of the sedentary or fit Type Bs. Lake et al. (1985) pointed out that these last results may indicate that aerobic fitness may be protective for Type As. Finally, the snake presentation, mental arithmetic, and cold pressor conditions failed to elicit A-B differences. Lake et al.'s (1985) results also indicate that Type As were more reactive than were Type Bs during the SI, with sedentary Type As showing the greatest amount of reactivity. Unfortunately, Lake et al. (1985) failed to statistically control for baseline differences in HR and DBP that were due to fitness, which renders their conclusions tentative at this point. Further, they did not examine the components of the TABP that are suspected of being coronary-prone, in relation to aerobic fitness. This would have been especially important in light of recent evidence which suggests that the global Type A measure is composed of both toxic and protective elements (Williams & Barefoot, 1987).

THE PRESENT RESEARCH

As discussed in the preceding sections, several large scale prospective studies have shown that Type A behaviour is a risk factor for coronary heart disease. Some recent

studies, however, failed to support the hypothesis that the TABP is associated with CHD, suggesting that the TABP hypothesis is less empirically robust than initially believed (Williams & Barefoot, 1987). Finally, recent meta-analyses of the Type A literature concluded that the SI-defined TABP is a reliable predictor of initial CHD events (Friedman & Booth-Kewley, 1988; Matthews, 1988). Further, it has been suggested that mostly those aspects concerned with hostility, anger, and time urgency, are coronary-prone behaviour, and they have yet to be adequately measured (Wright, 1988).

In contrast to the detrimental effects of the TABP, there is some support for the hypothesis that aerobic conditioning is associated with both physiological and psychological benefits, which may be protective against CHD. Furthermore, preliminary work has suggested that there is a relationship both between TABP and prostaglandins implicated in the development of CHD, and physical activity and prostaglandins. However, the interactive effects of TABP and exercise on prostaglandins have never been evaluated. Hence, the primary purpose of this research is to examine the relationship between (aerobic) physical fitness, the TABP and its toxic components (i.e., hostility, anger expression, and time urgency), and prostaglandin responses, in particular, prostacyclin and thromboxane. An extensive review of the literature shows that such a study has not been conducted to date.

More specifically, this study was designed to answer the following question:

Is the TABP-CHD relationship, such as measured primarily by platelet responses, affected by aerobic fitness?

Prostacyclin and thromboxane were chosen as the primary physiological measures because of evidence suggesting their role in the development of CHD, and their sensitivity to stress. The responses of these measures were also of primary interest because they have never been examined as a function of Type A behaviour (other than in our pilot study) and fitness level. Finally, blood platelets may provide an important biochemical link in the TABP-CHD relationship.

Cardiovascular measures (i.e., HR, SBP, DBP) were also included as secondary measures, to examine their relationship with prostacyclin and thromboxane, and to provide indices of cardiovascular reactivity and recovery to a psychosocial stressor. The cardiovascular measures were of secondary interest because they have been the most often used physiological endpoints in previous studies of Type A behaviour.

A quasi-experimental research design was adopted in this study, i.e., although fitness level and TABP were not experimentally manipulated, the stress level of subjects was manipulated both by a fitness test and by a psychosocial

stressor. In the first phase of this study, the fitness level of subjects was assessed, as well as the production of thromboxane and prostacyclin both under resting conditions and in response to exercise. During the second phase of the experiment, subjects were again assessed during baseline and following exposure to a challenging psychosocial stressor.

This study was undertaken to test hypothesized relationships between fitness, the TABP and its toxic components, and their physiological concomitants. A descriptive approach appeared most appropriate at this time to investigate to which extent variations in fitness level are associated with variations in the Type A components and CHD endpoints (i.e., can variations in fitness level predict variations in both the psychological and physiological variables?). Although causality generally cannot be established with such a design (i.e., mostly because not all the variables being examined are under experimental control), it has however, the potential to identify the path of causality (Grosof & Sardy, 1985). That is, the identification of functional relationships between variables may be the first step before designing a study whose goal would be to identify causal relationships.

This study used the Structured Interview (SI) for university students (Rosenman, 1978) to assess behaviour type. In addition, recently developed psychometric

instruments were used to measure anger expression (Spielberger, Johnson, Russell, Crane, Jacobs, & Worden, 1985), and time urgency (Wright, 1988). Hostility was measured by the MMPI-based Ho scale (Cook & Medley, 1954). The physiological concomitants of TABP included the measurement of the bleeding time blood chemicals thromboxane A_2 and prostacyclin, SBP, DBP, and HR. Fitness level was measured through a graded treadmill test until exhaustion.

Based on the literature reviewed in the preceding sections, the following predictions/hypotheses were made:

1. Under resting conditions, fit Type As will show lower (values of) HR, SBP, DBP, higher level of prostacyclin, lower level of thromboxane, and longer bleeding time (BT), than less fit Type As and Type Bs.

2. Under conditions of psychosocial stress, fit Type As will be less physiologically reactive (i.e., lower increases in HR, SBP, DBP, higher increases of prostacyclin, lower increases of thromboxane, and longer BT) than less fit Type As and Type Bs.

3. Following the psychosocial stressor, the recovery of the fit Type As (measured in terms of HR, SBP, DBP) will occur at a faster rate than the recovery of the less fit Type As and Type Bs.

4. More fit individuals will obtain lower scores on the measures of the toxic components of TABP (i.e., hostility, anger, and time urgency), than less fit individuals. This prediction is based on the literature suggesting that aerobic fitness has an attenuating effect on negative mood states.

METHOD

Subjects

Male students, between the ages of 18 and 28, were recruited from the Introductory Psychology Course at the University of Manitoba. Only subjects who were non-smokers, who had not taken aspirin two weeks prior to the experiment (aspirin inhibits the production of thromboxane and prostacyclin), who had no known heart trouble, normal blood pressure, and who had not been advised to avoid strenuous physical exercise, were recruited. The final sample consisted of 97 subjects (mean age=20).

For the purpose of comparing the effect of different fitness levels on physiological and psychological measures, two groups with respect to fitness (i.e., aerobically fit and unfit) were arbitrarily defined. Further, to ensure that these groups were initially (approximately) equally represented, the final sample was randomly selected from a group of 500 (male) subjects based on their level of self-reported physical activity (see Lifestyle Questionnaire, Appendix A). Based on this questionnaire, half of the subjects were recruited for the group defined as "aerobically fit", i.e., individuals who participate in cardiovascular activities at least three times a week, for 30 min or more on each occasion, and at medium to heavy intensity. The other half of the sample was recruited for the "unfit" group, which

was defined as individuals who participate in few (if any) aerobic activities at light intensity. It should be noted that this preliminary fitness categorization which used the above mentioned self-report measure was done only for recruitment purposes. The final fitness categorization was done by using VO_2max values, such as explained in the following section.

All subjects received partial course credit for their participation in the experiment, in addition to $20.00 (i.e., $5.00 per bleeding time).

Apparatus and Measures

Type classification. The SI for university students (Rosenman, 1978; see Appendix B) was used to assess behaviour type. The interviews were conducted by this investigator, who was trained by Dr. Dyck, who was in turn trained by Rosenman and associates.

The student form of the SI consists of 22 questions with competitive, hostile, and time urgent themes. Both the content of answers, and the form of responding (e.g., vocal intonation, response latency) formed the basis upon which behavioral categorization is made. The interviews were tape recorded and subsequently rated by the investigator, who was trained to rate interviews by Dr. Dyck. Behavioral categorization was made according to the scoring system

developed by Rosenman and associates (Rosenman, 1978). Even though the initial scoring system of the SI suggested that individuals could be classified into one of four categories (i.e., from extreme Type A to extreme Type B), this study rated subjects as either Type A or Type B. This decision stemmed from the difficulty to differentiate between two levels of Type A or two levels of Type B, i.e., the subjectivity involved in using four categories with this sample would have reduced considerably the reliability of categorization.

Interrater reliability was based on a subsample of 20 subjects which were randomly selected and independently rated by both Dr. Dyck and the investigator. Interrater agreement was 90%, and exceeded chance level ($k=.89$, $z=.30$, $p<.01$; Cohen,1960).

Type A components. As discussed in the previous sections, three components of the TABP have been hypothesized to be associated with coronary-proneness, i.e., hostility, anger expression, and time urgency.

Hostility refers to a cynical attitudinal set and was measured here as elsewhere (Williams, Barefoot, & Shekelle, 1985) by the MMPI Ho scale (a 50-item scale; see Appendix C), developed by Cook and Medley (1954). As discussed earlier, this measure has been shown to have adequate validity and test-retest reliability.

In addition to hostility, high ratings of anger-in (i.e., as assessed by the SI) have been significantly associated with angiographically documented severity of coronary atherosclerosis (Dembroski et al., 1985). Although in this latter study anger expression (i.e., anger-in vs. anger-out) was based on the component scoring of the SI, the current research used the measure for the expression of anger (AX; see Appendix D) recently developed by Spielberger et al. (1985). The AX is a 24-item scale which has subscales measuring suppressed anger (anger-in) and expressed anger (anger-out). The psychometric properties and evidence of the validity of the Ax scale are discussed in Spielberger et al. (1985). In summary, they reported that the internal consistency of the scale is adequate (the alphas for the two subscales for males and females ranged from .73 to .84). Evidence of the concurrent and construct validity of the scale was found by comparing the responses of high school students (n=1114) to both the Ax scale and vignettes that required the students to report how they would feel in these various anger-provoking situations. These comparisons (ANOVA'S) indicated that students classified as "anger out" on the basis of their responses on the vignettes had significantly higher anger-out scores on the Ax scale, and significantly lower anger-in scores on the Ax scale, than students classified as "anger-in". Evidence of the convergent and divergent validity of the Ax scale are also

reported in the form of significant correlations with other anger and personality measures (see Spielberger et al., 1985).

A measure of the time urgency construct recently developed by Wright (1988; see Appendix E), was used in this study. This measure consists of two subscales, that is the Time Urgency scale (TU; 32 items) and the Perpetual Activation scale (PA; 105 items). The TU measures a concern not of large amounts of time, but more often over a few seconds, while the PA measures a state of chronic activation where an individual stays active or "keyed up" for most of the day, every day. Although the JAS has been purported to measure a similar construct with its speed/impatience subscale, two factor analytic studies conducted by Landy et al. (1988) showed poor test-retest reliability (i.e., between .30 and .50) of this factor, and low internal consistency (i.e., alpha coefficients below .50). It should be noted, however, that Wright's measure is still at the experimental stage and was used here in an exploratory fashion. Prior to its use, the reliability (i.e., item analysis) of the scale was assessed with the initial sample of 500 subjects, which revealed Cronbach's alpha of .74 for the PA scale, and .92 for the TU scale. In light of the adequate internal consistency of these two scales, this measure was included in the study, while keeping in mind that it is still at the experimental stage.

Psychosocial Stressor. The Stroop Color Word Test was used as a stressor to elicit reactivity. This test is a mental task that consists of presenting visually color words printed in incongruous colors, one per second. The subject is required to name the color of the print, and not the word itself. In addition to the time pressure, conflicting color words were simultaneously heard over the monitor. The stimuli for this test had been pre-taped on a video cassette recorder (Panasonic VHS), and were presented on a Panasonic 14" color video monitor. This task has been used in a number of studies to elicit reactivity (Hull et al., 1984; Keller & Seraganian, 1984; Roskies et al., 1986), and has been shown to consistently elicit reactivity (i.e., as measured by HR, SBP, DBP, NE, and E) in Type A individuals (Seraganian et al., 1985). In light of previous studies that have shown that Type A-B differences in physiological response are most pronounced under challenging environmental circumstances (Krantz & Glass, 1984), challenging instructions were included prior to the stressor (see Procedure).

Measurement of Thromboxane A_2 and Prostacyclin. This research utilized techniques developed by Dr. J.M. Gerrard in his laboratory for measuring prostacyclin and thromboxane production (a measure of the in vivo production in response to a standard vascular injury, the bleeding time). The bleeding time blood samples were analyzed for the stable metabolites of thromboxane (i.e., thromboxane B_2) and

prostacyclin (i.e., 6-keto-PGF_1). The bleeding time blood was collected by medical technicians, and analyses were conducted in Dr. Gerrard's laboratory at the University of Manitoba Health Sciences Centre. (Refer to Appendix F for a detailed description of the bleeding time procedure and analyses).

The bleeding time procedure was adopted by this study to measure thromboxane and prostacyclin, first because these chemicals are generally produced in response to specific stimuli (i.e., most often from a vessel wall injury), and second because the bleeding time blood reflects the production of these chemicals in response to a single standardized vascular injury (Gerrard et al., 1989). Previous studies have measured these prostaglandins through urine or plasma samples, measurements that may be difficult to interpret because they reflect total body production (i.e., which could include more than one injury), and not just the vascular production from a single injury site (Gerrard, 1989). While the bleeding time in itself provides an overall assessment of the effectiveness of the hemostatic system (Schottelius & Schottelius, 1973), "measurement of the quantity of thromboxane and prostacyclin produced in response to a specific and standardized vascular injury has advantages in measuring in vivo capability for de novo production" (Gerrard, 1989).

Measurement of VO_2max. Maximal oxygen uptake (i.e., VO_2max) was measured through a graded treadmill test until exhaustion. This fitness test was chosen mainly because it provides the most accurate (aerobic) fitness value through direct measurement of oxygen uptake at a maximum performance (as opposed to protocols that predict VO_2max from submaximal tests, which have a standard error of estimate of at least 15%) (Astrand, 1976). The treadmill (Quinton treadmill model Q65) speed remained constant at 7 m.p.h. with an initial 0% grade. The treadmill was programmed to then increase the grade by 2% every 2 min. This is an accepted protocol for testing healthy males below age 35 (Thoden, Wilson, & MacDougall, 1982). Heart rate was monitored continually throughout the entire test with a Cambridge electrocardiograph (model VS4). Finally, gas analysis was conducted during the last 30 sec of each stage with a Beckman gas analyzer (MMC Horizon System, model Sensormedics). That is, oxygen uptake measures were collected in the last 30 sec of every 2 min stage, and the test was terminated when the values reached maximum (level off) or when the subject could no longer continue. The highest level of oxygen uptake is called the maximal oxygen uptake (VO_2max), and is expressed in milliliters of oxygen per kilogram of body weight per minute. Although the measurement of VO_2 was made automatically with the Beckman on-line microprocessed system, it should be noted that VO_2 is based on the rate at which air

is expired and on the fractions of oxygen and carbon dioxide in expired air. Thus, because the fraction of oxygen in inspired air (0.2093) is known, VO_2 represents the difference between the inspired and expired volume flow rates, and the difference between inspired and expired concentration of oxygen (Holly, 1988). Fitness testing was conducted by qualified technicians at the Sport and Exercise Sciences Research Institute of the University of Manitoba.

The criterion used to classify individuals as fit or unfit was taken from previous studies of average male college students (Sharkey, 1979). Individuals with a VO_2max greater than 47 were considered fit, while subjects with a value lower than 47 were placed in the unfit group. Although this criterion did not ensure that the groups had equal number of subjects, it was felt to be more meaningful than arbitrarily using the median as a cut off point.

Measurement of Heart Rate and Blood Pressure. Heart rate and blood pressure were recorded with a Critikon Dinamap Vital Signs Monitor (model 1846 SX). This unit was programmed to automatically inflate and deflate the blood pressure cuff and to provide a digital readout (using the oscillometric method) of systolic and diastolic blood pressure (mmHg), and heart rate (beats per min), at regular intervals. The calibration of the Monitor was checked with the use of a mercury manometer on several occasions during the experiment, and the pressure was found to be well within

the recommended tolerance for different pressure levels.

Procedure

Subjects were asked to participate in a research project investigating the relationship between fitness, personality traits, and certain chemicals that are produced in the blood. The experiment consisted of 2 sessions, taking place a week apart. Except for the completion of the Ho scale, the Ax, and the Time Urgency scale, which were completed in group sessions several weeks prior to the experiment, subjects went through the entire procedure on an individual basis.

During the first session, subjects first completed a physical activities screening questionnaire, and then signed the consent forms (see Appendix G). The screening questionnaire called the PAR-Q, is a validated instrument which was designed to identify those individuals for whom certain physical activities might be inappropriate, in particular those who suffer from cardio-pulmonary diseases or other ailments which could be aggravated by exercise testing (British Columbia, 1975). The PAR-Q was used to determine the appropriateness of subjects for fitness testing. Subjects were then fitted with the blood pressure cuff (i.e., wrapped on the upper left arm with its sensor positioned over the brachial artery), and asked to stay seated and relax for

5 min, at which time baseline levels of HR, DBP, SBP, thromboxane and prostacyclin were taken. Fitness level was then assessed according to the procedure described above. At the end of the fitness test, each subject went through a "warm down" period (i.e., walking on the treadmill for 2 min to ease the subject into near normal blood flow), after which measurement of thromboxane and prostacyclin was again taken in order to assess the possible effect of acute exercise on these chemicals.

During the second session, which took place the following week, baseline levels of HR, DBP, SBP, thromboxane, and prostacyclin were taken following the same procedure as during the first session. Subjects then underwent the SI which was used to classify behaviour type. After a 5 min rest period, they were then exposed to the psychosocial stressor, i.e., the Stroop color-word test. The following recorded instructions were given over the monitor prior to the stressor in order to challenge the subject:

> You are now going to be presented with a task that measures your ability to think and act quickly and accurately. This task has been selected on the basis of its ability to predict academic success at the university level. Your answers will be recorded to allow for the evaluation of your performance.
>
> Color words will quickly be flashed on the monitor one at the time. Each color word is printed in different colors. Your task is to say the color of the print and not the word itself (2 examples were given). In addition you will be hearing at the same time other color names on the monitor; try to ignore these and only name the color of the print. If you make a mistake during the task, do not go back to

> correct yourself, simply continue. Remember to try as hard as you can, and try not to quit. Pay close attention as the words will be flashed very quickly. Do you have any questions? The color-word task will now begin!

Measures of HR, DBP, and SBP, were recorded at 1-min intervals during both the test and during the 4 min recovery period following the test (the recovery period time is based on previous work (Roskies et al., 1986) which shows that 4 min is long enough for most subjects to return to baseline after such a stressor). Measurement of thromboxane and prostacyclin were taken immediately after the stressor, to evaluate the influence of the stressor on these prostaglandins. The measures of HR, SBP, and DBP, taken during the stressor are indicative of the level of reactivity, while these same measures taken during the 4 min period that followed the stressor provide information on the cardiovascular recovery of the subjects.

In the present study, the order of the two sessions was held constant for all subjects. Although counterbalancing is generally used to control for possible effects due to the order of presentation of the stimulus or conditions (Grosof & Sardy, 1985), a preliminary study (Gerrard, Dyck, & Dion, 1988) suggested that subjects experienced anticipatory stress on the first bleeding time, which may have masked the influence of the variables of interest. Hence, since for this study the second session was

the one designed to test the major hypotheses, the order was kept constant to reduce the effect of anticipatory stress for the second session.

Data Analysis

The raw data for all subjects were entered into computer files for data management, and statistical analyses were conducted with the Statistical Package for the Social Sciences (SPSSx) as well as Statistical Analyses Systems (SAS).

Data reduction was required for the physiological scores of HR, SBP, and DBP, because of the multiplicity of measurements. For instance, there was 1 baseline measure, 4 recordings taken at 1-min intervals during the stress task, and 4 recordings taken at 1-min intervals during the poststressor recovery period. For this study, three scores were of particular interest: a baseline score, a peak score, and a recovery score.

The peak score represented the intensity of arousal, and was defined as the difference between a subject's baseline score on a given response parameter and the highest value achieved in response to the stressor (i.e., variations from baseline were used instead of absolute values). This approach was adopted in order to better capture exaggerated physiological reactivity. That is, since exaggerated

reactivity is generally of short duration (i.e., one or two minutes at most; Seraganian et al., 1985), it was felt that the effect of such reactivity would be better captured with the peak score (i.e., highest) than by averaging the four scores taken during the entire stressor. For thromboxane and prostacyclin, since there was only a single sample drawn at the end of the stressor, reactivity was determined by subtracting the sample taken at the end of the stressor from the baseline score.

The recovery score reflected the duration of arousal, i.e., the mean deviation from baseline following the cessation of the psychosocial stressor. The recovery score was defined as the difference between baseline and the mean of the four recovery points. Only the measures of HR, DBP, and SBP, were used to provide information on the recovery period.

The calculation of change scores from baseline is an approach that has received considerable attention in psychophysiological research. The main reason for this attention is due to the possible (non-linear) relationship between the pre and post stimulus level for a given physiological function. That is, the Law of Initial Values (LIV) introduced by Wilder (1931; see Levey, 1980) states that the amount of change exhibited by a psychophysiological measure in response to a stressor is negatively related to

the initial level of that measure. That is, individuals with lower resting levels should show greater increases in response to a stressor than individuals starting at a higher level of arousal. To assess whether the LIV was operating for the measures taken in this study, the correlations (for the physiological measures) between change scores and their respective baseline were calculated. As shown in Table 1, these correlations were all significant and negative. Though significant, in 4 of the 6 cases the coefficients were below .4, indicating that only a small percentage of the variance was accounted for by these variables and their baselines, thus suggesting that the LIV was not clearly operating for these measures. However, in the cases of thromboxane and prostacyclin, the higher correlations tended to suggest that the LIV was operating. Hence, when change scores (i.e., peak score or recovery score) for thromboxane and prostacyclin were entered in various analyses, their respective baselines were also entered (as covariate) in the analyses to control for the baseline effect. The use of covariance techniques with physiological data where the LIV is thought to be operating, is seen as appropriate (Levey, 1980). Personal communication with statistical consultants has also confirmed that such an approach is appropriate.

Preliminary examination of the data also revealed that thromboxane and especially prostacyclin values were not normally distributed. The distributions for both measures

TABLE 1

Correlations Between Reactivity Scores and Baselines

Measures	PeakHR	PeakDBP	PeakSBP	PeakTXB	PeakPGI	PeakBT
Baselines						
HR	-.26**	.04	.04	-.05	-.07	.10
DBP	-.10	-.35***	-.07	-.20*	-.04	.16
SBP	-.06	.01	-.26**	-.06	.04	.03
TXB	-.10	-.11	-.04	-.66***	.20*	.01
PGI	.27**	.10	.02	-.03	-.46***	.09
BT	.04	.09	-.03	.05	.02	-.38***

* p<.05 ** p<.01 *** p<.001

Notes. HR=heart rate, DBP=diastolic blood pressure, SBP=systolic blood pressure, TXB=thromboxane, PGI=prostacyclin, BT=bleeding time, Peak=change score, i.e., the highest score's variation from baseline.

were positively skewed (i.e., a preponderance of low scores). Data transformation techniques were therefore applied to determine whether the distribution could be normalized. It was found that although logarithmic transformations improved the distributions of these measures sufficiently to satisfy the assumption of normality, the transformation was also found to change the relationship among the means. Therefore, it was decided not to transform the data in this study, mostly because it appeared to distort the original observations. This position appears to be consistent with the statistical literature which cautions against data transformation in general, and especially when using the analysis of variance because of the known robustness of the F test (Keppel, 1973; Kirk, 1968; Tabachnick & Fidell, 1983).

Finally, multivariate analyses of variance (MANOVAs) were used to compare various groups (e.g., fit vs. unfit; type A vs. Type B; hostile vs. non-hostile; etc.) for physiological differences at rest, during exercise and the psychosocial stressor, and during the recovery from the psychosocial stressor. Repeated measures analyses of variance were also used to compare these groups across the two baselines (i.e., was baseline 1 different from baseline 2 for various groups), and to assess the effectiveness of the psychosocial stressor. Box's M test was used to evaluate the homogeneity of variance, while Tukey tests were used for post-hoc comparisons, with alpha set at the .05 level.

RESULTS

The results will be presented according to the predictions made at the outset of the study. The first set of results will examine the impact of individual difference variables on physiological measures taken at baseline, i.e., prior to any manipulation. The second set of results will examine differences in physiological reactivity during the psychosocial stressor and the fitness test. Finally, the results pertaining to the recovery period that followed the psychosocial stressor will be presented. Although the results of both session 1 and 2 will be presented, the emphasis will be placed on the results of session 2 mostly because the latter was designed to test the major hypotheses, and the anticipatory stress of the subjects is expected to be lower than at session 1.

Preliminary descriptive analyses examined the sample as a function of behaviour type, the relationship between the Type A measure and its components, and the intercorrelations between the physiological measures at baseline. As shown in Table 2, for this sample the distribution according to behaviour type was 53 Type A subjects and 44 Type Bs. No significant differences were noted between Type As and Bs, other than the Type As showing marginally higher DBP than the Type B subjects.

TABLE 2

Characteristics of the Type A and Type B Subjects

Groups	Type A		Type B		p*
Characteristics					
n of subjects	53		44		
mean VO_2max	50.2	(7.3)	49.3	(5.8)	NS
resting HR	68.7	(13.6)	69.3	(9.9)	NS
resting SBP	128.6	(12.0)	131.1	(12.7)	NS
resting DBP	66.9	(8.5)	64.0	(8.0)	.09

Notes. VO_2max=maximal oxygen uptake, HR=heart rate, SBP=systolic blood pressure, DBP=diastolic blood pressure.

Abbreviations: ( )=standard deviation, NS=not significant.

* Two-tailed t tests

Table 3 presents the intercorrelations between the Type A components and the global Type A rating (note that Type A was scored as "2", and Type B as "1"). As expected and reported in the literature, time urgency and hostility were found to be correlated with Type A behaviour. Further, the inhibited expression of anger (anger-in) which has been hypothesized to be a toxic component, was found to be correlated with hostility, but not with global Type A. On the other hand, the self-reported tendency to express anger (anger-out) was significantly associated with hostility, time urgency, perpetual activation, and global Type A. Taken together these results suggest that the Type As in the present sample suppressed less their anger, and were more time urgent and hostile, than were the Type Bs.

Table 4 presents the intercorrelations between the physiological measures taken at the baseline of session 2. As expected, most of the cardiovascular measures were positively correlated with one another. In addition, the observation that HR and DBP were negatively correlated with bleeding time was consistent with earlier suggestions that increased HR and DBP may be associated with platelet activation (Gerrard & Peterson, 1985). Finally, that thromboxane was also negatively associated with bleeding time has been reported previously by Gerrard et al. (1989), and is consistent with the platelet aggregation actions of thromboxane.

TABLE 3

Intercorrelations Between the Type A Components

Measures	PA	TU	HO	AXIN	AXOUT	TYPE A
PA	1.00	.54***	.27**	.02	.26**	.25**
TU		1.00	.33**	.16	.30**	.34**
HO			1.00	.29**	.51***	.20*
AXIN				1.00	.24**	.11
AXOUT					1.00	.27**
Type A						1.00

* p<.05 ** p<.01 *** p<.001

Global Type A=2 Global Type B=1

Notes. PA=perpetual activation, TU=time urgency, HO=hostility, AXIN=anger in, AXOUT=anger-out.

TABLE 4

Intercorrelations Between The Physiological Measures at Baseline of Session 2

Measures	HR	DBP	SBP	TXB	PGI	BT
HR	1.00	.37***	.13	.05	.06	-.24*
DBP		1.00	.35***	.10	.16	-.23*
SBP			1.00	-.01	.11	.15
TXB				1.00	.02	-.28**
PGI					1.00	-.17
BT						1.00

* p<.05 ** p<.01 *** p<.001

Notes. HR=heart rate, DBP=diastolic blood pressure, SBP=systolic blood pressure, TXB=thromboxane, PGI=prostacyclin, BT=bleeding time.

Table 5 presents the intercorrelations between the (physiological) reactivity scores (i.e., change scores). As expected, the cardiovascular measures are all positively correlated with one another. Increases in diastolic blood pressure were also found to be moderately associated with increases in thromboxane level.

Fitness categorization was based on the VO_2max values of the subjects. Although subjects were not retested for fitness, the fact that 93% of them performed at their maximum heart rate (195-205 bpm), suggests that the values represent an accurate and reliable estimate of fitness level. Table 6 summarizes the descriptive characteristics of the fit and unfit groups. Among the differences between the two groups, the fit group had a significantly lower heart rate [$F(1,95)=4.12$, $p<.05$] than did the unfit group, as well as significantly lower diastolic blood pressure [$F(1,95)=4.35$, $p<.05$].

TABLE 5

Intercorrelations Between Reactivity Scores

Measures	PeakHR	PeakDBP	PeakSBP	PeakTXB	PeakPGI	PeakBT
PeakHR	1.00	.26**	.53***	.19	-.12	.01
PeakDBP		1.00	.46***	.22*	-.10	-.13
PeakSBP			1.00	.12	-.02	-.09
PeakTXB				1.00	-.09	.13
PeakPGI					1.00	.02
PeakBT						1.00

* p<.05 ** p<.01 *** p<.001

Notes. HR=heart rate, DBP=diastolic blood pressure, SBP=systolic blood pressure, TXB=thromboxane, PGI=prostacyclin, BT=bleeding time, Peak=change score, i.e., the highest score's variation from baseline.

TABLE 6

Characteristics of the Fitness Groups at Session 2

GROUPS	FIT	UNFIT	p*
Characteristics			
n of subjects	61	36	
mean VO_2max	53.9 (4.3)	42.8 (3.0)	
range of VO_2max	47.2 - 69.1	32.7 - 46.7	
resting HR	67.1 (11.4)	72.1 (12.5)	.05
resting SBP	128.6 (10.8)	131.7 (14.4)	NS
resting DBP	64.2 (7.1)	67.8 (9.7)	.05

Notes. VO_2max=maximal oxygen uptake, HR=heart rate, SBP=systolic blood pressure, DBP=diastolic blood pressure.

Abbreviations: standard deviation in parentheses; NS, not significant.

* Two-tailed t tests

Individual Differences at Resting State

Session 1. Multivariate analysis of variance (MANOVA) was used to compare various groups (e.g., fit vs. unfit; Type A vs. Type B) for physiological differences while at resting state. This analysis revealed a significant main effect for fitness [$F(6,88)=2.70$, $p<.02$]. As shown in Table 7, the differences observed included fit individuals having lower SBP than unfit subjects [$F(1,93)=3.82$, $p<.05$], and fit subjects having lower levels of thromboxane than subjects in the unfit group [$F(1,93)=5.74$, $p<.02$]. None of the interaction terms were found to be significant.

Session 2. MANOVA was again used to compare different groups for physiological differences while at resting state at the beginning of session 2. This analysis revealed a significant interaction between behaviour type and fitness [$F(6,86)=3.28$, $p<.01$]. The measures that reflected this significant interaction were heart rate [$F(1,91)=8.18$, $p<.01$] and thromboxane [$F(1,91)=8.57$, $p<.004$]. Tukey post-hoc tests indicated that unfit Type A subjects had higher heart rate than did both fit Type A ($p<.01$) and Type B subjects ($p<.05$). Similarly, unfit Type As had higher levels of thromboxane than did both fit Type As and Bs ($p<.05$) and unfit Type Bs ($p<.01$) (see Table 8). It was also noted that unfit Type As had marginally shorter bleeding times than did the other groups ($p<.12$), which is consistent with the higher level of thromboxane observed for that group.

TABLE 7

Baseline Physiological Values at Session 1

	FIT A (n=35)		UNFIT A (n=18)		FIT B (n=26)		UNFIT B (n=18)	
Measure	M	SD	M	SD	M	SD	M	SD
HR	69.1	11.2	73.9	11.7	69.6	8.9	70.3	7.9
SBP	121.3	11.0	130.2	16.6	124.0	12.6	125.8	13.3
DBP	69.4	9.5	72.4	11.9	68.9	8.9	72.9	12.2
TXB	3.2	2.2	5.1	3.3	3.0	2.6	3.8	2.8
PGI	2.6	2.2	2.0	1.2	2.1	1.8	2.5	2.0
BT	4.2	1.5	3.8	0.6	4.5	2.3	3.8	1.1

Notes. HR=heart rate, SBP=systolic blood pressure, DBP=diastolic blood pressure, TXB=thromboxane, PGI=prostacyclin, BT=bleeding time.

TABLE 8

Baseline Physiological Values at Session 2

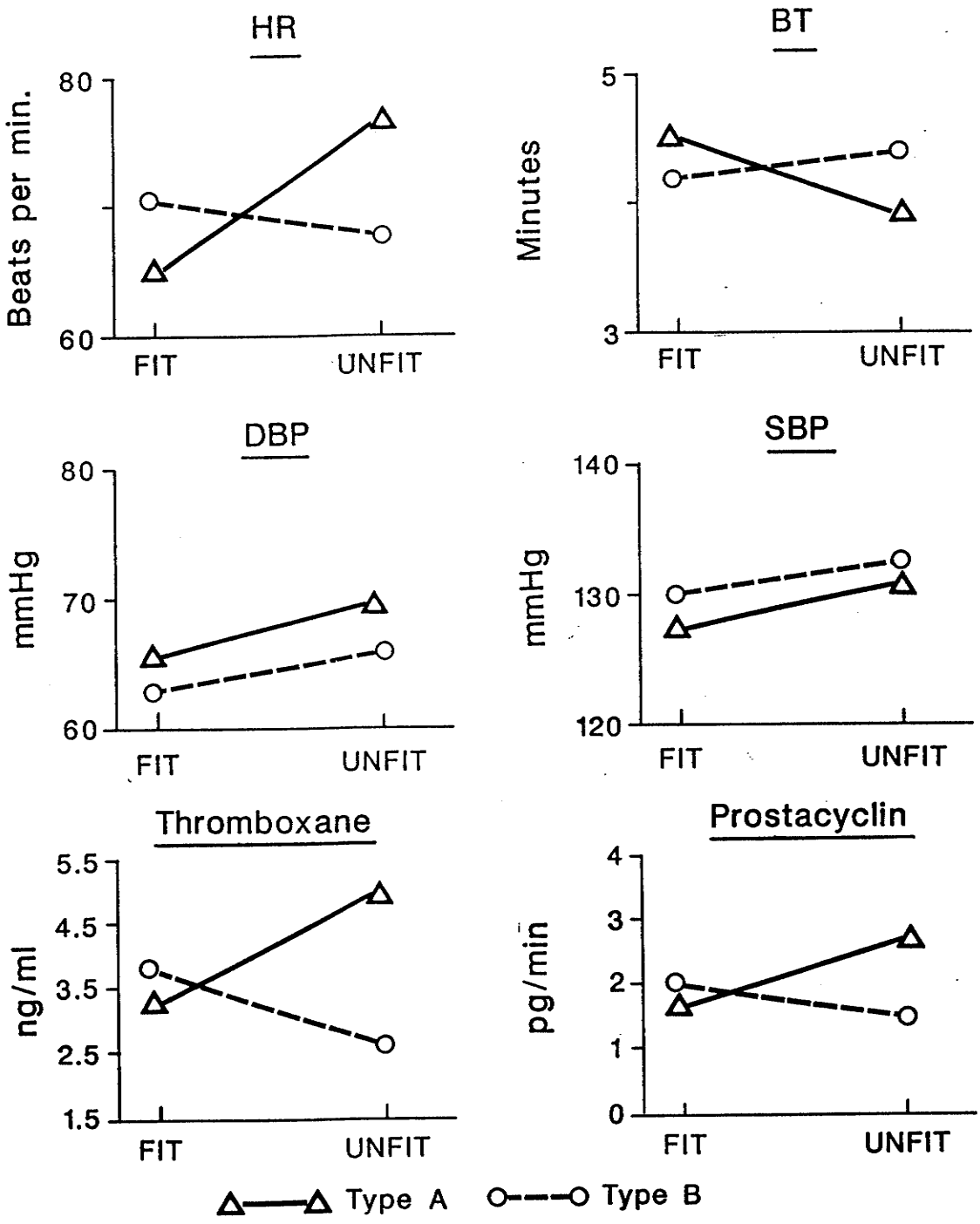
	FIT A (n=35)		UNFIT A (n=18)		FIT B (n=25)		UNFIT B (n=17)	
Measure	M	SD	M	SD	M	SD	M	SD
HR	64.7	11.5	76.4	14.4	70.0	10.9	67.6	8.7
SBP	127.5	11.5	130.7	12.9	129.4	9.7	131.0	14.7
DBP	65.3	7.0	70.0	10.2	62.8	7.3	65.1	8.9
TXB	3.3	2.4	5.1	2.9	3.8	2.1	2.7	1.6
PGI	1.7	1.9	2.7	3.0	2.0	1.3	1.4	1.7
BT	4.5	1.5	3.9	0.9	4.2	1.2	4.4	1.1

Notes. HR=heart rate, SBP=systolic blood pressure, DBP=diastolic blood pressure, TXB=thromboxane, PGI=prostacyclin, BT=bleeding time.

Despite some differences between the two sessions, in general it appears that both a lack of fitness and Type A behaviour were associated with increased levels of thromboxane. Further, as expected, increased fitness was also associated with lower heart rate and systolic blood pressure. These results generally confirm the predictions made at the outset of this study with respect to fitness level being inversely related to the magnitude of physiological risk factors and its interaction with TABP. Figure 1 depicts the relationship of fitness and behaviour type with the physiological endpoints examined during baseline at session 2.

Comparisons between Session 1 and Session 2. The order of the sessions was held constant in this study to reduce the potential anticipatory stress engendered by the first exposure to the bleeding time procedure. Because different methods were used at session 1 and 2 to measure heart rate and blood pressure (i.e., the auscultatory method was used at session 1, while the Vital Signs Monitor was used at session 2), it was not possible to compare the baseline measures of HR, SBP, and DBP, across sessions. However, the same procedure was used to measure thromboxane and prostacyclin in both sessions. To evaluate the effect of session on the prostaglandins, repeated measures analyses of variance were used.

Fig. 1: Individual Differences at Resting State (SESSION 2).



Effects of fitness level and behaviour type on baseline heart rate (HR), diastolic (DBP) and systolic (SBP) blood pressure, thromboxane, prostacyclin, and bleeding time (BT).

These analyses revealed no significant changes in thromboxane levels, while a significant interaction was observed between Time, Behaviour Type, and Fitness, for the measure of prostacyclin [$F(1,91)=6.10$, $p<.02$]. Tukey post-hoc tests revealed that the Fit Type A subjects and the Unfit Type Bs showed a significant decrease in prostacyclin at session 2 ($p<.05$). The theoretical significance of this interaction is, however, obscure.

Individual Differences under Conditions of Stress

The Stroop Color Word Test was found to be effective in eliciting cardiovascular arousal for the entire sample. That is, using absolute values, repeated measures analyses of variance revealed that the entire sample showed an increase in heart rate [$F(1,89)=353.71$, $p<.0001$], in diastolic blood pressure [$F(1,84)=223.68$, $p<.0001$], and in systolic blood pressure [$F(1,84)=215.38$, $p<.0001$].

Since there were no differences at baseline for SBP and BT, MANOVA was used for these measures to compare various groups for physiological differences as a result of their reactivity (i.e., changes from baseline) to the stressor. This analysis revealed a marginally significant main effect for behaviour type [$F(2,82)=2.75$, $p<.07$]. It was found that Type A subjects showed greater reactivity than did Type Bs with respect to SBP [$F(1,83)=5.16$, $p<.03$] (see Table 9).

TABLE 9

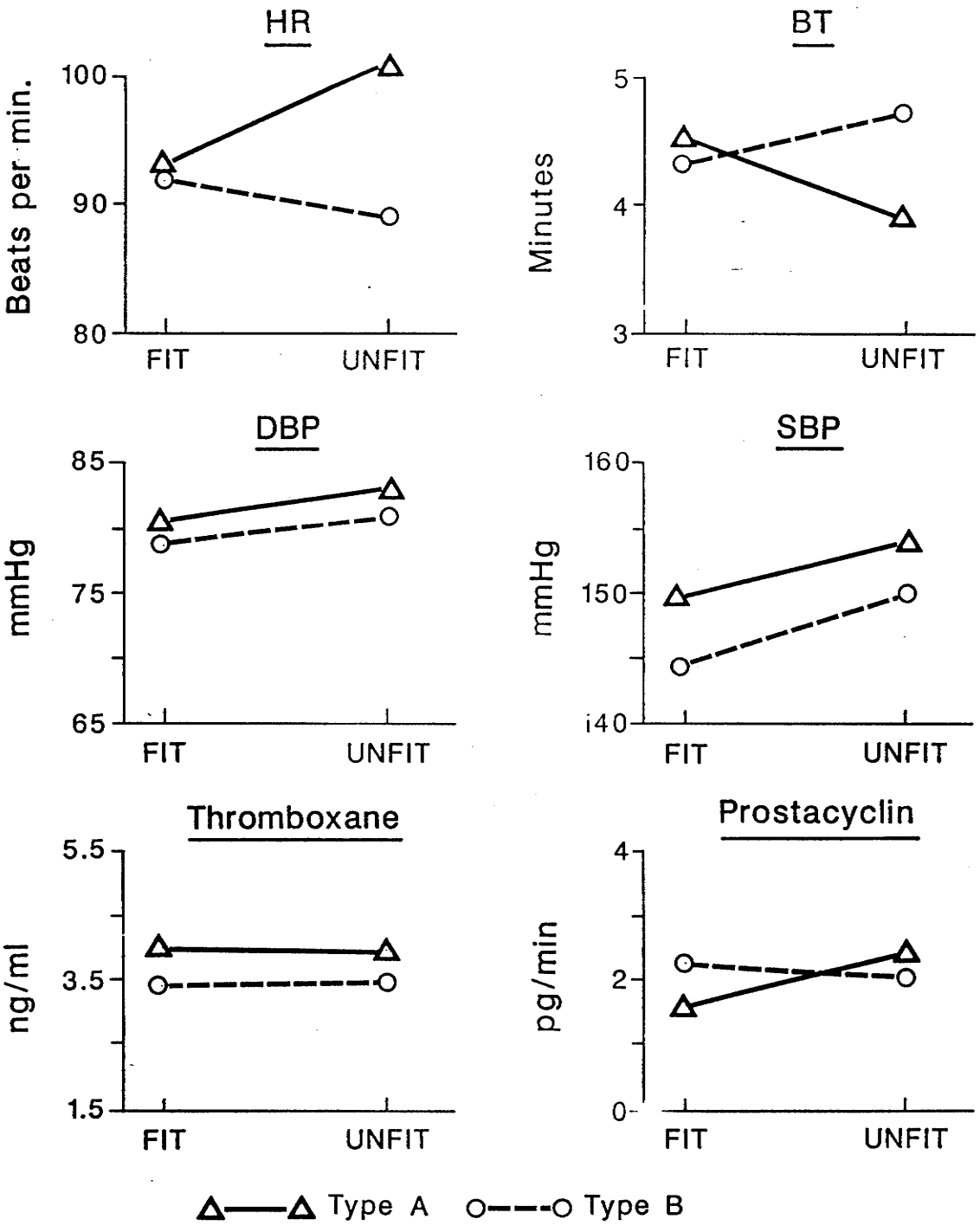
Reactivity Scores
(changes from baseline)

	FIT A (n=32)		UNFIT A (n=16)		FIT B (n=23)		UNFIT B (n=16)	
Measure	M	SD	M	SD	M	SD	M	SD
HR	28.5	12.4	27.1	16.6	21.7	9.7	19.8	7.1
SBP	21.3	12.1	22.1	12.4	13.7	10.8	18.2	8.9
DBP	16.5	9.5	14.5	12.6	15.9	7.3	15.8	6.1
TXB	1.1	2.3	-1.5	2.9	-0.6	2.2	0.8	1.9
PGI	-0.2	2.1	-0.2	2.5	0.1	1.4	0.6	1.9
BT	0.2	1.2	-0.1	0.6	0.2	1.2	0.2	0.6

Notes. HR=heart rate, SBP=systolic blood pressure, DBP=diastolic blood pressure, TXB=thromboxane, PGI=prostacyclin, BT=bleeding time.

With respect to HR, DBP, thromboxane and prostacyclin, covariates were used in the group comparisons because of differences at baseline for HR and DBP, and the LIV operating for the prostaglandins. MANCOVA was not used for these comparisons because it was found to reduce the cells' sample size well below the number required to ensure multivariate normality (see Tabachnick & Fidell, 1983). As a result, separate ANCOVAs were conducted for the above mentioned comparisons. These analyses revealed that Type A subjects showed greater reactivity than did Type B individuals with respect to heart rate [$F(1,88)=5.53$, $p<.02$]. A marginally significant interaction was also found between behaviour type and fitness for thromboxane [$F(1,90)=3.50$, $p<.06$]. As shown in Figure 1 and 2, this interaction was due to the changes in level of thromboxane in different directions for the different groups. That is, despite no significant differences in the absolute values of the level of thromboxane for the different groups following the stressor (see Figure 2), there were significant differences between the change scores as a result of a decrease for the unfit Type As as compared to the increase of the unfit Type Bs ($p<.05$). Finally, even though the stress related changes in bleeding times were not significant, ANOVA comparing the absolute values of bleeding times after stress (see Fig. 2) revealed that the unfit Type As had significantly shorter bleeding times than did the unfit Type B subjects [$F(1,92)=3.81$, $p<.05$].

Fig. 2: Individual Differences under Conditions of Stress.



Effects of fitness level and behaviour type on heart rate (HR), diastolic (DBP) and systolic (SBP) blood pressure, thromboxane, prostacyclin, and bleeding time (BT), under conditions of psychosocial stress.

Hence, the predictions made earlier are only partially supported by these results. That is, as predicted Type As did show increased reactivity with respect to HR and SBP when compared to Type Bs. On the other hand, the prediction related to the positive effect of fitness with respect to reactivity was not supported. Fitness failed to alter the response (i.e., reactivity) to the psychosocial stressor in either cardiovascular functions or prostaglandin responses. Furthermore, the differences observed in thromboxane production at resting state were not seen immediately following the stressor.

Individual Differences in the Post-Stressor Recovery

Group comparisons with respect to the cardiovascular recovery of subjects immediately following the stressor were made in order to assess the possible effect of fitness and/or behaviour type upon recovery. MANCOVA was used to compare cardiovascular functions for different groups during recovery (i.e., using change scores that reflect variation from baseline after the stressor) to control for fitness related baseline differences in both heart rate and diastolic blood pressure. This analysis revealed a significant main effect for behaviour type [$F(2,90)=4.93$, $p<.01$], where it was found that Type As recovered at a much slower rate than Type Bs in terms of heart rate [$F(1,91)=7.75$, $p<.01$] (see Table 10).

TABLE 10

Recovery Scores
(distance from baseline after the stressor)

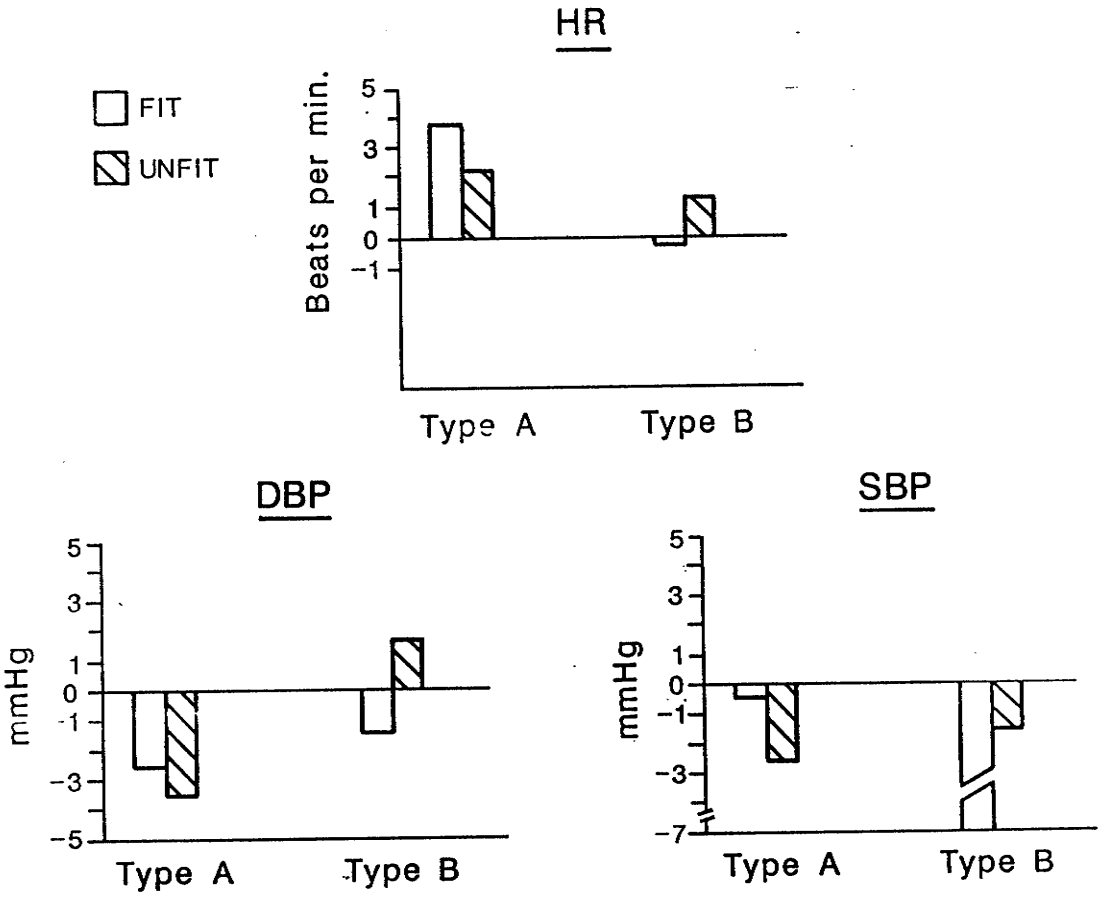
	FIT A (n=35)		UNFIT A (n=18)		FIT B (n=26)		UNFIT B (n=18)	
Measure	M	SD	M	SD	M	SD	M	SD
HR	3.8	5.7	2.3	7.8	-0.3	4.6	1.2	5.0
SBP	-0.5	10.1	-2.6	11.0	-7.0	9.2	-1.7	9.3
DBP	-2.8	8.9	-3.8	7.5	-1.6	6.1	1.7	5.4

Notes. HR=heart rate, SBP=systolic blood pressure, DBP=diastolic blood pressure.

With respect to blood pressure, however, the recovery data may be more difficult to interpret. That is, as shown in Figure 3, both DBP and SBP values were lower at recovery than at baseline for most groups. Repeated measures ANOVA was used to assess whether the lower recovery values were significant. These analyses revealed that the DBP of Type A subjects at recovery was significantly lower than at baseline [$F(1,93)=4.64$, $p<.03$], while the SBP of all subjects was found to be lower at recovery [$F(1,93)=8.24$, $p<.01$]. In light of these results, it may be difficult to interpret the blood pressure recovery data. In taking into consideration only the HR recovery data, however, the predictions made earlier were again only partially supported, i.e., behaviour type was shown to affect the recovery of subjects after the stressor, while fitness failed to alter the recovery.

In light of the variability in aerobic fitness within both the fit and unfit groups, correlational analyses were performed to further assess the effect of fitness on cardiovascular recovery. These analyses examined within each level of fitness whether changes in fitness (i.e., VO_2max) were associated with changes in cardiovascular recovery. Separate analyses were done for the recovery at 2 min, and at 4 min, after the psychosocial stressor. These analyses failed to reveal any significant findings. Hence, greater fitness was not found to be associated with faster cardiovascular recovery from stress.

Fig. 3: Individual Differences in the Recovery Following the Psychosocial Stressor.



Effects of fitness level and behaviour type on heart rate (HR), diastolic (DBP) and systolic (SBP) blood pressure, during post-stressor recovery.

Type A Components, Fitness, and Physiological Responding

The Type A components that have been hypothesized to be "toxic" in their association with CHD, i.e., hostility, suppressed anger, and time urgency, were not found to be associated in any way with the physiological measures (i.e., MANOVA failed to reveal any significant effect). Comparisons of the Type A components as a function of behaviour type and fitness revealed, however, a significant main effect for behaviour type [$F(5,83)=2.71$, $p<.03$]. That is, Type A subjects were marginally more hostile [$F(1,87)=3.14$, $p<.07$], more apt to express their anger outwardly [$F(1,87)=7.80$, $p<.01$], more time urgent [$F(1,87)=8.22$, $p<.01$], and showed higher perpetual activation [$F(1,87)=4.81$, $p<.03$], than did the Type B subjects (see Table 11). These results suggest that hostility, anger, and time urgency, are components of the TABP, but in light of their nonassociation with the physiological measures, they may not be independently "toxic", as it had been predicted. Further, the failure to find an association between the Type A components and fitness is contrary to the prediction made earlier with respect to fitness having an attenuating effect on Type A behaviour and negative mood states.

TABLE 11

Means of Type A Components

	FIT A (n=35)		UNFIT A (n=18)		FIT B (n=26)		UNFIT B (n=18)	
Measure	M	SD	M	SD	M	SD	M	SD
HO	26.9	6.7	26.3	6.5	25.2	5.6	22.8	7.0
AXIN	17.9	4.1	20.1	4.2	17.1	3.8	18.2	5.5
AXOUT	16.5	4.2	16.7	3.7	14.8	3.1	13.9	3.0
TU	296.7	29.8	288.1	17.1	272.8	24.4	279.6	26.2
PA	78.3	8.1	77.4	6.2	73.8	7.2	74.6	7.6

Notes. HO=hostility, AXIN=anger in, AXOUT=anger out, TU=time urgency, PA=perpetual activation.

Effects of Exercise on Prostaglandins

Using repeated measures ANOVA, levels of both thromboxane and prostacyclin, as well as the bleeding times, taken before and after the fitness test were compared. These analyses revealed a marginal increase in thromboxane levels after exercise for all subjects [$F(1,90)=2.87$, $p<.09$], as well as significantly shorter bleeding times for the fit subjects when compared to the unfit ones after exercise [$F(1,90)=5.47$, $p<.02$]. A slight increase in prostacyclin for all subjects was also noted after exercise, but this was not found to be significant.

DISCUSSION

The major result of this study was that Type A behaviour and aerobic fitness have an interactive effect on cardiovascular functions and the production of thromboxane. At rest, less fit Type A individuals had higher levels of thromboxane and higher heart rate compared to more fit Type As and Type Bs. This finding clearly supports the predictions made at the outset of this study, and contradicts a previous report which failed to find an association between aerobic fitness and behaviour type with respect to cardiovascular functions (Hull et al., 1984). A possible reason for the differences between the results of Hull et al. (1984) and those seen here is that in the present study a more precise and reliable measurement of fitness was used. The present study measured the maximal rate of oxygen (VO_2max) of subjects through direct gas analysis using a graded treadmill test until exhaustion, while Hull et al. (1984) estimated the VO_2max from a submaximal test. The protocol used by this study provides the most reliable marker of maximal aerobic power with a test-retest reliability of 0.95 (Holly, 1988), while estimated VO_2max values from submaximal exercise test have a standard error of estimate of at least 15% (Astrand, 1976). In any event, the present results strongly suggest that aerobic fitness has a protective effect among Type A individuals with respect to both cardiovascular functions and prostaglandin responses.

Possible Mechanisms for Individual Differences at Resting State

With respect to the group heart rate differences, the present results suggest that the sedentary Type As had higher resting heart rate compared to fit Type As and Type Bs. This interpretation seems consistent with one hypothesized link between TABP and CHD, i.e., chronic heightened cardiovascular reactivity (Krantz & Manuck, 1984), especially among the lesser fit subjects. Regular aerobic exercise is known to affect central cardiac functions, and to reduce heart rate and blood pressure at rest and at submaximal exercise levels (Clausen, 1977). These effects of fitness were clearly observed among the fit Type As of this sample.

The higher level of thromboxane observed among the sedentary Type As relative to other groups, should be interpreted in light of evidence which suggests that both physical (e.g., blood pressure and heart rate) and chemical (e.g., plasma lipids, catecholamines) factors are implicated in the development of atherosclerosis (Herd, 1984). The pattern of results is such that the higher thromboxane level among unfit Type As was found in conjunction with higher heart rate for that group, as well as marginally shorter bleeding time. Although not significant, the shorter bleeding time appears to be consistent with the higher level of thromboxane (and its aggregating action), which is also

consistent with previous reports (Gerrard et al., 1989). Further, as reported earlier, significant negative correlations were observed between the bleeding time and heart rate and diastolic blood pressure, which appears consistent with the suggestion that increased heart rate and blood pressure increase platelet activation via their mechanical influence (i.e., flow turbulence) (Gerrard & Peterson, 1985). On the assumption that the bleeding time provides an assessment of the overall hemostatic system, the (marginally) shorter bleeding time of the unfit Type As suggests a higher risk status. Hence, taken together, the higher heart rate, higher diastolic blood pressure, higher thromboxane level, and shorter bleeding time, indicates that at resting state the more sedentary Type As present with higher cardiovascular risk status.

Physiological Responsiveness to Stress

The second major set of findings of this study is related to the responses to the psychosocial stressor. Type A subjects were found to respond to the stressor with greater increases in systolic blood pressure and heart rate than their Type B counterparts. These differences remained significant even after statistically controlling for the baseline differences due to fitness. The A-B differences that were observed in this study confirmed the predictions made earlier, and are also consistent with previous reports

that have consistently shown the heightened cardiovascular reactivity of Type As to challenging stressors (Dembroski et al., 1979; Diamond et al., 1984; Glass et al., 1979; Hull et al., 1984; Krantz & Manuck, 1984; Matthews & Haynes, 1986; Williams & Barefoot, 1987). A number of studies have also shown that A-B differences in physiological response are most pronounced under challenging environmental circumstances (Krantz & Glass, 1984). This literature also suggests that Type A behaviour may be a style of coping with stressful events that threaten the individual's sense of environmental control (Glass, 1977). On this view, the competitiveness, hostility, and time urgency exhibited by Type A individuals reflects an effort to assert and maintain mastery over uncontrollable events. The initial reaction of Type As to such stressors would be one of behavioral hyper-responsiveness. In this study, the instructions given before the psychosocial stressor were designed to challenge the subject, and clearly they succeeded.

Contrary to the predictions made earlier, however, aerobic fitness was not found to moderate reactivity to the stressor. This association between behaviour type and aerobic fitness has received little attention to date. For instance, two studies failed to find an association between fitness and both JAS and SI-defined Type A behaviour in response to the Stroop task (Hull et al., 1984; Roskies et al., 1986), while Lake et al. (1985) found that sedentary Type As responded

with greater increases in blood pressure and heart rate to the SI than did fit Type As and Type Bs. Unfortunately, this latter study used a submaximal fitness test, and also failed to control for baseline differences due to fitness. Hence, it is not clear if Lake et al.'s (1985) reported interaction between behaviour type and fitness is in fact due to initial baseline differences among the fitness groups.

The failure to observe physiological reactivity differences to a psychosocial stressor as a function of fitness is consistent with several studies which also failed to observe such effects (Hull et al., 1984; Plante & Karpowitz, 1987; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983; Sinyor, Golden, Steinert, & Seraganian, 1986). Although this negative pattern of results suggests that aerobic fitness does not alter the response to stress, it should be noted that this study (and those mentioned above) used highly stressful laboratory tasks, such that aerobic fitness related differences in response to the task used may have been masked. As suggested by Sinyor et al. (1986), the failure to observe fitness related differences while using a powerful stressor may be due to a "ceiling effect". That is, subjects may be responding maximally to such stressor which could prevent the demonstration of fitness related differences in their responding. Another possible explanation may be that other factors such as anticipation to the second session's bleeding time test may

have contributed to the masking of individual differences in prostaglandin responses. Further, it is also possible that the "mild" stress of the Structured Interview had a "carry over" effect on the psychosocial stressor. In light of the known fitness effects on cardiovascular functions, the use of less powerful psychosocial stressors may reveal fitness related differences in the reactivity of Type A and B subjects. Such differences cannot be ruled out at this point, especially in light of the cardiovascular and prostaglandin differences that were observed in this study between the lesser fit and more fit Type As.

Although the psychosocial stressor was effective in eliciting significant cardiovascular responses for all subjects, such was not the case with prostaglandin responses. That is, the individual differences that were observed in thromboxane levels were at resting state and appear to have been cancelled by the psychosocial stressor. The reason for this effect of the stressor on the prostaglandins is not clear. Although little research has been done to date on the effect of emotional stress on prostaglandin responses, increases in platelet secretion in healthy individuals as a result of the stress related to public speaking has been reported (Levine et al., 1985). In this latter study, however, prostaglandin responses were measured from plasma samples which were taken immediately before the stressor, thus suggesting that the increases reflected more

anticipatory stress than the actual effect of the stressor. Levine et al. (1985) also reported that the increase in platelet secretion was associated with an increase in catecholamine levels. The present study differs from Levine's in that the prostaglandins were measured from bleeding time blood which was taken immediately after what could be considered a powerful stressor. Thus, both studies examined the relationship between emotional stress and prostaglandin responses, but since the measurement parameters in these studies were different, and the blood collected at different times during the course of the stressor, it may not be possible to make meaningful comparisons between Levine et al.'s (1985) pre-stressor results and the measures that were taken following the stressor in the present study. Furthermore, because of the lack of empirical evidence on the effect of stressors on prostaglandins, only tentative explanations can be suggested at this point.

For instance, in examining the effects of the stressor on physiological responsiveness (see Fig. 2), the unfit Type As continued to show higher heart rate and a shorter bleeding time. That is, even though the unfit Type As no longer showed an increased thromboxane level, they continued to have a shorter bleeding time. Thus, since there is evidence showing that a number of hemostatic variables are influenced by stress (i.e., catecholamines, cholesterol, von Willebrand factor, Factor VIII) (Gerrard & Peterson, 1985; Herd, 1984),

a potential explanation may be that an (unknown) hemostatic variable inhibited the production of thromboxane during stress, and accounted for the shorter bleeding time seen among the unfit Type As. Further, the present results on prostacyclin also suggest that this inhibitor is not prostacyclin, i.e., no significant decrease in prostacyclin was observed.

Another potential factor that may have contributed to the stress related pattern of results, is the learned ability of the more fit subjects to be better able to match arousal level to task requirements (Lake et al., 1985). That is, despite the cardiovascular benefits associated with fitness, it is conceivable that the fit subjects engaged in the task with more vigour, as they are accustomed to do in aerobic activities, such that possible individual differences in prostaglandin responses were masked. These hypotheses, however, await further empirical investigation.

The Effect of Exercise on Prostaglandins

With respect to the prostaglandin responses elicited by the fitness test, the marginal increase observed in levels of thromboxane as a result of exercise stress appears to be consistent with other findings. For instance, Mehta et al. (1983) reported similar increases in levels of thromboxane when comparing healthy subjects with CHD patients, i.e., the

healthy subjects showed a marginal increase while the CHD patients showed a marked and significant increase as a result of exercise. Carter et al. (1989) also found similar results using a small sample (n=12) of male athletes who underwent 17 min of steady state submaximal exercise, i.e., moderate exercise was associated with a significant increase in the production of thromboxane. Carter's results also revealed that the increase in thromboxane was associated with an increase in the volume of bleeding time blood, an interesting result since the increase in thromboxane (a vasoconstrictor and platelet aggregating agent) cannot account for the increase in the volume of the bleeding time blood. These results were interpreted as implicating prostacyclin in the exercise associated increase of bleeding time blood volume. Although the volume of bleeding time blood was not measured in the present study, similar results were observed in terms of the (not significant) increases in prostacyclin. That is, changes in prostacyclin levels appear to have followed the changes in thromboxane. This observed equilibrium was seen in this study immediately after exercise, and immediately before and after the psychosocial stressor. As suggested by Carter et al. (1989), these results suggest a beneficial effect of thrombotic risk associated with moderate exercise.

The present study also observed significantly shorter bleeding times for the fit subjects as a result of exercise, which is again similar to Carter et al.'s (1989) report that

intensive exercise such as long distance running was not found to be associated with changes in either thromboxane or prostacyclin, but nevertheless with a significant decrease in bleeding time. These authors suggested that as a result of intensive/exhaustive exercise, both a lack of increase in prostacyclin and decreased bleeding time, may be due to an exhaustion of the ability of endothelial cells to produce prostacyclin. Since little is known on the effects of acute exercise on prostaglandins, relationships between exercise and prostaglandin production are open to a variety of interpretation at this point.

Cardiovascular Recovery From Stress

Finally, a third set of results examined the rate of cardiovascular recovery in the minutes that followed the psychosocial stressor. The heart rate results revealed that once the stressor had ended, Type A subjects took longer to return to baseline than Type Bs. This result appears consistent with the greater reactivity of Type As to the onset but not the offset of the stressor, i.e., it is conceivable that since Type As reacted more to the stressor, other things being equal, it would take longer for these individuals to return to baseline. Of the few studies that have examined the relationship between behaviour type and fitness to date (Blumenthal et al., 1980; Hull et al., 1984; Lake et al., 1985; Roskies et al., 1986), only Roskies et al.

(1986) reported recovery data, which identified no fitness related differences in cardiovascular recovery among a Type A sample (Type As were not compared to Type Bs in the latter study). Hence, the present study appears to be the first to date to report A-B differences in heart rate recovery to psychosocial stress.

Studies that have examined the relationship between fitness and stress responsivity have, however, reported data on physiological recovery. These data provide only modest support for the hypothesis that more fit individuals differ than lesser fit ones in their recovery from stressful psychosocial tasks. Furthermore, it is not clear if these modest effects would remain if these studies (e.g., Lake et al., 1985; Sinyor et al., 1983) had used more appropriate statistical procedures, i.e., they failed to control for initial baseline differences due to fitness, thus making it difficult to conclude that the differences they observed were due to a fitness effect. A recent study that used covariance techniques to control for fitness related differences at baseline, found that when using such techniques, fitness related differences in stress responsivity disappeared, and only the pulse rate recovery measure remained significant (Plante & Karpowitz, 1987). Although this latter study demonstrated the effect of better statistical control, their recovery data was difficult to interpret because of the method used to assess fitness (i.e., fitness was estimated

from self-report questionnaires). Despite these inconsistent results, it has been argued that fit and unfit groups may be distinguished by the magnitude of their responses during a mildly stressful task, whereas in a highly stressful task, fit and unfit groups may be distinguished more by their rate of recovery after the task (Blumenthal & McCubbin, 1987).

The recovery data of this study also showed that the values reported as "baseline" may not represent a true baseline, but rather may reflect anticipation of the task. The lower blood pressure values at recovery (see Figure 3) tend to support this observation. This suggests that the individual differences that were observed among the groups at "baseline", might be more accurately considered as individual differences during anticipatory stress. Since it is conceivable that powerful laboratory psychosocial stressors may mask fitness related differences because of a "ceiling effect" (Sinyor et al., 1986), or because of differences in which fit and less fit individuals engage in such demanding task, this may explain why this study observed fitness related differences only during the very mild stress experienced in anticipation of the stressor. In light of this anticipation stress, the results of this study can be seen as consistent with the ones reported by Levine et al. (1985), where an increase in platelet secretion was observed just before public speaking.

Type A Components

Based on the literature reviewed earlier, it had been hypothesized that hostility, suppressed anger, and time urgency, would be associated with physiological responsiveness, and that fitness would interact with this association. Contrary to these predictions, Type A components were not associated with either cardiovascular or prostaglandin responses. Only marginal associations between Type A and hostility were observed. As well, Type As expressed more anger and were more time urgent than were Type Bs. Although these latter findings have been previously reported in the literature, it was somewhat surprising that an association between the components of Type A and the physiological measures was not observed. A likely explanation for this may reside in the characteristics of the sample used by this study. That is, although the subjects were randomly selected, they were nevertheless all young and healthy university students, which may have masked potential differences among the groups (i.e., low vs. high hostile subjects, etc.) due to the homogeneity of the population examined. In contrast, studies that have reported an association between these toxic Type A components and physiological reactivity, or with the severity of CHD, have often used larger and more varied cross-section of the population, or population with at-risk subjects (e.g., hypertensives, patients referred for angiography, etc.).

Hence, it is possible that if this study had used an older and more diversified sample, different results might have been observed with respect to the association of these so-called toxic Type A components and physiological responsiveness. These results nevertheless suggest that despite claims that the global Type A measure contains a considerable amount of "noise" (Williams & Barefoot, 1987), the SI-defined Type A behaviour has a strong association with CHD relevant endpoints. This observation is consistent with Matthews' (1988) recent meta-analysis of the Type A literature which showed a strong association between SI-defined Type A and CHD.

General Discussion

In summary then, this study has provided evidence of an interactive effect between Type A behaviour, prostaglandins, and aerobic fitness. That is, aerobic fitness was found to moderate Type A subjects' cardiovascular functions and prostaglandin responses to the mild stress of anticipation to an experimental task. The present study is believed to be the first to report of such an interaction. In light of recent meta-analyses of the Type A literature (Friedman & Booth-Kewley, 1988; Matthews, 1988) which suggest that TABP is a reliable predictor of initial CHD events in population-based studies, that a disordered balance in the production of thromboxane and prostacyclin has been

implicated in the development and propagation of atherosclerotic lesions (Gerrard, 1985), and that aerobic fitness is associated with lower incidence of CHD and lower mortality rate (Rigotti et al., 1983), the findings reported by the present study have important implications for the prevention of CHD.

This study showed that aerobic fitness was implicated in a reduction in heart rate, blood pressure, and platelet aggregation, in response to the mild psychological stress of anticipation to perform an experimental task. Although fitness-related differences in response to a powerful stressor were not observed, it can be argued that the mild stress of anticipation is more similar to the level of daily stress experienced by most than the level of stress produced by the experimental stressor. Hence, there is evidence supporting the prescription of regular aerobic exercise for stress management. Although it is possible that aerobic fitness does not modify the Type A behaviour pattern, as reported by Roskies et al. (1986), it is nevertheless possible that the benefits associated with aerobic fitness may protect Type A individuals against the adverse effects of their coping style to stress.

At this point, it would appear appropriate to address a few important methodological issues that were raised by this study. The first issue has to do with baseline

differences due to fitness. As mentioned earlier, a number of the fitness related studies reviewed failed to control for baseline differences due to fitness. Failure to statistically control for initial (resting) differences can lead to erroneous interpretation of the results. Hence, this may explain why some studies (e.g., Keller & Seraganian, 1984; Sinyor et al., 1983) reported fitness related differences in cardiovascular reactivity, whereas the present study (which used covariance techniques) failed to observe such effects.

The second issue is related to the Law of Initial Values (LIV) (Wilder, 1931/see Levey, 1980). It was found that most studies examining physiological responsiveness as a function of TABP and/or fitness, failed to assess and/or report whether the LIV was operating for the measures being used. Although the LIV has not been found to be operating consistently in a wide range of psychophysiological measures (Jamieson, 1987), failure to empirically evaluate whether the LIV is operating for the measures used can again lead to erroneous conclusions. As discussed earlier, the LIV was found to be operating for the measures of thromboxane and prostacyclin. Thus, for these measures covariance techniques were used to control for baseline effects.

Finally, the issue of aerobic fitness assessment (i.e., VO_2max) was raised several times throughout this

document. As mentioned earlier, estimated submaximal exercise tests have a standard error of estimate of at least 15% (Astrand, 1976), and unfortunately have been used in most of the studies reviewed. Although these submaximal tests are generally less costly and time-consuming, experimental control and accuracy are sacrificed. It is possible that the inconsistency of results observed could be improved with a more accurate measurement of VO_2max.

Several questions were also left unanswered by this study. For instance, the psychophysiological pathways that mediate the interaction between TABP, prostaglandins, and aerobic fitness, are not well understood. As discussed earlier, hemostatic variables which are influenced by stress (e.g., catecholamines, elevated blood pressure and heart rate, von Willebrand factor, Factor VIII), are believed to play an important role in that interaction. It is thought that differences in which fit and less fit individuals engage in demanding tasks may also have contributed to the effects observed. Finally, future research should investigate further the interaction between TABP, prostaglandins, and fitness, with stressors of various intensity in order to better understand the effect of stress on this interaction. In addition, a more extended baseline period should also be considered in order to adequately assess the subjects' physiological activity at rest.

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APPENDIX A

Lifestyle Questionnaire

Lifestyle Questionnaire

NAME OF PARTICIPANT _____ DATE _____

1. Indicate the physical activities in which you have participated over the last month during your leisure time.

	FREQUENCY: Number of occasions over the last month	DURATION: Average number of activity minutes spent on each occasion				INTENSITY: Slight change from normal state	Some perspiration, faster than normal breathing	Heavy perspiration, heavy breathing
		1–15	15–30	31–60	60+	LIGHT	MEDIUM	HEAVY
Walking for Exercise	_____	☐	☐	☐	☐	☐	☐	☐
Bicycling	_____	☐	☐	☐	☐	☐	☐	☐
Swimming	_____	☐	☐	☐	☐	☐	☐	☐
Jogging/Running	_____	☐	☐	☐	☐	☐	☐	☐
Gardening	_____	☐	☐	☐	☐	☐	☐	☐
Home Exercises	_____	☐	☐	☐	☐	☐	☐	☐
Ice Skating	_____	☐	☐	☐	☐	☐	☐	☐
Cross Country Skiing	_____	☐	☐	☐	☐	☐	☐	☐
Tennis	_____	☐	☐	☐	☐	☐	☐	☐
Golf	_____	☐	☐	☐	☐	☐	☐	☐
Popular Dance	_____	☐	☐	☐	☐	☐	☐	☐
Baseball/Softball	_____	☐	☐	☐	☐	☐	☐	☐
Alpine Skiing	_____	☐	☐	☐	☐	☐	☐	☐
Ice Hockey	_____	☐	☐	☐	☐	☐	☐	☐
Bowling	_____	☐	☐	☐	☐	☐	☐	☐
Exercise Classes	_____	☐	☐	☐	☐	☐	☐	☐
Racquetball	_____	☐	☐	☐	☐	☐	☐	☐
Curling	_____	☐	☐	☐	☐	☐	☐	☐
Others – Please specify:								
_____	_____	☐	☐	☐	☐	☐	☐	☐
_____	_____	☐	☐	☐	☐	☐	☐	☐
_____	_____	☐	☐	☐	☐	☐	☐	☐

APPENDIX B

Structured Interview for University Students

Structure Interview (Student Form)

INTRODUCTION: Most of the questions are concerned with your superficial habits and none of them will embarrass you. I would appreciate it if you would answer the questions to the best of your ability. Your answers will be kept in the strictest confidence. (Begin taping: emphasize capitalized words).

1. May I ask your age, PLEASE?

2. What is your student classification?
a. How long have you been at this university?

3. Are you SATISFIED with your school work thus far? (Why not?)

4. Do you feel that university carries HEAVY responsibility?
a. Is there any time when you feel particularly RUSHED or under PRESSURE?
b. When you are under PRESSURE does it bother you?

5. Would you describe yourself as a HARD-DRIVING, AMBITIOUS type of person in accomplishing the things you want, getting things done as QUICKLY as possible, OR would you describe yourself as a relatively RELAXED and EASY-GOING PERSON?
a. Do you have a boyfriend/girlfriend? (Close friend?)
b. How would he/she describe you ... as HARD-DRIVING and AMBITIOUS or as relaxed and easy-going?
c. Has he/she ever asked you to slow down in your work? NEVER? How would he/she put it ... in HIS/HER OWN words?

6. When you get ANGRY or UPSET, do people around you know it? How do you show it?

7. Do you think you drive HARDER to ACCOMPLISH things than most of your associates?

8. Do you complete homework assignments before they are due? How often?

9. Do you know any children between the ages of 6 and 8? Did you EVER play competitive games with them, like cards, checkers, Monopoly?
a. Did you ALWAYS allow them to WIN on PURPOSE?
b. WHY? (WHY NOT?)

10. When you play games with people your own age, do you play for the fun of it, or are you really in there to WIN?

11. Is there a lot of COMPETITION in school? Do you enjoy this?
    a. Are you competitive in other areas .... sports for example?

12. When you are in your automobile, and there is a car in your lane going FAR TOO SLOWLY for you, what do you do about it? Would you MUTTER and COMPLAIN to yourself? Would anyone riding with you know that you were ANNOYED?

13. Most people who go to school have to get up fairly early in the morning ... in your particular case ... what ... time ... do you ... ordinarily ... get up?

14. If you make a DATE with someone for, oh, two o'clock in the afternoon, for example, would you BE THERE on TIME?
    a. If you are kept waiting, do you RESENT it?
    b. Would you SAY anything about it?

15. If you see someone doing a job rather SLOWLY and you KNOW that you could do it faster and better yourself, does it make you RESTLESS to watch?
    a. Would you be tempted to STEP IN AND DO IT yourself?

16. What IRRITATES you most about this university, or the students here?

17. Do you EAT RAPIDLY? Do you WALK rapidly? After you've FINISHED eating, do you like to sit around the table and chat, or do you like to GET UP AND GET GOING?

18. When you go out in the evening to a restaurant and you find eight or ten people WAITING AHEAD OF YOU for a table, will you wait? What will you do while you are waiting?

19. How do you feel about waiting in lines: BANK LINES, SUPERMARKET LINES, CAFETERIA LINES, POST OFFICE LINES ...?

20. Do you ALWAYS feel anxious to GET GOING and FINISH whatever you have to do?

21. Do you have the feeing that TIME is passing too RAPIDLY for you to ACCOMPLISH all the things you'd like to GET DONE in one day?
    a. Do you OFTEN feel a sense of TIME URGENCY? TIME PRESSURE?

22. Do you HURRY in doing most things?

APPENDIX C

Hostility Scale

HO SCALE

This scale consists of numbered statements. Read each statement and decide whether it is true as applied to you or false as applied to you.
If a statement is TRUE or MOSTLY TRUE, as applied to you, circle the letter T at the left of the statement. If a statement is FALSE or NOT USUALLY TRUE, as applied to you, circle the letter F at the left of the statement.
Remember to give YOUR OWN opinion of yourself. Do not leave any blank spaces if you can avoid it. Now go ahead.

T F 1. When I take a new job, I like to be tipped off on who should be gotten next to.

T F 2. When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing.

T F 3. I prefer to pass by school friends, or people I know but have not seen for a long time, unless they first speak to me.

T F 4. I have often had to take orders from someone who did not know as much as I did.

T F 5. I think a great many people exaggerate their misfortunes in order to gain the sympathy and help from others.

T F 6. It takes a lot of argument to convince most people of the truth.

T F 7. I think most people would lie to get ahead.

T F 8. Someone has it in for me.

T F 9. Most people are honest chiefly through fear of being caught.

T F 10. Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it.

T F 11. I commonly wonder what hidden reason another person may have for doing something nice for me.

T F 12. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.

T F 13. I feel that I have often been punished without cause.

T F 14. My relatives are nearly all in sympathy with me.

over

T F 15. I am against giving money to beggars.

T F 16. Some of my family have habits that bother and annoy me very much.

T F 17. My way of doing things is apt to be misunderstood by others.

T F 18. I don't blame anyone for trying to grab everything he can get in this world.

T F 19. No one cares much what happens to you.

T F 20. It is safer to trust nobody.

T F 21. I do not blame a person for taking advantage of someone who lays himself open to it.

T F 22. I have often felt that strangers were looking at me critically.

T F 23. Most people make friends because friends are likely to be useful to them.

T F 24. I am sure I am being talked about.

T F 25. I am likely not to speak to people until they speak to me.

T F 26. Most people inwardly dislike putting themselves out to help other people.

T F 27. I tend to be on guard with people who are somewhat more friendly than I had expected.

T F 28. I can be friendly with people who do things which I consider wrong.

T F 29. I have sometimes stayed away from another person because I feared doing or saying something that I might regret afterwards.

T F 30. People often dissapoint me.

T F 31. I like to keep people guessing what I'm going to do next.

T F 32. I frequently ask people for advice.

over

T F 33. I have often met people who were supposed to be experts who were no better than I.

T F 34. I would certainly enjoy beating a crook at his own game.

T F 35. It makes me feel like a failure when I hear of the success of someone I know well.

T F 36. I have at times had to be rough with people who were rude or annoying.

T F 37. People generally demand more respect for their own rights than they are willing to allow for others.

T F 38. There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.

T F 39. I am often inclined to go out of my way to win a point with someone who has opposed me.

T F 40. I am quite often not in on the gossip and talk of the group I belong to.

T F 41. The man who had most to do with me when I was a child (such as my father, stepfather, etc.) was very strict with me.

T F 42. I am not easily angered.

T F 43. I have often found people jealous of my good ideas, just because they had not thought of them first.

T F 44. When a man is with a woman he is usually thinking about things related to her sex.

T F 45. I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.

T F 46. I have frequently worked under people who seem to have things arranged so that they get credit for good work but are able to pass off mistakes onto those under them.

T F 47. I strongly defend my own opinions as a rule.

T F 48. People can pretty easily change me even though I thought that my mind was already made up on a subject.

over

T F 49. Sometimes I am sure that other people can tell what I am thinking.

T F 50. A large number of people are guilty of bad sexual conduct.

APPENDIX D

The Anger Expression (AX) Scale

Name _____ M F : Age _____ Date _____

Directions: Everyone feels angry or furious from time to time, but poeple differ in the ways that they react when they are angry. A number of statements are listed below which people have used to describe their reactions when they feel angry or furious. Read each statement and then circle the number to the right of the statement that indicates how often you generally react or behave in the manner described. There are no right or wrong answers. Do not spend too much time on any one statement.

WHEN ANGRY OR FURIOUS	Almost Never	Some-times	Often	Almost Always
1. I control my temper	1	2	3	4
2. I express my anger	1	2	3	4
3. I keep things in	1	2	3	4
4. I am patient with others	1	2	3	4
5. I pout or sulk	1	2	3	4
6. I withdraw from people	1	2	3	4
7. I make sarcastic remarks to others	1	2	3	4
8. I keep my cool	1	2	3	4
9. I do things like slam doors	1	2	3	4
10. I boil inside, but I don't show it	1	2	3	4
11. I control my behavior	1	2	3	4
12. I argue with others	1	2	3	4
13. I tend to harbor grudges that I don't tell anyone about ...	1	2	3	4
14. I strike out at whatever infuriates me	1	2	3	4
15. I can stop myself from losing my temper	1	2	3	4
16. I am secretly quite critical of others	1	2	3	4
17. I am angrier than I am willing to admit	1	2	3	4
18. I calm down faster than most other people	1	2	3	4
19. I say nasty things	1	2	3	4
20. I try to be tolerant and understanding	1	2	3	4
21. I'm irritated a great deal more than people are aware of ..	1	2	3	4
22. I lose my temper	1	2	3	4
23. If someone annoys me, I'm apt to tell him or her how I feel	1	2	3	4
24. I control my angry feelings	1	2	3	4

APPENDIX E

The Time Urgency and Perpetual Activation Scale (TUPA)

INSTRUCTIONS

Answer the following questions by circling the best answer as it applies to you throughout your entire life (and not just the recent past). It is usually better if you circle the first answer to come to mind rather than thinking about it for a long time. Please give one, but only one, answer to each question.

1. When driving around town, I wait until the last minute to leave, and therefore must move with haste to avoid being late.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

2. I open things quickly and forcefully, sometimes ripping boxes or letters open rather than easing things open, or cutting them open gently with an opener.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

3. I have the sense that I am falling behind or that things are gaining on me.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

4. I speed up and brake when driving.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

5. When other people talk, if they do not come to the point, I try to direct the conversation toward the central issue or otherwise keep things on track.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

6. I anticipate a green light by looking at the yellow light for the opposing traffic.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

7. When picking something out of a container, I dig for it quickly.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

8. I look ahead at stoplights and try to time them so I won't have to come to a complete stop.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

9. If I drop something, I attempt to grab it before it hits the ground even resorting to using my foot for this purpose on occasions.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

10. I chew food or gum vigorously.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

11. I will experience a surge of anxiety or other energy if I realize a needed object is lost. I will begin a search for it immediately thinking that this loss has set me behind on my schedule.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

12. I will run a red light, especially if it has just turned red.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

13. I eat on schedule and when hungry, but rarely for pleasure or social reasons.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

14. My eyes are more wide open than most people's.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

15. In traffic, I change lanes rather than staying in a slow one.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

16. I am demanding or hard on machinery, mechanical items, or vehicles.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

17. I can get ready faster than most people.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

18. Once they are set, I find it difficult to abandon activities or plans.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

19. I walk into the street a little early before the light has changed.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

20. When moving about with a group, I go first and lead the way, rather than standing around waiting for someone else to go first or figure out when to move.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

21. I feel stressed over time even when there is no reason, and regardless of circumstances.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

22. I will walk on a "Don't Walk" traffic signal if no traffic is near.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

23. I find that automated doors open too slowly, and that I must slow down a step to avoid running into them.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

24. When I have 5 minutes free, I stay busy doing something, even if I know the task may take 10 minutes or more.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

25. I do more than one thing at a time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

26. I have more than one iron in the fire at a time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

27. If I have spare time between activities, I attempt to "make some progress" on another project.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

28. People close to me have told me to slow down.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

29. I reflect upon upcoming events.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

30. I drive a little above the speed limit but not enough to be stopped for speeding.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

31. I will interrupt activities to take care of something small which has crossed my mind (i.e. something not requiring much time), so I won't have to remember it and take care of it later.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

32. My speech is logical and my points are well supported.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

33. I feel surges of energy when performing household or grooming activities, such as drying hair, taking shoes out of the closet, or buttoning a shirt.
   a. Always
   b. Almost always
   c. Almost never.
   d. Never

34. I prepare for activities ahead of time, so I won't waste time or have to go back and get something I forgot.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

35. I like to make quick departures from stop signs.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

36. I keep my teeth pressed together, without grinding but with my jaw muscle tense.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

37. The only times I feel really comfortable when moving slowly is when I am sick.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

38. I ease through yellow lights or edge forward when waiting for a green light.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

39. I speed up when two lanes of traffic converge, assuming the people in the other lane will either slow down or keep the same speed.
a. Always
b. Almost always
c. Almost never
d. Never

40. I screw lids on containers, such as toothpaste or jars, tighter than most people.
a. Always
b. Almost always
c. Almost never
d. Never

41. I like to be sure to take the shortest, quickest, or otherwise most efficient route on both short or long trips.
a. Always
b. Almost always
c. Almost never
d. Never

42. When planning something (a vacation or my working day), I leave little time for unstructured or spontaneous activity.
a. Always
b. Almost always
c. Almost never
d. Never

43. When turning left and faced with oncoming traffic, I edge out into the street so as to be able to complete the turn on a yellow light.
a. Always
b. Almost always
c. Almost never
d. Never

44. When I have spare time, I use the occasion to plan or think about a task.
a. Always
b. Almost always
c. Almost never
d. Never

45. I have an organized, well-planned day.
a. Always
b. Almost always
c. Almost never
d. Never

46. I seem to anticipate that certain jobs will take less time than they eventually wind up taking.
a. Always
b. Almost always
c. Almost never
d. Never

47. When driving, I coast instead of braking when possible. Because of this I may get close to the car in front of me.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

48. I plan social activities with a fairly precise beginning and/or stopping time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

49. I keep frequently used objects in the same familiar place.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

50. I am careful to run errands in an orderly sequence so as to do them in a minimum amount of time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

51. I experience a surge of energy at the beginning of a work task.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

52. I keep unexpected contacts with people at a minimum so as not to get too far behind my schedule.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

53. While driving, I gather up in advance the items I am going to need for my destination, but before I ever stop the car.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

54. When I must sit still, I handle an object (like a pencil), produce finger movements, move my teeth, or otherwise do not keep completely still.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

55. I write fast and/or press down heavily.
a. Always
b. Almost always
c. Almost never
d. Never

56. I shut or slam doors and/or drawers more vigorously than most people.
a. Always
b. Almost always
c. Almost never
d. Never

57. I work on something up until the last minute, allowing just enough time to go to the next place where I am headed.
a. Always
b. Almost always
c. Almost never
d. Never

58. I have a facial grimace which I exhibit when exerting myself.
a. Always
b. Almost always
c. Almost never
d. Never

59. I carry more things at one time than can be easily managed in order to avoid an extra trip.
a. Always
b. Almost always
c. Almost never
d. Never

60. If I forget to do something, immediately I do it upon remembering, even though there is no real urgency to do it then.
a. Always
b. Almost always
c. Almost never
d. Never

61. A person who rambles when they talk makes me want to coach or otherwise structure their way of talking.
a. Always
b. Almost always
c. Almost never
d. Never

62. I hate to keep anyone waiting, even for 5 minutes.
a. Always
b. Almost always
c. Almost never
d. Never

63. It is difficult for me to sit down to a long meal.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

64. It is difficult for me to sit around and talk after finishing a meal.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

65. I push elevator buttons several times rather than only once.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

66. I want the meetings I attend to follow the agenda.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

67. I dial phone numbers rapidly.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

68. I hate to make a mistake dialing a phone number and to have to start all over again.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

69. I place deadlines on others as well as myself.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

70. I go without a watch ...
    a. Always
    b. Almost always
    c. Almost never
    d. Never

71. I check my watch ...
    a. Always
    b. Almost always
    c. Almost never
    d. Never

72. My free time is spent in planned or organized activity.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

73. I like to spend time in meditative activities such as introspective thinking, prayer, yoga, long walks or other reflective rather than doing activities.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

74. My speech is orderly and precise.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

75. I like to get things done, rather than put them off.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

76. My tone of voice could be termed "forceful" or "dramatic."
    a. Always
    b. Almost always
    c. Almost never
    d. Never

77. I engage in discussions about "non-doing" activities such as art, literature, music, or some other aesthetic subject.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

78. I do not like to sit without anything to do.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

79. I remove keys or other objects from my pocket before I reach the door; therefore, I do not have to stand in front of the door and look for the key.
    a. Always
    b. Almost always
    c. Almost never
    d. Never

80. My tone of voice varies during a conversation.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

81. It bothers me to have to wait for people, particularly if it is somebody who works for me or over whom I have supervisory authority.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

82. I have people say that I am a very busy person, one of the busiest that they have ever known.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

83. I change my route of travel on streets depending on whether or not I hit a red light. (i.e. If I come to a red light and I can turn right and go a different route instead of wait through the red light, I will.)
   a. Always
   b. Almost always
   c. Almost never
   d. Never

84. I sometimes go up stairs two at a time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

85. I get angry, because I feel that nothing gets done at work until I get there and take control.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

86. I get angry with drivers who sit at a red light in the right-hand lane when I am behind them and want to turn right on the red light.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

87. I get irritated with drivers ahead of me, in the left-hand lane, who drive slower than I do.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

88. I get irritated with people who don't do what I want immediately upon my asking.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

89. I get angry with myself when I make mistakes.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

90. I get angry when items are not where I expect them to be.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

91. I can not stand constant interruptions.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

92. I find that while doing one physical activity (e.g. painting, etc.) that my mind is concerned with 1 or more other projects that I have under way.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

93. I would rather do something myself than wait for someone else to do it-- the other party is never fast enough for me.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

94. I get bored with mundane things.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

95. I like crises.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

96. I like being needed and in demand.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

97. I enjoy an extremely full day.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

98. If I have an appointment, I will watch the clock in order to try to do just one additional thing before time for that appointment.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

99. I catch myself estimating the number of minutes it will take me to get to my appointment so I can leave at the last minute and still be on time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

100. I schedule activities as close as possible to both sides of an appointment in order not to waste time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

101. During one appointment, I am already thinking about my next appointment.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

102. I make sure the other person knows that I have another appointment or that I am a busy person during an appointment; thereby moving our meeting along more "crisply."
   a. Always
   b. Almost always
   c. Almost never
   d. Never

103. I make notes or think of other things to do while attending meetings.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

104. I follow a very structured schedule when getting ready in the mornings-- if this schedule varies in the time it takes, it throws me off.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

105. When stopping at a 4-way stop sign with cars ahead of me, I watch cars as they take their turn leaving the stop sign and have figured out before my turn as to which car I will follow in leaving the stop sign.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

106. In meetings, I watch the agenda very closely. If the agenda does not stay close to the schedule time, this worries me and I find myself thinking of a way I can help us get back on schedule.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

107. My desk is cluttered because I will work on several things at the same time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

108. I wake up during the night, thinking of something I need to do the next day.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

109. I keep a pad of paper handy, so that I can write down ideas or plans that come to me , even during the night.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

110. I become impatient with people who are able to operate at a slower, less-structured pace.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

111. I work out a daily schedule of events and become frustrated when someone talks to me too long and it causes me to get off schedule.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

112. I become frustrated when someone is talking or explaining an event and they go into such detail that it takes, in my estimation, an excessive amount of time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

113. I am structured, energetic, and something of a perfectionist. I tend to believe that others could and should be similar.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

114. Before going to bed, I plan and/or lay out the clothes that I want to wear the next day.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

115. Other people on the same job worry me when they do not pull their share of the work.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

116. I get frustrated when fellow workers want to "visit" or casually talk with me while on the job.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

117. I rush when shopping.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

118. I do not like to be interrupted when working on any project.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

119. I do not like company who has not been invited.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

120. I do not like to travel a long distance.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

121. I tend to go to bed at the same time every night.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

122. I wad or fold up paper that I am putting in the trash can.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

123. I "toss" waste paper rather than gently placing it inside the trash can.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

124. If a meeting lasts longer than the time it is scheduled to end, I will go ahead and leave at the scheduled time.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

125. When the plans I make for the day do not go smoothly, I start changing them.
a. Always
b. Almost always
c. Almost never
d. Never

126. I find myself competing with fellow workers.
a. Always
b. Almost always
c. Almost never
d. Never

127. I will skip lunch on order to do some work during the lunch hour.
a. Always
b. Almost always
c. Almost never
d. Never

128. I do not like it when conferences or meetings are interrupted.
a. Always
b. Almost always
c. Almost never
d. Never

129. I become inpatient in restaurants when the waitress or waiter is slower than I think she or he should be.
a. Always
b. Almost always
c. Almost never
d. Never

130. When I arrive early for a meeting, I get impatient waiting for the meeting to start.
a. Always
b. Almost always
c. Almost never
d. Never

131. I look at my watch on the way to work, checking at specific locations or places to be sure that I will not be late.
a. Always
b. Almost always
c. Almost never
d. Never

132. I will take a business related phone call during a personal conversation.
a. Always
b. Almost always
c. Almost never
d. Never

133. I make a special effort to be first at the office or at my job each day.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

134. I work more than 8 hours per day.
   a. Always
   b. Almost always
   c. Almost never d. Never

135. I work more than 5 days per week.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

136. I enjoy competitive-type recreational activities.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

137. I find it difficult to sit still and do nothing.
   a. Always
   b. Almost always
   c. Almost never
   d. Never

APPENDIX F

The bleeding times were performed on the volar surface of the arm, distal to the antecubital fossa, avoiding surface veins, using a sterile, disposable, standardized device (Simplate, General Diagnostics) to produce a vertical incision 5 mm long by 1 mm deep. A sphygmomanometer cuff on the upper arm inflated to 40 mm Hg maintained a constant pressure. A stop watch was started once the incision was made. Blood from the edge of the incision was collected into heparinized microhematocrit tubes containing 10 ul of 1 mm/ml indomethacin. The tubes were then centrifuged, the plasma volume measured, and the plasma removed into 1.4 ml polyethylene Eppendorf tubes and acidified with the addition of 1.4 ml of pH 3.0 citric acid.

The plasma samples to be analyzed for thromboxane B_2, the stable metabolite of thromboxane A_2, and 6-keto-PGF_1, the stable metabolite of prostacyclin, were loaded onto a c18 Sep-Pak Cartridge, prewashed with 20ml of absolute ethanol, and 40 ml of double-distilled water. In succession, water (20 ml), 10% ethanol (20 ml) and petroleum ether (10 ml) solvents were passed through the cartridges. Then, the prostaglandins were extracted in 7.5 ml methyl formate. This fraction was evaporated under a nitrogen evaporator and the sample then reconstituted in 0.5 ml 1% BSA (bovine serum albumin) in PBS (phosphate buffered saline). The amounts of 6-keto-PGF_1 and thromboxane B_2 were assayed using highly

specific anti-6-keto-PGF_1 and anti-thromboxane B_2 in an enzyme-linked immunosorbent assay (ELISA) (Gerrard, Taback, Singhroy, et al., 1988). Because the reliability of Elisa has been assessed in previous studies (see Gerrard et al., 1989), and the results of this study were consistent with previous samples (Gerrard et al., 1989; Gerrard et al., 1988), the results of the assay were not double-checked in this study. Finally, thromboxane, produced by blood platelets is expressed in a concentration (ng/ml), while prostacyclin, produced by the locally affected endothelium, is better expressed by rate of production (pg/min) (Gerrard et al., 1989).

Prostacyclin is expressed as production per time period, or picograms per minute, mostly because it could be potentially erroneous and misleading to express this as a concentration in the blood (Gerrard, 1989). That is, it is quite possible for the diameter of the skin vessels to vary significantly, for example, in response to the vasodilator action of prostacyclin itself), which would then increase the blood flow. "Thus, to express prostacyclin production in proportion to the volume of blood or as a concentration in the blood, will give falsely high values under conditions of vasoconstriction or low flow, and falsely low values under conditions of high flow and high bleeding time blood volume" (Gerrard, 1989).

With respect to thromboxane, a previous study (Gerrard

et al., 1989) compared thromboxane production expressed as rate of production (pg/min) and as concentration (ng/ml). The results from the two methods gave reasonably comparable results. However, three subjects in this study showed erratic patterns in response to aspirin when thromboxane was expressed as rate of production. The reason for the results was traced to a considerable postaspirin increase in blood flow associated with a decreased, but still significant, thromboxane production because these subjects were high thromboxane producers to begin with. When thromboxane production was expressed as a concentration (ng/ml), the results were consistent in showing a similar pattern of inhibition after aspirin as the other subjects (Gerrard et al., 1989).

APPENDIX G

The Physical Activity Readiness Questionnaire (PAR-Q)

AND

The Consent Forms for the Fitness Test and Bleeding Time

Physical Activity Readiness Questionnaire (PAR-Q)*

NAME OF PARTICIPANT _____

DATE _____

PAR Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check (√) the ☐ YES or ☐ NO opposite the question if it applies to you.

YES	NO	
☐	☐	1. Has your doctor ever said you have heart trouble?
☐	☐	2. Do you frequently have pains in your heart and chest?
☐	☐	3. Do you often feel faint or have spells of severe dizziness?
☐	☐	4. Has a doctor ever said your blood pressure was too high?
☐	☐	5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
☐	☐	6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
☐	☐	7. Are you over age 65 and not accustomed to vigorous exercise?

Reference: PAR-Q Validation Report. British Columbia Ministry of Health. 1978.

* Produced by the British Columbia Ministry of Health and the Department of National Health & Welfare

INFORMED CONSENT FORM FOR A MAXIMAL TREADMILL TEST

I, the undersigned, do hereby acknowledge:

- my consent to perform a graded treadmill test to exhaustion in order to determine my maximal oxygen uptake for use in a research study;

- my understanding that there exists the possibility of certain changes occurring during and after the test including abnormal blood pressure, fainting, light-headedness, leg cramps, nausea, and in rare instances, heart attack or heart rhythm disturbances;

- my obligation to immediately inform the appraiser of any pain, discomfort, fatigue, or any other symptoms that I may suffer during and immediately after the test;

- my understanding that I may stop or delay any further testing if I so desire and that the testing may be terminated by the appraiser upon observation of any symptoms of distress or abnormal response;

- my understanding that I may ask any questions or request further explanation or information about the procedures at any time before, during, and after the test;

- my understanding that I will receive $20.00 for my participation in this study; the payment will be made at the end of the 2nd session.

- that I have read, understood, and completed the screening questionnaire (PAR-Q) and obtained medical clearance if required.

- that I hereby release the Target Fitness Centre, the University of Manitoba, its agents, officers and employees from any liability with respect to any damage or injury (including death) that I may suffer during the administration of the maximal treadmill test except where damage or injury is caused by the negligence of the Target Fitness Centre, the University of Manitoba or its agents, officers and employees acting within the scope of their duties.

_____ _____

SIGNATURE DATE

_____ _____

WITNESS DATE

CONSENT FORM

During the course of studies on chemicals produced in the blood from the site of a bleeding time incision, we have made casual observations which suggest that there might be an association between behaviour patterns and the production of these chemicals. We wish to evaluate this association by asking you a series of questions about your responses to a number of normal life situations, and to measure the production of these chemicals (thromboxane and prostacyclin) in the blood from the site of a small cut on four separate occasions: i.e., before and after the fitness test, and before and after a mental task (which is preceded by the interview).

I, _____, consent to have a small cut, less than 1/4 inch, made on the inner side of my forearm on four separate occasions; before and after a fitness test, and before and after a mental task (which is preceded by the interview). I understand that the cuts may leave a small scar. I understand that I may withdraw from this study at any time without prejudice.

Date _____

Signature _____

Witness _____