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## Type A Behavior and Coronary Heart Disease: A Methodological Review Based on Decision Theory

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TYPE A BEHAVIOR AND CORONARY HEART DISEASE:  
A METHODOLOGICAL REVIEW BASED ON DECISION THEORY

by

Todd Q. Miller

A Dissertation Submitted to the Faculty of the Graduate  
School of Loyola University of Chicago in Partial  
Fulfillment of the Requirements of the Degree of  
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## VITA

The author, Todd Q. Miller, is the son of Ralph Howard Miller and Janet Arlene (Mitchell) Miller. He was born June 25, 1957, in Salt Lake City, Utah. His elementary education was obtained in the public schools of Salt Lake City, Utah. His secondary education was completed in 1975 at Skyline High School, Salt Lake City, Utah. In September, 1975, Mr. Miller entered the University of Utah, receiving the degree of Bachelor of Science in psychology in June, 1984. In September, 1984, Mr. Miller enrolled in the applied social psychology program at Loyola University of Chicago and completed his Master of Arts in May of 1987. From 1986 to 1989 Mr. Miller worked as a statistical consultant for Dr. Steven Sontag at Hines Veterans Administration Hospital. He was awarded the Arthur J. Schmidt fellowship for the 1988-89 academic year. He received his Doctorate of Philosophy in January of 1990. Mr Miller is currently working as the Senior Research Specialist at Prevention Research Center at the University of Illinois. His work involves developing statistical models for describing the psychological processes related to smoking and drug use. In addition, Mr. Miller provides statistical consultation and supervision for ongoing projects of randomized intervention studies.

## PUBLICATIONS

- Donrad, K.J., & Miller, T.Q. (1987). Measuring and testing program philosophy: A framework for implementation studies. In L. Bickman (ed.) New directions in program evaluation: Program evaluation theory (Vol. 33). Beverly Hills: Sage.
- Miller, T. Q., Turner, C. W., Tindale, R.S., & Posavac, E.J. (1988). Disease based spectrum bias in referred samples and the relationship between Type A behavior and coronary artery disease. Journal of Clinical Epidemiology, 41, 1139-1150.
- Miller, T.Q., & Best, W. (In press). The use of structural analysis in medicine. Journal of Clinical Gastroenterology.
- Sontag, S., Schnell, T., Miller, T.Q., O'Connell S., Serlovsky, R., & Nemchausky, B. (in press). Esophageal acid profiles in gastroesophageal reflux. Journal of Clinical Gastroenterology.
- Sontag, S., O'Connell, S., Khandelwal, S., Miller, T., Nemchausky, B., Schnell, T. G., & Serlovsky, R. (In press). Most asthmatics have gastroesophageal reflux with or without bronchodilators therapy. Gastroenterology.
- Gollapudi, P., Sontag, S., & Miller, T.Q. (1988). Surgery for complications of peptic ulcer before and after

availability of cimetidine. Gastroenterology, 94(5), (Abstract), A151.

Schnell, T., Sontag, S., Chejfec, G., & Miller, T. (1988). Detection of Barrett's esophagus and curable esophageal adenocarcinoma by endoscopic screening based on GER symptoms: Can it be cost effective? Gastroenterology, 94(5), (Abstract), A411.

Schnell, T., Sontag, S., Chejfec, G., Miller, T., Chintam, R., Kurucar, C., & O'Connell S. (1989). Effect of indication for endoscopy and source of patient on prevalence of Barrett's esophagus and adenocarcinoma of the esophagus. Gastroenterology, (Abstract)

Sontag, S., Schnell, T., Mak, E., Adelman, K., Miller, T., Schleifer, J., & Skibbins, K. (1989). NSAIDS with or without prednisone: Bad news for the gut mucosa. Gastroenterology. (Abstract)

Sontag, S., O'Connell, S., Khandelwal, S., Miller T., Serlovsky, R., & Nemchausky, B. (1989). Does wheezing occur in association with an episode of gastroesophageal reflux? Gastroenterology. (Abstract)

TABLE OF CONTENTS

ACKNOWLEDGMENTS . . . . .	ii
VITA . . . . .	iii
LIST OF TABLES . . . . .	ix
LIST OF FIGURES . . . . .	x
CONTENTS OF APPENDICES . . . . .	xi
INTRODUCTION . . . . .	1
A DECISION THEORY MODEL OF SUBJECT SELECTION BIASES .	4
Study Designs Used in Research on Type A	
Behavior and Coronary Heart Disease . . . . .	4
A Decision Theory Model for Describing Subject	
Selection Biases . . . . .	8
The need for a statistical model . . . . .	8
The decision theory model . . . . .	10
Assessment of statistical association . . . . .	12
Disease Based Spectrum Bias	
in High Risk Studies . . . . .	15
Selection Biases . . . . .	18
A REVIEW OF EXPLANATIONS FOR NULL FINDINGS . . . . .	23
Hypotheses Proposed by Previous Researchers . . . . .	23
Disease based spectrum bias . . . . .	23
Selection bias . . . . .	23
Insufficient sample size . . . . .	24
Disease criteria . . . . .	24
Indicator of Type A behavior . . . . .	24
The Current Research . . . . .	25
METHOD . . . . .	27
Locating Studies . . . . .	27
Criteria for Including Articles . . . . .	27
Study selection criteria . . . . .	27
Selection criteria for prevalence estimates	29
Selection criteria for studies used to test	
for the presence of disease based	
spectrum bias . . . . .	29
Statistical Analyses . . . . .	30
Use of biserial and	
tetrachoric r coefficients . . . . .	30
Controlling for sampling variability . . . . .	31
Methods used to sum r's . . . . .	31
Testing for inter-study variability . . . . .	32
Study Variables . . . . .	32
Disease criteria . . . . .	32
Measures of TAB . . . . .	32

Study design . . . . .	33
Culture . . . . .	33
Sex . . . . .	33
Year of publication . . . . .	33
Unit of Analysis . . . . .	34
Testing for the Presence of DBS Bias . . . . .	36
Testing for a higher percentage of Type A's in high risk populations . . . . .	37
Estimating the reduction in the magnitude of the r produced by range restriction in recurrent CHD studies . . . . .	38
Distinguishing between DBS bias and other subject selection biases . . . . .	39
RESULTS . . . . .	41
Study Characteristics Associated With the Trend Towards Null Findings . . . . .	41
High risk studies . . . . .	41
Healthy population studies that assessed TAB by the SI . . . . .	43
Studies that assessed TAB by self-report measures . . . . .	44
Studies that used fatal MI as a criterion . . . . .	47
Evidence of DBS Bias in High Risk Studies . . . . .	47
Evidence of smaller r's in high risk studies . . . . .	47
Evidence of a lower percentage of Type A's found in healthy populations . . . . .	49
Evidence for the influence of culture . . . . .	49
Evidence of a higher prevalence of TAB in high risk comparison and disease groups . . . . .	53
Evidence of range restriction . . . . .	55
Evidence of selection biases . . . . .	58
Evidence of a trend towards more DBS bias in angiography studies . . . . .	60
DISCUSSION . . . . .	65
Reasons for the Trend Towards Null Findings . . . . .	66
Increased use of recurrent CHD studies . . . . .	66
Increase in extent of CAD in angiography patients . . . . .	67
Increased use of self-report measures . . . . .	67
The use of fatal MI as a disease criterion . . . . .	68
The MRFIT study . . . . .	68
Suggestions for Future Research . . . . .	70
POTENTIAL APPLICATIONS OF THE DT MODEL FOR FUTURE RESEARCH . . . . .	72
Mortality Bias in Cross-sectional High Risk Studies . . . . .	72
Implications of the DT Model for Future Research . . . . .	74



Research to determine what biases are important . . . . .	74
Interpretation of results . . . . .	74
Detecting mortality bias . . . . .	75
Investigating other types of spectrum biases . . . . .	75
Research to assess the prevalence of risk factors . . . . .	76
Planning sample sizes for future research .	76
Using previous research from other sciences .	78
REFERENCES . . . . .	80
APPENDIX A . . . . .	88
APPENDIX B . . . . .	94
ENDNOTES . . . . .	100

## LIST OF TABLES

Table	Page
1. A Contingency Table of Disease versus Risk Factor Status . . . . .	13
2. Description of the WCGS at the 4.5 year follow-up . . . . .	19
3. Summary the of Characteristics Studies with Null Findings . . . . .	42
4. Findings for Each Type of Study Design . . . .	45
5. Studies that Used Fatal MI as a Criterion . .	48
6. The Prevalence of Type A's in Healthy Population Studies that Assessed TAB by the SI . . . . .	51
7. Percentage of Type A's Found in High Risk Studies . . . . .	52
8. Comparison of Observed r's between TAB and CHD in Recurrent CHD Studies with Expected r's . . .	57
9. Year of Publication, % Ill and r in Angiography Studies that Assessed TAB by the SI . . . . .	63

LIST OF FIGURES

Figure	Page
1. A Decision Theory Model of Diseased Based Spectrum and Mortality Bias . . . . .	11
2. A Decision Theory Model of a Prospective Study .	21

CONTENTS OF APPENDICES

APPENDIX A	List of Studies not included in previous reviews . . . . .	88
APPENDIX B	Estimating reduction in risk . . . . .	94

## INTRODUCTION

Over the past thirty years, numerous studies have attempted to determine whether Type A behavior (TAB) is a risk factor for coronary heart disease (CHD; see Booth-Kewley & Friedman, 1987). TAB has been defined as a pattern of behaviors including "extremes of competitive achievement striving, hostility, aggressiveness, and a sense of time urgency, evidenced by vigorous voice and psychomotor mannerisms" (Matthews, 1988). Type B's are individuals who do not display the characteristics of Type A's.

Contradictory findings in research on TAB and CHD have spurred so much debate that Dimsdale (1988) has suggested the 'A' in TAB might well stand for acrimony. The purpose of this dissertation is to identify the study characteristics associated with positive and null findings.

At the heart of the debate over inconsistent findings in research on TAB and CHD is the failure of recent studies to replicate the findings of previous research. Before 1978, the findings from a number of studies converged in suggesting that TAB is a risk factor for CHD. In fact, the evidence in 1978 was sufficiently convincing that a review panel from the National Heart, Lung and Blood Institute (NHLBI) concluded that TAB is a risk factor for CHD of equal magnitude to the traditional risk factors (smoking, serum cholesterol, and blood pressure).

Suddenly after the NHLBI panel's findings were published, Type A's no longer seemed to be at greater risk for CHD. In contrast to the consistent and strong relationship reported prior to 1978, a remarkable number of subsequent studies failed to find an association between TAB and CHD (for reviews, see Booth-Kewley, 1987; Matthews, 1988). The trend towards null findings has been so consistent that the participants of the 1987 annual meeting of the Psychosomatic Society questioned the relevance of the whole Type A concept (Staff, 1987).

Perhaps the most disconcerting evidence has been the results of Ragland and Brand's (1988a,b) twenty-two year follow-up study; this study was based on subjects who participated in the first prospective study to report an association between TAB and CHD. Ragland and Brand (1988a) reported that Type B's with prior CHD incurred a second fatal myocardial infarction (MI) in less time than Type A's. Furthermore, healthy Type A's were no more likely than Type B's to incur a fatal MI (Ragland & Brand, 1988b). To the chagrin of many researchers (Correspondence, 1988), these results were interpreted by some as suggesting that TAB is not a risk factor for CHD.

However, not all of the recent research has contradicted earlier findings. One true experimental study successfully reduced the number of subsequent episodes of CHD in a treatment condition that was designed to modify TAB

(Friedman et al., 1986). These results suggest TAB produces CHD.

The research discussed in the current dissertation attempts to identify some of the reasons for the confusing results in research on TAB and CHD by a quantitative review of the literature. The first chapter introduces a Decision Theory (DT) model that can be used to define subject selection biases that may have occurred in research on TAB and CHD. The DT model is used to develop hypotheses concerned with how subject selection biases can influence the results of a study. The second chapter discusses the methods used in the quantitative review and a third chapter discusses the results. The results lend support to the value of the DT model by suggesting that a type of subject selection bias referred to as diseased based spectrum (DBS) bias has produced some of the null findings in recent studies. The fourth chapter discusses the implications of the results for future research on TAB and CHD. The final chapter discusses general implications of the DT model for future research. This chapter illustrates how another subject selection bias (mortality bias) can be described by the DT model. In addition, a formula is presented for estimating the reduction in correlation between TAB and CHD in a study that is vulnerable to DBS bias.

A DECISION THEORY (DT) MODEL OF SUBJECT SELECTION BIASES  
Study Designs Used in Research on TAB and CHD

Before discussing the DT model, the following section reviews the different types of study designs used in research on TAB and CHD. A major purpose of the research described in this dissertation is to determine how subject selection biases influence the magnitude of the relationship between TAB and CHD. Study design determines subject selection procedures. Therefore, clear and explicit categories of study design based on the method of subject selection are necessary for assessing the influence of subject selection on the results of research on TAB and CHD.

The most frequently used study designs in research on TAB and CHD have included (a) recurrent CHD studies, (b) angiography studies, (c) studies that select patients on the basis of their risk factor status, (d) population studies, (e) case-control studies and (f) healthy population prospective studies. These study designs have been referred to as epidemiological study designs because these types of study designs have frequently been employed in epidemiological research. These study designs vary in how subjects are selected by whether the design is (a) high risk or healthy population, (b) cross-sectional or prospective designs and (c) the method used to recruit subjects.

For the current paper, the phrase "high risk" study design refers to a study design in which only subjects with



at least some disease are selected for study. Therefore, only a part of the full range of disease severity is examined in high risk studies<sup>1</sup>. In contrast, "healthy population" studies sample subjects across the full spectrum of disease. In particular, study designs are considered to be "high risk" if a comparison group includes individuals with substantial disease. These comparison groups with substantial disease are referred to as high risk comparison groups. Alternatively, comparison groups that include individuals who are healthy are referred to as healthy comparison groups. Studies that use healthy comparison groups are referred to as healthy population studies.

Research on TAB and CHD has used three types of high risk study designs: (a) angiography studies, (b) recurrent CHD studies and (c) studies that select subjects on the basis of their risk factor status. Healthy population studies include the following study designs: (a) healthy population prospective studies, (b) case-control studies and (c) cross-sectional population studies.

In recurrent CHD studies, subjects are selected who have incurred a MI; these subjects are followed over time to determine which subjects develop recurrent CHD. Individuals with a previous MI who do not develop future CHD (a high risk comparison group) are compared with individuals who develop recurrent CHD in the future (a diseased group). Researchers have expected that a higher percentage of

individuals with recurrent CHD should be Type A than individuals who do not experience recurrent CHD. Recurrent CHD studies constitute a high risk study design.

A second type of high risk study is an angiography study. An angiography is a diagnostic test that is used to determine the extent of a patient's coronary artery disease (CAD); CAD is a precursor to most cases of MI. In research on TAB, individuals who are diagnosed by the results of an angiography as having clinically relevant CAD are compared with subjects who are diagnosed as having less CAD. Researchers have expected that a higher proportion of the diseased group would be Type A than the high risk comparison group.

An angiography study is a type of "high risk" study because the comparison group includes many individuals with CAD. Diagnostic screening procedures (e.g., stress testing) insure that most patients undergoing diagnostic coronary angiography have substantially more CAD than healthy individuals. Therefore, almost all subjects who undergo angiography have substantial CAD. This fact may account for why researchers have found only minimal differences in the degree of disease severity found in comparison and diseased groups in angiography studies (Fried & Pearson, 1987).

Some studies selected subjects on the basis of their risk factor status; these studies only select subjects who are considered to be at high risk for future CHD. The

subjects in these studies are at high risk for future CHD because they possess some known risk factor (e.g., serum cholesterol) for CHD. The comparison groups in these studies frequently include subjects with subclinical disease produced by the presence of traditional risk factors. Thus, a study that selects subjects on the basis of their risk factor status is a type of high risk study.

There are two types of healthy population studies: (a) population studies and (b) case control studies. In population studies, subjects are sampled from community populations (e.g., towns or a factory). For healthy population studies, subjects without CHD are compared with subjects with CHD who live in the same population. In case control studies, individuals with CHD are selected from hospital(s) or physician(s) practices. Healthy individuals are selected from the same population as the individuals with CHD. For example, some case control studies have used patients in the same hospital without CHD as control subjects.

In prospective studies, subjects are selected on the basis of their disease status, and they are followed over time to determine which alleged risk factors predict future disease. In cross-sectional studies, individuals are assessed at a single point in time.

Recurrent CHD and healthy population prospective studies are specific types of prospective studies. For

healthy population prospective studies, only healthy individuals are initially selected for study. Population studies, case-control studies and angiography studies are types of cross-sectional studies. Studies that select subjects on the basis of their risk factor status may be cross-sectional or prospective.

Studies have recruited subjects with CHD by four methods: (a) selection on the basis of risk factor status, (b) selection by the results of an angiography in which subjects were diagnosed as having clinically significant CAD, (c) selection because the individual developed CHD during the course of a prospective study and (d) selection because the subject worked in a company or town participating in a study. Studies have either recruited healthy subjects from (a) specific populations (e.g, towns, communities, patients in hospitals without CHD disease) or (b) used volunteers.

### A Decision Theory (DT) Model for Describing Subject Selection Biases

The need for a statistical model. All of the different types of study designs used in research on TAB and CHD are vulnerable to subject selection biases. However, not all designs are vulnerable to the same biases. The degree to which the various subject selection biases influence research on TAB and CHD is unknown. Thus, the magnitude of the association between TAB and CHD found in any given study

may be biased by subject selection biases (Matthews, 1988). Therefore, there is a great deal of confusion regarding the interpretation of results in research on TAB and CHD (Dimsdale, 1988).

Researchers can test hypotheses concerning how subject selection biases by examining how the magnitude of the association between TAB and CHD varies across different study designs. Thus, determining the extent to which subject selection biases have contributed to different findings in research on TAB and CHD has implications for the types of study designs that are employed in future research and how the results of research on TAB and CHD is interpreted.

The study designs used in research on TAB and CHD are vulnerable to two types of subject selection biases: (a) spectrum and (b) selection biases. Research suggests spectrum and selection biases have led to a number of confusing and contradictory findings in epidemiological research (Ransohoff & Feinstein, 1978).

Given that biases are an important problem in epidemiological research, a general strategy for defining and testing for such biases would be useful. The differences between spectrum and selection bias become clearer when a statistical model is used to define these biases. Decision Theory (DT) is a statistical model that can be used to define spectrum and selection biases (Miller

et al., 1988). The following paragraphs describe the DT model introduced in Miller et al. This DT model is then used to develop hypotheses to detect the presence of spectrum and selection biases in research on TAB and CHD. In addition, the DT model can be used to develop hypotheses regarding which biases influence each type of study design.

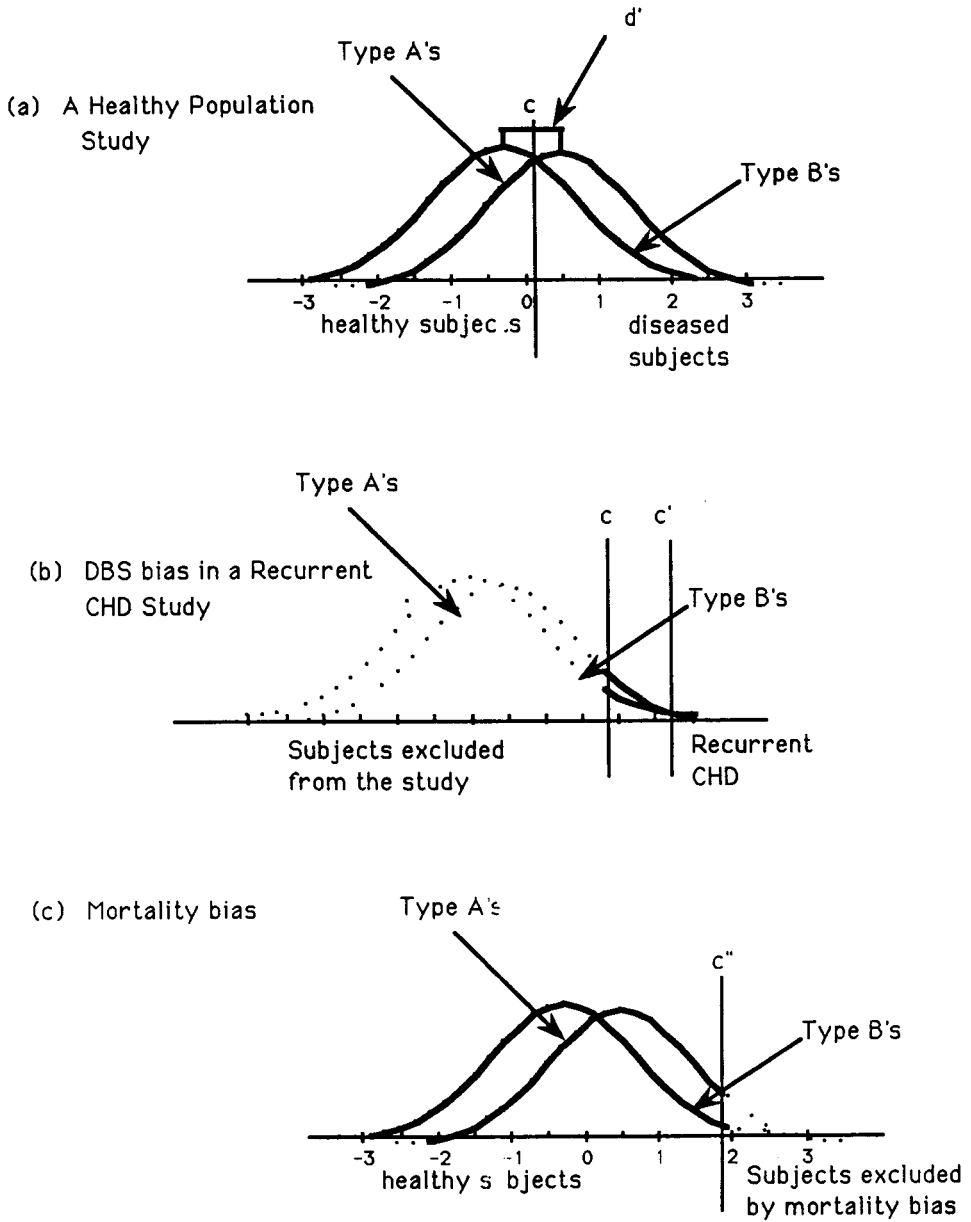
The DT model. Figure 1(a) illustrates the results of a hypothetical healthy population study of the relationship between TAB and CHD in terms of the DT model. The x-axis represents a continuum of the degree of CHD in the sample. Higher values indicate greater severity of CHD. Negative values on the x-axis represent the healthy range and positive values represent the diseased range. The y-axis indicates the frequency of individuals at each level across the full spectrum of CHD.

The cutpoint labeled  $\underline{c}$  in Figure 1(a) is used to distinguish healthy individuals from individuals with CHD. The  $\underline{c}$  indicates the point at which CHD is sufficiently severe that some evidence of CHD (i.e., MI or angina) is present. Of course, no exact point exists at which CHD is universally expressed. The  $\underline{c}$  value only represents a mean value.

One of the two frequency distributions illustrated in Figure 1(a) represents all Type A's in the hypothetical study sample and the other distribution represents all Type B's. The DT model illustrated in Figure 1 assumes Type A's

Figure 1

A Decision Theory Model of DBS and Mortality Bias



develop CHD more frequently than Type B's. Thus, the frequency distribution associated with more CHD represents individuals who are Type A's and the distribution associated with less CHD represents individuals who are Type B's<sup>2</sup>. The frequency distribution of Type A's is the same magnitude as the Type B distribution because prior research suggests that healthy populations include equal numbers of Type A's and B's (see Miller et al., 1988).

Assessment of statistical association. The effect size ( $d'$ ) illustrated in Figure 1(a) is an indicator of the degree of statistical association and represents the standardized mean difference in degree of disease between the two distributions of individuals with and without the risk factor (Glass, Smith & McGraw, 1981). In Figure 1(a),  $d'$  represents the standardized mean difference in degree of CHD between the Type A's and Type B's. However, this method of assessing statistical association in research on TAB and CHD has not been used. Instead, researchers have analyzed their results by contingency tables.

A contingency table can be used to describe the degree of statistical association between TAB and CHD in the hypothetical study sample illustrated in Figure 1(a). The frequency distributions can be arranged into the four groups in the contingency table presented in Table 1. In this table, Type B's with CHD are referred to as false negatives (FN) because CHD is not expected to be present. Healthy



Table 1

A Contingency Table of Disease versus Risk Factor Status

		Disease Status	
		Comparison Group	Diseased Group
Risk factor status	Type A's	False Positives (FP)	True Positives (TP)
	Type B's	True Negatives (TN)	False Negatives (FN)

$$\text{Relative Risk (RR)} = \frac{(TP)(TN + FN)}{(FP + TP)(FN)}$$

$$\text{Odds ratio} = \frac{(TP)(TN)}{(FP)(FN)}$$

$$\text{Sensitivity} = \frac{TP}{(TP + FN)}$$

$$\text{Specificity} = \frac{TN}{(FP + TN)}$$

Type A's are referred to as false positives (FP). Type A's with CHD are referred to as true positives (TP) and healthy Type B's are referred to as true negatives (TN).

Two indices--the sensitivity and the specificity--have been used to describe the relation between TAB and CHD. The sensitivity indicates the degree to which TAB can identify individuals with CHD in the diseased group. For example, 69% of the subjects who developed CHD were Type A's in the Western Collaborative Group Study (WCGS; Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975). The WCGS was the first prospective study that examined the relationship between TAB and CHD. Therefore, the sensitivity in the WCGS study is 69%.

The specificity is estimated with data from the comparison group. The percentage of healthy subjects who do not possess the risk factor (i.e., Type B's) in the subsample is referred to as the specificity. The specificity indicates the degree to which the absence of the risk factor is associated with the absence of the disease. For example, the specificity of TAB was 52% in the WCGS because 52% of the individuals who remained healthy in that study were Type B's. High values of sensitivity and specificity indicate there is a strong association between the risk factor and the disease.

Two indices have been used in research on CHD to indicate the degree of statistical association present in

Table 1. The relative risk (RR) is an index that indicates the increased risk for CHD associated with the presence of TAB in a prospective study. In research on TAB and CHD, the RR represents the increased odds of Type A's developing CHD compared with Type B's. A slightly different statistic--the odds ratio--is used for cross-sectional studies. The formulas for these two types of risk ratios are presented at the bottom of Table 1. Risk ratio indices indicate rate of change just as the slope in a regression equation indicates rate of change.

The chi-squared test is used to determine whether the association between TAB and CHD in the contingency table is statistically significant. The magnitude of the chi-squared statistic is influenced by both the sensitivity and the specificity. That is, larger values of the chi-squared statistic are associated with higher risk ratios.

#### Disease Based Spectrum (DBS) Bias in High Risk Studies

Spectrum bias is synonymous with range restriction. That is, spectrum bias is present when the variability on one variable is restricted so that the correlation between the variable with other variables is different than it would be if the entire range of values on the variable were examined. DBS bias occurs when disease status is used to select subjects for study independently of their risk factor status (Miller et al., 1988). When subjects are selected in this manner, the range of disease in the sample is

restricted. For example, the level of disease severity may not vary greatly between the high risk comparison group and diseased group if only individuals with severe disease are selected for study. This narrow spectrum of disease in the study sample may decrease any existing differences between the percentage of Type A's found in a high risk comparison group and a diseased group.

DBS bias may occur frequently in high risk studies. For example, Miller, Turner, Tindale and Posavac (1988) found evidence to suggest that the narrow spectrum of disease severity in angiography studies reduces correlations between TAB and CAD. Therefore, researchers may find no relation between TAB and CHD in high risk studies and falsely conclude that there is no relation between TAB and CHD.

Figure 1(b) can be used to describe DBS bias in terms of the DT model and to illustrate how DBS bias reduces statistical associations in high risk studies. For example, Figure 1(b) could represent the relation between TAB and CHD in a recurrent CHD study. The point labeled c' in Figure 1(b) represents the point at which individuals who have already incurred a MI develop sufficient CHD to incur recurrent CHD (angina or a second MI). A comparison of Figure 1(a) and (b) can be used to illustrate the difference between healthy population and recurrent CHD studies.

The spectrum of CHD in the high risk comparison group is attenuated in Figure 1(b). That is, the range of values on the x-axis is decreased in the recurrent CHD study. Similarly, other types of high risk studies exclude most healthy individuals from the study. Therefore, Figure 1(b) illustrates a situation that occurs in all types of high risk studies.

For the healthy population studies described in Figure 1(a), more Type B's than Type A's are present in the healthy range of individuals. Recall that the healthy and diseased range of individuals is defined by the  $\underline{c}$ . Thus, the individuals with low negative scores on the x-axis represent healthy individuals included in healthy population comparison groups.

Only individuals between  $\underline{c}$  and  $\underline{c}'$  are included in the high risk comparison groups used in recurrent CHD studies. For high risk studies, the specificity is estimated as the percentage of the sample located between  $\underline{c}$  and  $\underline{c}'$  that is Type B. More Type A's than B's are included in this range of the continuum of CHD. Subjects from the healthy end of the CHD continuum--where more Type B's are present--are excluded from recurrent CHD studies. Therefore, the specificity found in high risk studies is generally much lower than the specificity found in healthy population studies (Miller et al., 1988). The high percentage of Type A's found among those subjects in the high risk comparison

group lowers the specificity and the value of the chi-squared statistic. Therefore, high risk studies may find only a small association between TAB and CHD even when a strong relationship exists in the entire population.

An example may help to illustrate the degree to which DBS bias can attenuate statistical associations in high risk studies. Table 2 illustrates the healthy population and recurrent CHD study findings of the WCGS. For the healthy population results, the specificity (49%) is adequate and the study reported a statistically significant chi-squared statistic between TAB and CHD and the RR was 2.5. The specificity in the recurrent CHD study is much lower (30%), the relation between TAB and recurrent CHD is not statistically significant, and the RR is smaller (RR = 1.1).

### Selection Biases

Selection bias is defined as occurring when the "subjects' status on a hypothesized risk factor and the disease jointly determine which subjects are selected into the study" (Miller et al., 1988). That is, some third variable that is correlated with both the risk factor and the disease is used to select subjects.

For the current paper, one type of selection bias is used to illustrate how the DT model can be used to describe selection biases. This selection bias is referred to as age bias. Age bias is defined as occurring in research on TAB and CHD when TAB and CHD status jointly determine which

Table 2

Description of the WCGS at the 4.5 year follow-up

(a) Healthy population prospective results

	Healthy	Diseased	Total
Type A	1577	52	1598
Type B	1532	21	1584
Total	3109	73	3182

Percentage diseased = 2.3

Sensitivity = 71

Specificity = 49

RR = 2.5

d' = .373

Chi-squared test = 11.19 p < .0001

(b) Actual results for the recurrent CHD study of the WCGS at 4.5 years.

	Without Recurrent Disease	With Recurrent Disease	Total
Type A	114	23	137
Type B	56	10	66
Total	170	33	203

Estimated results

Sensitivity = 75%

Specificity = 30%

RR = 1.2 d' = .37

Chi-squared = p > .05

Actual results

Sensitivity = 70

Specificity = 33

RR = 1.1 d' = .14

Chi-squared = p > .05

subjects are included in the study sample as a function of the age of the study sample. For the current paper, an example of how age bias could occur in research on TAB and CHD was used to illustrate how age bias can alter the magnitude of associations between risk factors and diseases.

The DT model in Figure 2 can be used to describe age bias. Figure 2 illustrates how the percentage of Type A's and B's with CHD changes the sensitivity and degree of statistical association between TAB and CHD for the same men when they are examined at different ages in a healthy population prospective study.

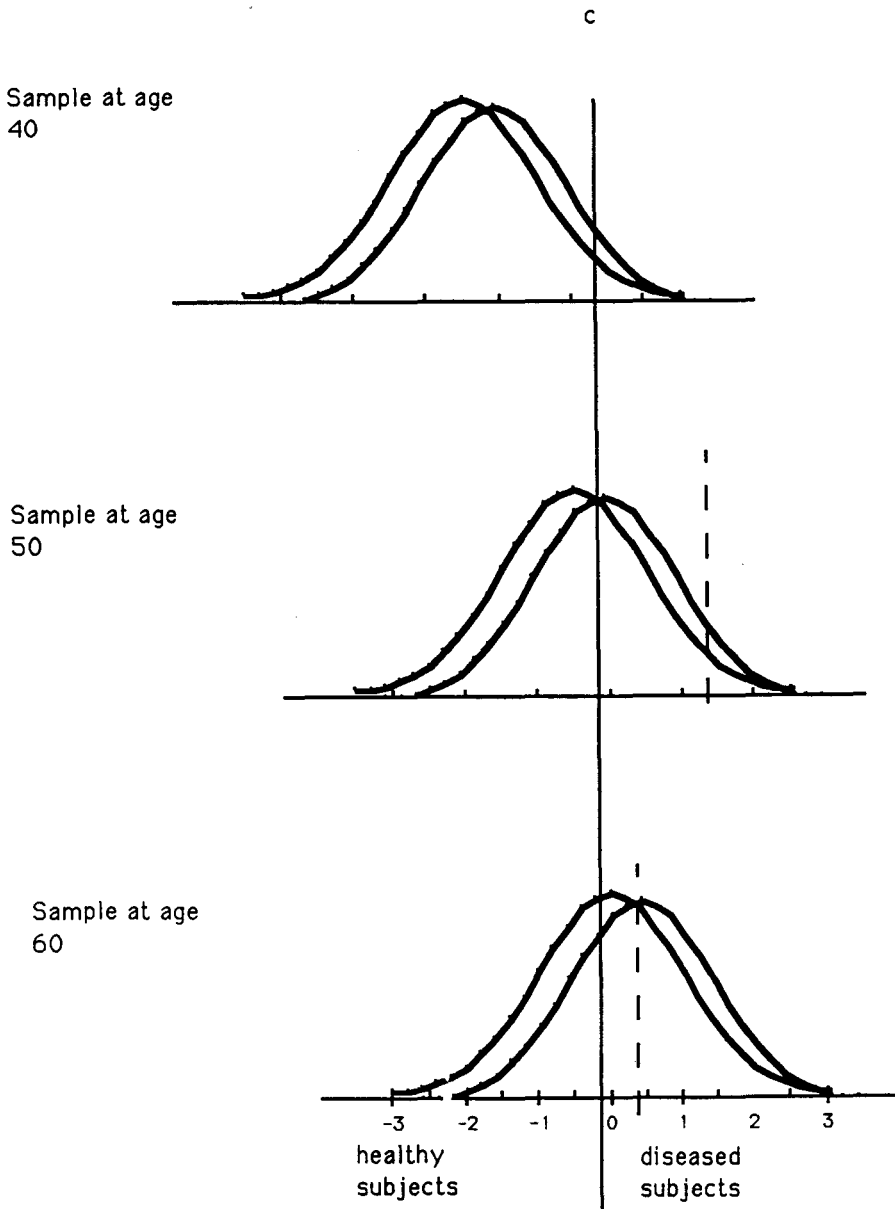
Figure 2 illustrates the development of CHD in a hypothetical sample of men who all eventually develop CHD. The degree of CHD in the sample is illustrated when the men are 40, 50 and 60 years old. Over half the sample has CHD when the patients are 60 years old. In contrast, less than a quarter of the individuals have CHD when the subjects are 40 years old.

The percentage of individuals with CHD who are Type A's varies with each age group. For the forty year olds, most of the individuals with CHD are Type A's. Thus, the sensitivity is very high. The new cases of CHD at age 50 and 60 are the subjects that represented in the section of the distribution that is defined by  $c$  as healthy at age 40 but are defined as having CHD at age 50 (see Figure 2). In



Figure 2

A DT Model of a Prospective Study



Notes. As in Figure 1, the distribution with more CHD represents Type A's, the distribution with less CHD represents Type B's and the x-axis indicates the severity of CHD.

the 50 year old group, a higher percentage of Type B's have developed CHD. Finally, more of the individuals who developed CHD after they were 50 are Type B's than Type A's.

Figure 2 illustrates that most Type A's will incur CHD when they are younger than Type B's. Therefore, one would expect that most Type A's would be younger than Type B's who are selected for recurrent CHD studies. Individuals who develop CHD when they are younger tend to have less severe CHD than individuals who develop CHD when they are older. These younger and hardier individuals with CHD may survive longer after their initial MI and not die within the time frame of the recurrent CHD study. Therefore, one may find an inverse association between TAB and recurrent MI.

## A REVIEW OF EXPLANATIONS FOR NULL FINDINGS

### Hypotheses Proposed by Previous Researchers

At present, no entirely satisfactory explanation for these null findings has been proposed (Dimsdale, 1988; Matthews, 1988). Several hypotheses have been forwarded. The current section frames these hypotheses in terms of the DT model.

Diseased based spectrum (DBS) bias. Matthews (1988) provided one clue to a possible explanation. She noted that most of the null findings have occurred in high risk studies. In support of her hypothesis, Miller et al. (1988) found evidence suggesting that DBS bias has produced the null findings in research on TAB in angiography studies. Perhaps, DBS bias accounts for some of the confusion in research on TAB and CHD. That is, high risk studies have DBS bias. Therefore, high risk studies find null results because DBS bias attenuates correlations between TAB and CHD in these studies. In contrast, studies not vulnerable to DBS bias find positive results.

Selection bias. There are other possible explanations for the trend towards null findings in research on TAB and CHD. The study designs that have been used in research on TAB and CHD are vulnerable to a number of different types of selection biases (Matthews, 1988). Thus, the method used to

select subjects may be associated with the magnitude of the  $d'$  between TAB and CHD.

Several third variables have been implicated in research on TAB and CHD. For example, some (e.g., Rosenman, 1986) have suggested that the culture or sex of the study population may mediate the strength of the relationship between TAB and CHD.

Insufficient sample size. Booth-Kewley and Friedman (1987) proposed another plausible explanation for the null findings. Their research suggests the null findings may have occurred because the correlation between TAB and CHD is low. That is, some studies may have used a small sample size that lacked sufficient statistical power to detect the presence of the low correlation between TAB and CHD.

Disease criteria. The location of the  $c$  can influence the strength of the association between two variables (Rorer, Hoffman, & Hsieh, 1966). Each different disease criteria has a different  $c$ . Therefore, different disease criteria may influence the magnitude of the relation between TAB and CHD. In fact, Miller et al. (1988) reported evidence suggesting that different methods of defining CAD influence the magnitude of the relation between TAB and CAD.

Indicator of TAB. Many researchers (e.g., Rosenman, 1986; Williams et al., 1988) have suggested that the magnitude of the relationship between TAB and CHD may vary for different measures of TAB. In terms of the DT model,

the  $d'$  between a measure of TAB and CHD may vary for different measures of TAB.

### The Current Research

The current research attempts to identify the study characteristics (i.e., high risk studies, method used to select subjects, sample sizes, culture, sex, disease criteria, indicator of TAB) that are associated with null findings by a quantitative review. In addition, reanalyses of some studies are conducted to test whether the signs of DBS bias identified by Miller et al. (1988) are present in all types of high risk studies. The presence of these signs would suggest that DBS bias has produced the null findings in high risk studies.

The current research is theoretically important because a DT model is developed and tested that can be used to identify subject selection biases in research on TAB and CHD. In addition, the current research attempts to identify study design characteristics that play an important role in mediating the strength of the relationship between TAB and CHD. These mediating variables may have important theoretical implications for understanding why TAB has been found to be associated with CHD in some studies.

The current research also has practical significance. The identification of the study design characteristics that mediate the relationship between TAB and CHD may lead to an increased understanding of the reasons for the trend towards

null findings. In addition, subject selection biases may produce misleading results. The current research attempts to identify study designs that lead to misleading findings. Therefore, the current research has implications for the types of study designs that future researchers can use to obtain the less biased estimates of the magnitude of the relationship between TAB and CHD.

## METHOD

### Locating Studies

Most of the studies included in the current review have been used in previous reviews (Booth-Kewley and Friedman, 1987; Matthews, 1988; Miller et al., 1988). Additional articles were found using Booth-Kewley and Friedman's (1987) procedures for locating studies. Briefly, these procedures include searching through journals that have published known articles and using computerized searches such as Psychological Abstracts and Medicus Indicus to find new articles. An attempt was made to include all studies that were published before 1989. Appendix A includes a list of new articles presented in neither Booth-Kewley and Friedman's, Miller et al.'s nor Matthews' reviews.

### Criteria for Including Articles

Study selection criteria. Studies were included in the current review only if an article reported an attempt to find a statistical relation between CHD and some measure of TAB. In addition, the current review was limited to articles published in the English language. For the current research, only used at one of three types of indicators of CHD were used in the current study: (a) MI, (b) MI or angina (this category is referred to as CHD for the remainder of this paper) or (c) CAD from the results of an angiography. This selection criteria for CHD is slightly narrower than

the criteria used by Booth-Kewley and Friedman (1987). In contrast, to Booth-Kewley and Friedman, studies that assessed CHD by infrequently used disease outcomes were not included in the current review. For example, only a few studies (see Booth-Kewley & Friedman, 1987) have used angina as a disease outcome. For such studies, insufficient data were available to perform the detailed reanalyses and comparisons across different study designs conducted in the current review.

Similarly, an insufficient number of studies were available to examine other psychological predictors of CHD (e.g., hostility, depression or subcomponents of TAB such as time urgency). Thus, studies that only used these predictor variables were not included in the current review.

The Friedman et al. (1986) true experimental study (Brackett & Powell, 1988) is not included in the current review. This report collapsed results across a treatment and control group. This analysis strategy is inappropriate because the treatment successfully modified TAB. Therefore, collapsing across treatment and control conditions confounds the results.

In addition, the results of one study (Williams et al., 1988) were not included because this study was a follow-up of the Williams et al. (1980) study and not an independent study. The findings of the earlier study were used because



a correlation coefficient ( $r$ ) could be calculated from the 1980 report and not from the 1988 report.

Finally, three studies (Friedman & Rosenman, 1959; Rosenman & Friedman, 1961; Schwertner, Troxler, Uhl, & Jackson, 1984) selected extreme Type A's and B's for study. Selecting extreme groups is a useful method to insure that the constructs of interest are represented in the sample. However, selecting extreme groups also increases the magnitude of the  $r$  between the two variables. In fact, all three studies found the largest  $r$ 's between TAB and CHD of any of the studies included in the current review. Thus, the results of these studies support the hypothesis that TAB is associated with CHD. However, the  $r$ 's for these studies are not comparable with other studies and so the results of these studies were not included in the current review.

Selection criteria for prevalence estimates. The sex or age composition of the sample may mediate the strength of association between TAB and CAD (Rosenman, 1986). To avoid the problem of confounding results for sex and age, the current research only used prevalence estimates of TAB from studies of middle-aged men.

Selection criteria for studies used to test for the presence of DBS bias. The current research only tested for signs of DBS bias in studies that assessed TAB by the SI. There were two reasons why only these studies were examined.

First, the SI is the measure of TAB that is the most highly associated with CHD (Booth-Kewley & Friedman, 1987).

Second, only studies that assessed TAB by the SI presented their results in sufficient detail for reanalysis. The SI has often been treated as a dichotomous variable. Therefore, the results have been presented in contingency tables that yield sufficient information for reanalysis. In contrast, most studies that used self-report measures of TAB have typically reported their results only in terms of tests of significance that provide insufficient information for reanalysis.

### Statistical Analyses

Use of biserial and tetrachoric  $r$  coefficients. In the current review, the correlation coefficient ( $\underline{r}$ ) was used as the indicator of effect size. The  $\underline{r}$  was used so as to allow comparison with other meta-analyses of research on TAB and CHD that used  $\underline{r}$  to indicate the effect size (e.g., Booth-Kewley & Friedman, 1987). The Pearson  $\underline{r}$  underestimates the degree of association between two variables when the base rate for an artifactually constructed dichotomous variable is low (see Cohen & Cohen, 1984; Hunter, Schmidt & Jackson, 1982; Kemsey, Dunlap & Griffeth, 1988). Both TAB and CHD are artifactually constructed dichotomous variables. In prospective and population studies, the base rate for CHD is low. Therefore, the magnitude of the Pearson  $\underline{r}$  between TAB and CHD would vary with type of study design even if the

magnitude of the actual  $r$  between TAB and CHD does not vary with study design. To avoid the base rate problem, biserial and tetrachoric  $r$ 's were calculated from the results reported in the original studies. The magnitude of these coefficients is not influenced by the base rate. The biserial coefficient is used when one variable is dichotomous and one variable is continuous. The tetrachoric coefficient is used when both variables are dichotomous.

Controlling for sampling variability. A weighted mean  $r$  (WMR) was estimated by multiplying each individual study  $r$  by the study sample size and taking the average of the products. Matthews (1988) and others (e.g., Hedges & Olkin, 1985; Hunter et al., 1982) have argued that the weighted mean is more appropriate than an unweighted mean as an indicator of the average effect size across a series of studies. The WMR adjusts for the sampling variability produced by studies of different sample sizes. Sampling variability could be a severe problem in the current research because sample sizes varied from 25 to 11,364.

Methods used to sum  $r$ 's. Eleven studies reported null results with no specific information that could be used to calculate an  $r$ . As recommended by meta-analysts (Hedges & Olkin, 1985), the  $r$  was considered to be equal to zero in these cases. To compute WMR's,  $r$ 's were transformed to Fisher  $z$ 's, weighted by study sample size and then transformed back into the  $r$  metric<sup>3</sup>.

Testing for inter-study variability. An inter-study variation index (IVI; Hunter et al., 1982) was used in the current research to ascertain the homogeneity of summed study effect sizes. The IVI is an index of the percentage of inter-study variation in study  $\bar{x}$  that is beyond the amount explained by sampling variability. A large IVI suggests that the studies are not comparable and the magnitude of the WMR may be misleading.

### Study Variables

Disease criteria. Four different disease outcomes were examined in the current review: (a) CHD, (b) MI, (c) CAD and (d) fatal MI. In addition, the CAD category was divided into several subcategories according to the CAD scoring method; these subcategories for CAD were used because different CAD scoring methods appear to have a small influence on the magnitude of the  $\bar{x}$  between TAB and CAD (see Miller et al., 1988).

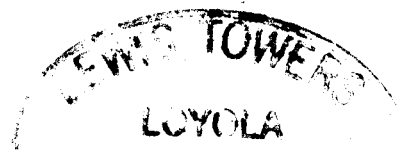
Measures of TAB. Measures of TAB were divided into three categories: (a) the structured interview (SI; Rosenman, 1978), (b) the Jenkins Activity Survey (JAS; Jenkins, Zyzanski, & Rosenman, 1971) and (c) other self-report measures. The JAS has been used in a sufficient number of studies to compare results across different study designs. Therefore, studies using the JAS are considered separately from other less frequently used self-report measures.

Study design. Every study was classified as either a cross-sectional or prospective design, and as either a healthy population or high risk design based on the definitions presented in the review section. In addition, study designs were classified according to how subjects were recruited for study.

Culture. The prevalence of TAB appears to be related to culture (Rosenman, 1986). Therefore, the results were examined separately for studies conducted within the United States and studies conducted in other countries.

Sex. The sex of the subject may mediate the strength of the relationship between TAB and CHD because sex is correlated with both TAB (Rosenman, 1986) and CHD. Although most studies only examined men, some studies included women. For studies reporting results for men and women separately, only the results for men were used when summing  $r$ 's to calculate WMR's. In studies that only reported combined results for both men and women, the percentage of the sample that was female was recorded and an attempt was made to determine if the percentage of females in these studies was correlated with the magnitude of the study  $r$  between TAB and CHD. An association between sex and study  $r$  would suggest sex mediates the relation between TAB and CHD.

Year of publication. The year 1978 was chosen as a point at which to divide the studies into two groups. Studies published before or during 1978 were considered to



be early studies and studies published after 1978 were categorized as more recent studies. This year was chosen because it was the year of the NHLBI's review. Presumably, any influences over time occur because of historical trends. Therefore, the time the data were collected would be a more exact indicator of the history of the study. Unfortunately, many studies did not report this information so the year of publication was chosen as the best available proxy.

The findings of some studies were reported in several articles in which some were published before and others after 1978. In these cases, the study was classified as occurring before 1978 because the earliest publication date is nearest to the time the data were collected.

#### Unit of Analysis

One purpose of the current review was to determine why some studies have reported null findings. Presenting results in smaller subunits (e.g., estimating different  $r$ 's for different age groups within a single study) would not be consistent with this purpose. That is, identifying differences produced by different subpopulations is not the purpose of the current research. Therefore,  $r$ 's were not estimated for subgroups within a single study. A total of 61 independent studies were included in the current review.

When summing across  $r$ 's, only the results of independent studies were combined. When a single study reported more than one result, several decision rules were

used to decide which  $r$  should be used for the meta-analysis. First, previous research (Booth-Kewley and Friedman, 1987) has established that the  $r$ 's between TAB and CHD and between TAB and MI are similar. Therefore, the results of studies that used either of these two disease outcomes were combined. Only the  $r$  for the CHD outcome was used to avoid double weighting in studies that reported results for both CHD and MI. In addition, only the results of the longest follow-up period were used to calculate a  $r$  to insure that the results of prospective studies were not double weighted.

Some of the analyses presented in the current review examined the association between study characteristics (e.g., study design) and number of positive (statistically significant) versus null findings. A few studies were difficult to code for these analyses because a  $r$  could be calculated for more than one study characteristic. For example, a study might be weighted twice if the results were reported for both prospective and cross-sectional designs or for two different measures of TAB. In these cases, the study was treated as one finding if the results were the same for all study characteristics; otherwise, the study was not used in the analysis. The exclusion of studies did not lead to missing data problems. No more than three studies were excluded from an analysis because the studies reported mixed results.

## Testing for the Presence of DBS Bias

DBS bias is defined as occurring when the range of disease in the sample has been restricted to subjects with moderate to severe disease (Miller et al., 1988). Thus, DBS bias is present in studies in which completely healthy individuals are not selected for study. DBS bias produces null findings to the extent that the level of disease in the high risk comparison group is equivalent to the level of disease in the diseased group. As the spectrum of disease across the two groups narrows, the percentage of Type A's in the high risk comparison and diseased groups becomes equivalent so there is no significant difference in the percentage of Type A's found in these two groups.

There are two signs of DBS bias that researchers can test for. One would have evidence to suggest that DBS bias has produced the null findings in high risk studies if these signs are present. One sign of DBS bias is a high percentage of Type A's found in both the diseased and comparison groups used in high risk studies and a low percentage of Type A's found in healthy populations. This pattern of results would suggest more Type A's than Type B's develop CHD. However, this finding is obscured in high risk studies in which all of the subjects have some CHD because a high percentage of Type A's are found in both the high risk comparison group and the diseased group in high risk studies.



A second way to test whether DBS bias may have produced the low  $\underline{r}$  in recurrent CHD studies is to determine if range restriction can account for the reduced  $\underline{r}$ 's found in these studies. The  $\underline{r}$  obtained from recurrent CHD studies can be considered to have two parts: a part produced by range restriction and a part not attributable to range restriction (e.g., sampling variability, other biases). One can use a formula that estimates the attenuation in  $\underline{r}$  that would occur in a high risk study given a healthy population  $\underline{r}$  and an estimate of the percentage of the population that is excluded from the study sample. This formula is referred to as a range restriction formula. One can compare the  $\underline{r}$  obtained from the range restriction formula with the actual  $\underline{r}$  between TAB and CHD. A significant difference between the estimated and actual  $\underline{r}$ 's would suggest that study characteristics other than range restriction have contributed to the small  $\underline{r}$ 's found in high risk studies. In contrast, range restriction may have produced the smaller  $\underline{r}$ 's found in high risk studies if the estimated results are not significantly different from the actual results.

Testing for a higher percentage of Type A's in high risk populations. The binomial z test (for a discussion, see Miller et al., 1988) was used in the current research to determine whether significantly higher percentages of Type A's were present in high risk populations than in healthy populations. The binomial z test requires an accurate

estimate of the percentage of Type A's found in a healthy population. Therefore, a review of the literature was conducted to obtain an accurate estimate.

Estimating the reduction in the magnitude of the  $r$  produced by range restriction in recurrent CHD studies.

Alexander, Carson, Alliger and Barrett (1985) provide a range restriction formula that can be used to estimate the reduction in  $r$  in high risk studies<sup>4</sup>. The formula is used in the current research to estimate the reduction in  $r$  between TAB and CHD in a recurrent CHD study from the  $r$  obtained in a healthy population study.

The range restriction formula requires a healthy population estimate of the  $r$  between a disease and a risk factor. Unfortunately, no estimates of the  $r$  between TAB and CAD from healthy population studies are available. Therefore, no range restriction formulas were calculated for angiography studies. For recurrent CHD studies, the range restriction formula requires an estimate of the percentage of healthy subjects who are expected to incur a MI in the future and an estimate of the  $r$  between TAB and MI found in a healthy population study. The estimates used for the range restriction formula for the recurrent CHD studies were from the healthy population prospective results of the WCGS (4.3% MI rate and  $r = .33$ ).

Distinguishing between DBS bias and other subject selection biases. DBS bias can often be distinguished from other types of subject selection biases (Miller et al., 1988). One would suspect that DBS bias has produced null findings when the percentages of Type A's across diseased and comparison groups only vary with whether the study used a high risk or healthy population design. Alternatively, one would have evidence that other subject selection biases are present if the study  $r$ 's vary with the way subjects were selected. For example, high risk studies have selected subjects with CHD by four different methods: (a) selection on the basis of their risk factor status, (b) selection by the results of an angiography in which subjects were diagnosed as having clinically significant CAD, (c) selection because an individual developed CHD during the course of a prospective study or (d) selection because one worked in a company participating in a study.

The percentage of Type A's found in these high risk studies would be expected to vary with these different subjects selection methods if subject selection biases other than DBS bias have produced the null findings in high risk studies. To determine the extent to which subject selection biases may have reduced the relation between TAB and CHD, the current research examined whether the prevalence of TAB and the  $r$  between TAB and CHD varied with the method used to select subjects. One would have evidence that DBS bias has

produced the null findings in high risk studies if the r between TAB and CHD and the prevalence of TAB varies with whether the study was high risk or healthy population. Other subject selection biases may be present if the r between TAB and CHD and the prevalence of TAB varies with how the subjects were selected.

## RESULTS

### study Characteristics Associated with the Trend Towards Null Findings

The results of the current research support Booth-Kewley and Friedman's (1987) finding of a significant trend towards null findings in research on TAB and CHD. As indicated by the Fisher exact test, studies included in the current review were significantly more likely to report positive findings before 1979 for both the SI ( $p < .05$ ) and self-report measures ( $p < .005$ ). There were four times as many studies reporting null findings after 1978 for studies that assessed TAB by the SI and eight times as many studies reporting null findings for studies that used self-report measures.

Table 3 illustrates that three types of studies have been associated with null findings: (a) high risk studies, (b) studies that used self-report measures and (c) studies that used fatal MI as a disease criterion. All three of these types of studies have been conducted more frequently after 1978. Thus, all three types of studies have contributed to the trend towards null findings.

High risk studies. High risk studies accounted for the majority of the null findings. Among the 39 independent studies that reported null findings, 24 were high risk studies. High risk studies had significantly more null findings--by the Fisher exact test--than healthy population

Table 3

Summary of the Characteristics Studies with Null Findings

	HIGH RISK SI STUDIES	HEALTHY SELF-REPORT	BOTH SELF-REPORT AND HIGH RISK	FATAL MI	INADEQUATE SAMPLE SIZE	UNKNOWN
Number of studies with null findings	16	8	17	8	2	1
Percentage of studies that reported null findings	84	30	89	100	-	-
Percentage of studies published after 1978	81	79	87	100	100	100

Notes. The total number of independent studies that reported more null results than positive results was 30. HIGH RISK SI STUDIES are studies that assessed TAB by the SI and used a high risk study design. HEALTHY POPULATION SELF-REPORT studies used a self-report measure and a healthy population study design. Two studies clearly used inadequate sample sizes. Appels and Mulder (1985) only examined 18 subjects with CHD. Keefe, Castell and Blumenthal (1986) had a total sample size of 25. The UNKNOWN column represents one study that did not fall into any of the other categories. INADEQUATE SAMPLE SIZE and UNKNOWN could be any studies in the sample. Therefore, the percentage of studies that reported null findings is left blank. The UNKNOWN study was a case-control by Wielgosz, Wielgosz, Biro, Nichols, MacWilliam, and Haney (1988) that used the SI to assess TAB.

studies for all studies that assessed TAB by the SI ( $p < .005$ ) and by self-report measures ( $p < .005$ ). Overall, 24 independent high risk studies reported null results, three reported mixed results and only four reported positive results.

There was a trend towards null findings for all of the different types of high risk studies. Before 1979, three out of four angiography studies reported positive results. After 1978, only one study reported a positive result, three reported mixed results for different measures of TAB and 15 reported null results. Similarly, only one of the five recurrent CHD studies were conducted before 1979. One study conducted after 1978 reported mixed results and the other studies reported null results. Finally, two studies that selected subjects on the basis of their traditional risk factor status also reported null findings and were published after 1978. In sum, most high risk studies were conducted after 1978 and reported null results.

#### Healthy population studies that assessed TAB by the SI.

In contrast to high risk studies, healthy population studies that used the SI were associated with positive findings. For studies that used the SI, six of eight reported positive findings. Although there has been an increase in the number of high risk studies in recent studies, there has not been an increase in the number of healthy population studies that assessed TAB by the SI in recent years. Only two

prospective studies used the SI to assess TAB since 1978. In one of these studies (Appels & Mulder, 1985), the sample size was clearly insufficient (only 18 subjects with CHD) and the study reported null results. Thus, only one study in recent years has assessed whether the SI is a predictor of CAD and this study reported a positive result.

Studies that assessed TAB by self-report measures. The use of self-report measures in healthy population studies has also contributed to null findings in recent years. For studies that used self-report measures, 15 of 26 studies reported positive findings, three studies reported mixed results, and eight reported null findings. Only three of eight healthy population prospective studies that assessed TAB by self-report measures since 1978 reported positive results.

There has been an increase in the number of studies using self-report measures in recent years. For studies that used self-report measures, 19 of 26 healthy population studies were conducted after 1978.

Table 4 presents the number of studies, percentage of significant findings, WMR's, IVI's, confidence intervals (CI's) for the WMR's and the study  $N$  for each type of study design and measure of TAB. The table illustrates how study design and measures of TAB are related to the percentage of significant findings and the magnitude of the WMR.



Table 4

Findings for Each Type of Study Design

	Prospective		Cross-sectional		
	High risk	Healthy population	High risk	Healthy population	
	Recurrent	Population	Angiography	Case-	Population
	CHD			Control	
	Studies That Used the SI				
# of studies	4(4)	3(3)	13(11)	5(5)	1(1)
% of positive findings	0	67	23	80	100
WMR	.12	.33	.13	.35	.31
IVI	.001	.013	.168	.126	.000
CI	.06-.18	.31-.35	.09-.17	.31-.39	.28-.34
Total N	954	4,298	2,348	605	3,524
	Studies That Used the JAS				
# of studies	4(4)	7(4)	9(5)	7(7)	7(5)
% of positive findings	25	43	22	86	86
WMR	.02	.04	.00	.26	.07
IVI	.013	.003	.027	.006	.004
CI	-.02-.06	.03-.05	-.05-.05	.20-.32	.05-.09
Total N	2,308	18,330	1,367	933	10,101

Table 4 continued

Prospective		Cross-sectional	
High risk	Healthy population	High risk	Healthy population
Recurrent CHD	Population	Angiography Case-	Population
			Control

Studies That Used Other Self-report Measures

# of studies	0	5(4)	8(3)	4(3)	3(3)
% of positive findings		40	13	75	33
WMR		.03	.00	.21	.19
IVI		.015	.018	.031	.001
CI		.01-.05	-.03-.03	.12-.30	.17-.21
Total N		16,085	3,395	481	13,668

**Notes.** # of studies = the number in parentheses represents the number of reports in which an  $r$  could be calculated. The number not in parentheses is the total number of studies; % of positive findings = the percentage of studies that reported a significant positive association between TAB and CHD; IVI = percentage of variation in  $r$  across studies that cannot be attributed to sampling error; CI = 95% confidence interval; Total N = total number of subjects used to estimate the WMR's.

A small  $r$  between self-report measures of TAB and CHD appears to have produced some of the null findings. Prospective studies that assessed TAB by self-report measures were consistently associated with small  $r$ 's. The WMR's for these studies varied from .04 to .00 (see row in Table 4 labeled "WMR"). Many healthy population studies (30%; see Table 3) found null results. The reason for the larger WMR's in the cross-sectional healthy population studies is not clear<sup>5</sup>.

Studies that used fatal MI as a criterion. Table 5 presents the results for studies that used fatal MI as a disease criterion. All eight studies found no statistically significant association between TAB and fatal MI. In most of these studies, the  $r$  between TAB and fatal MI was in the negative direction. Thus, using fatal MI as a criterion produces results that are markedly different from studies using other disease criteria. The use of fatal MI has contributed to the trend towards null findings because all eight studies were published after 1978.

#### Evidence of DBS bias in High Risk Studies

Evidence of smaller  $r$ 's in high risk studies. One would expect smaller  $r$ 's in high risk studies than have been found in healthy population studies if DBS bias is present because DBS bias is a type of range restriction that may attenuate  $r$ 's in high risk studies. In fact, the  $r$  between TAB and CHD does appear to be larger in healthy population

Table 5

Studies that Used Fatal MI as a Criterion

Measure	Author	Year	N	r
-----	-----	----	-	-
<b>Healthy Population Prospective Studies</b>				
SI	Ragland	1988b	3,154	.09
Bortner	Mann	1987	7,214	-.07
FRHM	Eaker	1988	337	-.27
-----				
<b>High Risk Prospective Studies</b>				
SI	De Leo	1985	68	-.21
SI	Shekelle	1985	3,110	.03
SI	Ragland	1988a	231	-.34
JAS	Dimsdale	1981	189	
JAS	Case	1985	516	-.11
Unique	Ruberman	1984	2,320	

**Notes.** Measure = the measure of TAB used; Author = the first author of the article; Year = the year of publication of the article reporting the study findings; N = study sample size; FRHM = The Framingham Type A scale; Unique = a measure of TAB was used that has not been used in any other study. WMR's are not reported because of the substantial inter-study variation that is probably produced by the use of different measures of TAB. Appels and Mulder (1985) reported four fatal MI cases in their sample. All four were Type A. Koskenvuo, Kaprio, Langinvainio and Romo (1983) reported an analysis of fatal MI in which only three subjects had fatal MI.

studies than high risk studies. There is a moderate  $r$  between the SI and CHD in healthy population studies (see row in Table 4 labeled "WMR"; prospective healthy population = .33; case control = .35; cross-sectional population = .31). These WMR's are among the largest  $r$ 's presented in Table 4. In contrast, the WMR's for high risk studies that assessed TAB by the SI were smaller (recurrent CHD = .12; angiography = .13). A similar pattern of smaller  $r$ 's is found in studies that used self-report measures (see Table 4). Evidence of a lower percentage of Type A's in healthy populations. One would expect a lower percentage of Type A's in healthy populations than among individuals with CHD if TAB is associated with CHD. Table 6 presents the percentage of Type A's found in all healthy population studies that assessed TAB by the SI and that reported results in sufficient detail to estimate the prevalence of TAB. The results in Table 6 indicate that the percentage of Type A's found among healthy middle-aged American men is  $46\% \pm 1\%$  ( $N = 5,383$ ;  $IVI = .001$ ).

Evidence for the influence of culture. TAB is supposedly a product of the aggressive American lifestyle (Rosenman, 1986). In support of this hypothesis, the percentage of Type A's found in populations outside the United States appears to be somewhat lower. In these studies, the percentage of Type A's varied from 27% to 52% and was generally lower than the studies conducted in the

United States (see Table 6 column labeled "% of Type A's"). Similarly, the percentage of Type A's in high risk studies conducted outside the United States ( $60\% \pm 3\%$ ;  $N=1,111$ ;  $IVI = .016$ ) was lower than in studies conducted in the United States (see Table 7).

In spite of these base rate differences, the  $r$ 's between TAB and CHD in countries that were conducted outside the United States are similar in magnitude to those studies that were conducted in the United States. The IVI's for the WMR's presented in Table 4 are generally very small suggesting that there is little inter-cultural variation in WMR's. For example, there appears to be no substantial difference in the  $r$  between healthy population studies conducted in the United States that assessed TAB by the SI ( $WMR = .32 \pm .02$ ;  $N=3,443$ ;  $IVI = .009$ ) and studies conducted outside the United States ( $WMR = .36 \pm .03$ ;  $N=1,460$ ;  $IVI = .004$ ).

In sum, cultural differences among subjects do not appear to influence the magnitude of the  $r$  between TAB and CHD. Thus, WMR's are reported for all countries combined. However, culture is related to the prevalence of TAB. Therefore, prevalence rates for the SI are only reported for studies conducted in the United States in the current paper.

Table 6

The Prevalence of Type A's in Healthy Population Studies that Assessed TAB by the SI

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**Studies Conducted in the United States**

Author	Year	% of Type A's(N)
Keith+	1964	31(69)
Caffrey	1969	42(1521)
Rosenman	1975	49(2897)
Bryne	1985	46(582)
Moss	1986	47(314)

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**Studies Conducted Outside the United States**

Author	Year	% of Type A's(N)	Country
Kittel	1978	38(726)	Belgium
Verhagen+	1980	43(58)	Netherlands
Orth-Gomer+	1980	27(48)	Sweden
Appels	1987	48(191)	Netherlands
Wielgosz+	1988	52(71)	Canada

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Notes. Author = the first author of the article that reported the study findings; Year = the year of publication of the article reporting the study findings; % of Type A's(N) = the number not in parentheses indicates the percentage of Type A's and the number in parentheses indicates the number of subjects the percentage estimate was based on; Country = the country in which the study was conducted. The weighted mean percentage of Type A's for studies conducted in the U.S.A. was 46% (N = 5,383). The weighted average prevalence of TAB for countries other than the United States was not calculated because of substantial intra-study variation (IVI = .307).

Table 7

Percentage of Type A's Found in High Risk Studies

Author	Year	Disease criteria	% Type A's in		Country
			Diseased Group	Comparison Group	
Angiography Studies					
Blumenthal	1978	TOTCI	80(64)*	44(78)	U.S.A.
Krantz	1981	TOTCI	78(49)*	71(34)	U.S.A.
Dembrowski	1985	TOTCI			U.S.A.
Blumenthal	1985	TOTCI			U.S.A.
Frank	1978	>50%	80(118)*	45(29)	U.S.A.
Krantz	1979	>50%	67(45)*		U.S.A.
Dimsdale	1979	>50%	64(87)*	62(16)	U.S.A.
Scherwitz	1983	>50%			U.S.A.
Williams	1980	>75%	79(285)*	67(139)*	U.S.A.
Arrowood	1982	>75%			U.S.A.
Siegman	1987	>75%			U.S.A.
Blumenthal	1987	>75%	60(92)*	76(21)	U.S.A.
Murphy	1985				U.S.A.
Langeluddecke	1988	>50%	46(405)	41(114)	Australia



Table 7 continued

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Recurrent CHD studies					
Rosenman	1967	CHD	70(33)*	67(170)*	U.S.A.
Krantz	1979	CAD	77(13)	62(32)*	U.S.A.
Rechnitzer	1983	MI	76(79)*	70(539)*	Canada
De Leo	1986	MI	44(88)	43(51)	Italy

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Studies that selected on the basis of risk factor status					
Shekelle	1985	MI	73(129)*	74(2991)*	U.S.A.
Belmaker	1976	CHD	68(34)*	70(44)*	U.S.A.

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Notes. \* = the test of the hypothesis that the percentage of Type A's is greater than 47% is statistically significant by the binomial  $z$  test that is corrected for continuity and adjusted by the Bonferroni criteria (Individual alpha = .05/12 = .004); Author = the first author on the article that reported the study findings; Year = the year of publication of the findings of the study; CAD = increase in severity of CAD; >50% and >75% are CAD scoring methods in which only subjects with no occluded coronary arteries greater than 50 or 75% occlusion are classified as healthy; TOTCI is another CAD scoring method (Blumenthal et al., 1985); % of Type A's = the numbers not in parentheses indicate the percentage of Type A's and the numbers inside the parentheses indicate the number of subjects used to estimate the percentages of Type A's.

Evidence of a higher prevalence of TAB in high risk comparison and diseased groups. One would expect that a higher percentage of Type A's would be found in both the high risk comparison and diseased groups in high risk studies than in healthy populations if DBS bias has produced the null findings in high risk studies. The findings of the current research support this hypothesis.

Table 7 presents the percentages of Type A's in the diseased groups and the high risk comparison groups in high risk studies. In addition, Table 7 displays the binomial  $z$  test of the difference in the percentage of Type A's found in healthy population studies versus diseased comparison groups. A statistically significant binomial  $z$  test is indicated by a "\*" in Table 7. The upper bound of the confidence interval (47%) of the percentage of Type A's found in the healthy population studies was used as an estimate for the binomial  $z$  tests conducted in the current research. The test of the hypothesis that the percentage of Type A's is greater than 47% is statistically significant by the binomial  $z$  test was corrected for continuity and adjusted by the Bonferroni criteria (Individual alpha =  $.05/12 = .004$ ).

For studies conducted in the United States, a significantly higher percentage of Type A's than the 47% healthy population estimate were found in all but one study. A small sample size appears to be the reason for the lack a

statistical significance in the one study ( $N = 13$ ). The percentage of Type A's found in this study is similar to the percentages found in the other studies. Thus, this study may also have had a statistically significant result if a larger sample size was used. In addition, the high risk comparison groups with sufficient sample sizes to obtain significant results had significantly more Type A's than found in healthy populations.

The percentage of Type A's found in the high risk comparison groups is similar to that found in the diseased groups. For the 1,349 subjects with diagnosed CHD or CAD in the studies conducted in the United States, 70% ( $\pm 2\%$ ;  $IVI = .006$ ) were Type A's; a much higher percentage of Type A's than the 46% found in healthy populations. For the high risk comparison groups, the percentage of Type A's was similar to the diseased groups varying from 62% to 76% (see columns labeled "% Type A's Comparison Group").

Evidence of range restriction. DBS bias is a type of range restriction. Thus, one would expect that range restriction formulas could estimate the reduced  $r$ 's found in recurrent CHD studies if in fact DBS bias has reduced  $r$ 's in these studies. The  $r$  calculated from the range restriction formula is .14 for recurrent CHD studies that assessed TAB by the SI. To estimate the reduction in  $r$  in high risk studies that used the JAS, the healthy population  $r$  between the JAS and CHD was assumed to be .04; this is the WMR for

all healthy population prospective studies (see Table 4). The estimated  $\underline{r}$  for studies that assessed TAB by the JAS is .02.

Table 8 presents the expected  $\underline{r}$ 's estimated from the range restriction formula, the observed  $\underline{r}$ 's, the standard error associated with the 95% confidence interval of the observed  $\underline{r}$ 's and the absolute value of the difference between the expected and observed  $\underline{r}$ 's. Recall that the range restriction formulas used to derive the expected  $\underline{r}$ 's only use information regarding the  $\underline{r}$  found in healthy populations and the percentage of healthy subjects that have been excluded from the sample. Thus, the range restriction formula estimates the degree of range restriction that would be expected in a high risk sample given that a certain percentage of low risk subjects has been excluded from the study sample. Therefore, the restriction in range formula's results is independent of the actual study findings. The differences between the expected and observed  $\underline{r}$ 's in Table 8 are always smaller than the standard error. That is, the expected  $\underline{r}$ 's are within the 95% confidence intervals of the observed  $\underline{r}$ 's in every study. Thus, the expected  $\underline{r}$ 's estimated from the range restriction formulas accurately estimate the magnitude of the  $\underline{r}$  between TAB and CHD found in recurrent CHD studies. These results support the hypothesis that DBS bias has produced the reduction in  $\underline{r}$  found in recurrent CHD studies.

Table 8

Comparison of Observed r's between TAB and CHD in Recurrent CHD Studies with Expected r's

Author	N	Expected r	Observed r	Standard error	Absolute value of the difference
<b>Studies that assessed TAB by the SI</b>					
Rechnitzer	618	.14	.12	.08	.02
De Leo	88	.14	.17	.21	.03
Rosenman	203	.14	.05	.14	.09
<u>Weighted mean</u>	909	.14	.11		.03
<b>Studies that assessed TAB by the JAS</b>					
Jenkins	98	.02	.07	.20	.05
Shekelle	2070	.02	.02	.04	0
Appels	70	.02	-.11	.24	-.13
<u>Weighted mean</u>	2238	.02	.02		0

Notes. N = sample size; Expected r = the r estimated by the range restriction formula; Observed r = the r reported in the study; Standard error = the 95% confidence interval; Absolute value of the difference = the absolute value of the difference between the expected and observed r. Follow-up results for the 8.5 year WCGS for the JAS were not presented in a manner in which an r could be estimated. Therefore, the 4.5 year follow-up results by Jenkins et al. (1971) are reported in Table 8.

The base rates of MI in prospective studies of CHD conducted in this review varied from 6.2% to 2.2%. To test whether the results were influenced by the 4.3% base rate estimate used to calculate the expected  $\bar{r}$  by the range restriction formula, base rates from 10% to 1% were used to calculate different expected  $\bar{r}$ 's from the range restriction formulas. No observed  $\bar{r}$  was significantly different from any of the expected  $\bar{r}$ 's. Thus, the findings for the range restriction formulas appear to be robust with regards to the estimate of the base rate of CHD that is used.

Evidence of selection biases. One would expect the prevalence of TAB to vary with the way subjects were selected if selection biases have influenced research on TAB and CHD. The percentages of Type A's were similar across all different subject selection methods for subjects with CHD in the studies included in the current review. Subjects were selected by four different methods: (a) selection on the basis of risk factor status ( $72 \pm 7\%$  ;  $N = 163$ ;  $IVI = .065$ ), (b) selection by the results of an angiography in which subjects were diagnosed as having clinically significant CAD ( $74 \pm 3\%$  ;  $N = 740$ ;  $IVI = 0$ ), (c) selection because the individual developed CHD during the course of a prospective study ( $69 \pm 6\%$  ;  $N = 257$ ) and (d) selection because one worked in a company participating in a study ( $71 \pm 7\%$  ;  $N = 113$ ).

The prevalence of TAB in healthy population comparison groups is consistent across several different methods of subject selection (see Table 6). These studies selected subjects from towns, factories, monasteries and hospital wards that included patients without CHD. The findings suggest that the method used to select subjects does not greatly influence the prevalence of TAB in research on TAB and CHD.

In contrast, there was variation in the prevalence of TAB across three different types of high risk comparison groups: (a) studies that selected subjects with the traditional risk factors ( $74\% \pm 1\%$  ;  $N = 3,035$ ;  $IVI = 0$ ), (b) studies that reported the results of the two earliest angiography studies ( $44\% \pm$  ;  $N = 107$  ;  $IVI = 0$ ) and (c) studies that reported the results of other high risk studies ( $67\% \pm 4\%$  ;  $N = 412$  ;  $IVI = 0$ ). The two earliest angiography studies used subject selection methods that are similar to more recent studies. Thus, subject selection methods cannot account for the differences between these two studies and later angiography studies. However, there appears to be a slight increase-- $74\%$  versus  $67\%$ --in the percentage of Type A's found in studies that select subjects on the basis of their risk factor status.

The WMR's varied only with whether the sample was healthy population (prospective healthy population = .33; case control = .35; cross-sectional population = .31) or

high risk (recurrent CHD = .12; angiography = .13). The IVI's for high risk and healthy population studies were all low (see Table 4). Thus, different methods of subject selection other than the high risk versus healthy population distinction do not appear to influence the prevalence of TAB or the magnitude of the  $r$  between TAB and CHD<sup>6</sup>. Only the two studies that selected subjects on the basis of their risk factor status appear to have significantly more Type A's in their high risk comparison groups than have been found in other high risk comparison groups.

Evidence of a trend towards more DBS bias in angiography studies. Miller et al. (1988) suggested that screening procedures for diagnostic angiography have improved in recent years so that fewer completely healthy subjects have been selected for study. Thus, there has been an increase in the extent of CAD in the diseased comparison groups in angiography studies in recent years. Miller et al. proposed that this restriction in range of CAD severity has attenuated  $r$ 's between TAB and CAD in recent years.

In fact, the percentage of Type A's in the diseased comparison group in angiography studies has increased. The earliest two angiography studies had percentages of Type A's (45% and 44%; see Table 7) in their diseased comparison groups that are similar to the percentages of Type A's that have been found in healthy populations (see Table 5). More recent angiography studies have reported higher percentages



of Type A's in their diseased comparison groups (see Table 7).

The increase in the percentage of Type A's in the diseased comparison groups has apparently produced the null findings in angiography studies. Only three studies in Table 7 reported positive findings. The two angiography studies published in the United States before 1979 reported positive results. Only one other study (Williams et al., 1980;  $N=424$ ) achieved a statistically significant result by using a larger sample size than other angiography studies.

In further support of the hypothesis that screening procedures have improved, the angiography study  $r$ 's are inversely correlated with year of publication within each type of CAD scoring method (see Table 9). The IVI for angiography studies that assessed TAB by the SI is large (.168) because of a trend towards smaller  $r$ 's in recent years.

Finally, one would expect the percentage of angiography patients with diagnosed CAD would increase if diagnostic screening procedures have improved. In fact, the percentage of individuals diagnosed as having CAD in the sample is nearly perfectly correlated with the year of publication (see Table 9). In fact, the Spearman rank  $r$ 's between the percentage of individuals with CAD and year of publication is 1.0 for the studies using the TOTCI method and the >75% occlusion scoring methods.

Only two findings were inconsistent. First, Krantz found slightly fewer patients with CAD and a slightly higher correlation between the SI and CAD than Dimsdale did a year earlier (see Table 9). Both studies were associated with small sample sizes. Therefore, the discrepancy may simply be the result of sampling error. Second, Langeluddecke found a lower percentage of subjects with CAD. The lower percentage of patients with CHD found in this study may have been the result of a lower prevalence of CAD in Australia.

There appears to be no relation between sex and the magnitude of the  $r$  between TAB and CAD (see Table 8). However, an insufficient number of studies other than angiography studies have included women. Therefore, the current research could not determine whether the magnitude of the relationship between TAB and CHD is influenced by subject sex for studies that did not use angiography as a disease criteria.

Table 9

Year of Publication, % Ill and r in Angiography Studies that Assessed TAB by the SI

Author	Year	% ill	r	% female
<b>TOTCI Scoring Method</b>				
Blumenthal	1978	45	.57	44
Krantz	1981	59	.14	8
Dembrowski	1985	69	.09	25
Blumenthal	1985	73	-.09	31
<b>&gt;50% Occlusion Scoring Method</b>				
Frank	1978	80	.55	16
Dimsdale	1979	84	.03	0
Krantz	1981	78	.07	0
Scherwitz	1983	>85	.03	0
Langeluddecke	1988	78	.08	21

Table 9 continued

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Author	Year	% ill	r	% female
<b>&gt;75% Occlusion Scoring Method</b>				
Blumenthal	1975	51	.67	44
Williams	1980	70	.25	28
Arrowood	1982			
Siegman	1987	78	-.01	26
Blumenthal	1987	82	-.29	24

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Notes. Author = the first author of the article that reported study findings; Year = the year of publication of the study; % ill = the number of patients that were diagnosed as having significant CAD; % females = the percentage of women in the sample;. The  $r$ 's reported in this table vary slightly from Miller et al.'s (1988) report that reported adjusted  $r$ 's.

## DISCUSSION OF STUDY FINDINGS

Research on TAB and CHD has been fraught with many confusing and contradictory results. The purpose of the current quantitative review was to identify the study characteristics that have contributed to the trend towards null findings. Three study characteristics--high risk study design, use of self-report measures and use of fatal MI as a disease criterion--were found to be associated with null findings. The current research documented that the increased use of these types of studies in recent years has contributed to the trend towards null findings.

In addition, the current review found several results that converge to suggest that DBS bias has produced the null findings in high risk studies. First, the percentage of Type A's was high in both the diseased comparison and diseased groups. Second, the prevalence of TAB in patients with CHD was significantly higher than found in healthy populations. Finally, range restriction formulas accurately estimated the actual correlations found in recurrent CHD studies.

Other subject selection biases do not appear to offer a plausible alternative explanation for the higher percentages of Type A's found among subjects with CHD. One would suspect that the percentage of Type A's found in the study sample would vary with the way subjects were selected into

the study if other subject selection biases influence this area of research. However, the correlation between TAB and CHD was consistent across studies that selected subjects using a variety of methods. Therefore, the results of the current research suggest that the magnitude of the correlation between TAB and CHD is not greatly influenced by the methods used to recruit subjects with the exception of the high risk versus healthy comparison group distinction.

### Reasons for the Trend Towards Null Findings

Increased use of recurrent CHD studies. Recurrent CHD studies have only recently become popular; in the early 1970's, researchers reasoned that recurrent CHD studies were more cost effective than healthy population prospective studies. They arrived at this conclusion because they believed that recurrent CHD studies with higher base rates of disease permitted smaller sample sizes (e.g., Sondik, Brown & Silvers, 1974; The Multiple Risk Factor Intervention Trial; MRFIT group, 1977) than healthy population studies. These researchers reasoned that smaller sample sizes could be used in recurrent CHD studies because higher base rates increase the statistical power of a study. Unfortunately, these researchers failed to recognize that recurrent CHD studies increase the base rate at the cost of greater range restriction. The results of the current research strongly suggest that increasing the base rate by using high risk

subjects is by no means a cost-effective or methodologically sound strategy for research on CHD.

Increase in extent of CAD in angiography patients. A number of recent angiography studies have failed to find a relationship between TAB and CAD. Miller et al. (1988) suggested one possible explanation. As expertise has increased in screening procedures for selecting patients for diagnostic angiography, the likelihood has decreased for a healthy person to be scheduled for an angiography. Therefore, recent angiography studies have not selected healthy subjects for study and so DBS bias has attenuated the correlation between TAB and CHD in recent years. In support of this hypothesis, the correlation between CAD and the traditional risk factors (Fried & Pearson, 1987), and CAD and TAB (Miller et al., 1988 and the current review) has decreased in recent years. In addition, the percentage of Type A's in recent studies has been similar to that found in patients with CHD.

Increased use of self-report measures. Since the National Heart, Blood and Lung Institute (1978) declared that TAB was a risk factor for CHD, many CHD researchers began to use indicators of TAB in their studies. Perhaps in an effort to reduce costs and save time, self-report measures have been favored over the SI. Unfortunately, self-report measures of TAB have a low correlation with CHD. Thus, many studies have found null results and the trend

towards using self-report measures has contributed to the trend towards null findings.

The use of fatal MI as a disease criterion. In recent years, the use of fatal MI as a disease criterion has always led to null results. There are a number of potential explanations for these findings. For example, some researchers (e.g., Correspondence. 1988) have criticized the reliability of death certificate information used as an indicator of fatal MI in many studies. Thus, the reliability of the disease criterion is one potential explanation for the null findings.

Two other possible explanations are worth mentioning. Perhaps TAB may be less predictive of fatal MI because fatal MI occurs most frequently in older subjects whose TAB status has been altered by lifestyle changes accompanying retirement or a prior history of MI (age bias). Second, there are two types of fatal MI's: sudden death and nonsudden death. Perhaps TAB is only predictive of one of these types of fatal MI (Brackett & Powell, 1988).

The MRFIT Study. The results of the MRFIT (Shekelle, Hulley et al., 1985) are important to consider because the MRFIT has been claimed by many to be the largest and most sophisticated study to examine the relationship between TAB and CHD in recent years. MRFIT selected subjects on the basis of their traditional risk factor status. The percentage of Type A's among individuals who remained



healthy throughout the MRFIT was higher than any of the healthy population studies and very similar to that found in patients with a prior history of CHD.

One possible explanation for the higher percentage of Type A's in the MRFIT study is that TAB is positively correlated with the traditional risk factors that were used to select subjects for the MRFIT. Although previous research suggests there is no simple linear relationship between TAB and the traditional risk factors (see Booth-Kewley & Friedman, 1987), a nonlinear or multivariate relationship may exist.

The findings of the Belgian Heart Disease Prevention Project (Kittel, 1986) are also supportive of this hypothesis. This study was a successful intervention designed to prevent CHD by reducing individuals' levels of risk on the traditional risk factors. This study found a strong relationship between TAB and CHD in the control group and no relationship in the treatment group after treatment. Thus, the intervention appears to have altered the relationship between TAB and CHD in some unknown way. In addition, only one other study that selected subjects on the basis of their risk factor status (Belmaker, Pollin, Jenkins & Brensike, 1976) found significantly more Type A's in the diseased comparison group.

## Suggestions for Future Research

Healthy population studies are required to obtain accurate estimates of the percentage of Type A's found in healthy samples stratified by the other risk factors for CHD. Although healthy population estimates are available for the SI, only one study (Moss et al., 1986) has used representative sampling techniques. Such studies are required to assess the magnitude of the relation between TAB and CHD, and TAB and the traditional risk factors.

Future healthy population prospective studies that assess TAB by the SI would be useful for validating the results of the WCGS. At present, only three prospective studies have assessed TAB by the SI.

Future research to control for and confirm that DBS bias is a problem in high risk studies requires researchers to use continuous indicators of disease severity and TAB (Knaus et al., 1984; Miller et al., 1988). By using continuous indicators, researchers can demonstrate how the relation between TAB and CHD is influenced by limiting the range of CHD to only individuals with severe disease (e.g., Fried & Pearson, 1987).

There are other advantages to using continuous measures. Categorization reduces associations between variables. Thus, the association between TAB and CHD is underestimated when dichotomous variables are used. The artifactual categorization of TAB and CHD is also a problem

because optimal categorization can vary from study to study (Rorer et al., 1966). Therefore, categorization leads to arbitrary changes in the magnitude of the association between TAB and CHD across different studies. The development and use of continuous measures of TAB (e.g., Friedman et al., 1986; Knaus et al., 1984) is an important area for future research.

Another measurement issue is the development of self-report measures of TAB. The current research suggests that self-report measures of TAB are not highly correlated with CHD. The development of more valid self-report measures of TAB to is another area for future research.

The presence of age and sex bias could not be adequately tested in the current research because most previous research has been conducted on middle aged men. To understand how the relationship between TAB and CHD is influenced by age and sex requires studies that examine women and individuals at different ages.

Finally, future research is required to understand why there appears to be a relationship between TAB and CHD. One possibly suggested by the current research is that TAB may be more closely associated with the traditional risk factors than has been suspected. Another question is why TAB does not appear to be predictive of fatal MI.

## POTENTIAL APPLICATIONS OF THE DT MODEL TO FUTURE RESEARCH

The DT model can be used to define other biases that may influence the relationship between TAB and CHD. The following section of this dissertation describes how the DT model can be used to define another type of spectrum bias referred to as mortality bias. In addition, this section discusses suggestions for how the DT model can be applied to future research on TAB and CHD.

### Mortality Bias in Cross-Sectional High Risk Studies

Mortality bias reduces the degree of statistical association between TAB and CHD in a similar manner to DBS bias. For DBS bias, healthy observations are excluded from the study sample and this reduces the specificity. Mortality bias occurs when the sensitivity is reduced by patient mortality (Miller et al., 1988). Similar to the reduction in specificity produced by DBS bias, the decrease in sensitivity produced by mortality bias attenuates risk ratios.

Figure 1(c) can be used to illustrate how mortality bias reduces the sensitivity. The line labeled  $c'$  in Figure 1(c) is an exclusion point and indicates where patients' CHD is sufficiently severe that a fatal MI has occurred. In Figure 1(c), more Type A's are located in the section of the distribution where fatal MI's occur. The exclusion of subjects with fatal MI decreases the

sensitivity because more Type A's incur a fatal MI than Type B's in the extreme diseased end of the continuum (see Figure 1(c)). This loss of Type A's to mortality decreases the percentage of Type A's in the diseased group in the study sample. The sensitivity decreases as the number of individuals who have incurred a fatal MI before the beginning of the study increases.

Typically, mortality bias is not as great a problem as DBS bias because fewer subjects incur fatal disease than remain healthy. For example, 96% of the subjects did not incur an initial MI and so were excluded from the recurrent CHD study in the first 8.5 years of the WCGS study. In contrast, the percentage of fatal MI's in the WCGS that would not be included in future follow-up studies was only 1%. That is, only 1% of the sample was excluded that could produce mortality bias. The percentage of fatal MI's in the 22 year follow-up period was not substantially greater--only 7% (Ragland & Brand, 1988b). Recurrent CHD studies exclude all subjects who have not incurred an MI. Therefore, the spectrum of disease in these studies is limited to only the most severely diseased subjects. In comparison to mortality bias, a substantially higher percentage of subjects are excluded from studies that produce DBS bias. Therefore, mortality bias is typically not as great a problem as DBS bias. Yet, many researchers have argued that mortality bias may have influenced their results while the possibility of

DBS bias has been ignored (Feinstein, 1986; Ransohoff & Feinstein, 1978).

### Implications of the DT Model for Future Research

#### Research to determine what biases are important.

Methodological concerns have been dominated by a concern with selection biases (Sackett, 1979). For example, researchers have consistently suggested that contradictory findings in research on TAB and CHD have been produced by problems associated with selection biases (Matthews, 1988). One purpose of the current paper is to suggest that spectrum biases also deserve consideration.

Quantitative reviews (e.g., Miller et al., 1988) can be used to determine the impact of various biases within a research domain (Einarson, Leeder, & Koren, 1988). These reviews can be used to determine which biases have a great influence on the magnitude of the correlation between TAB and CHD and which biases may not be important.

Interpretation of results. Quantitative reviews can be useful for determining the relative importance of risk factors. These are many well known reasons why a researcher may not find an association between a risk factor and a disease (e.g., inadequate statistical power, unreliability in measurement). Spectrum and selection biases are potential explanations for null findings that should be carefully considered before researchers are certain there is

no substantial association between a risk factor and a disease.

Detecting mortality bias. Researchers have not tested the extent to which mortality bias may have influenced the magnitude of the relationship between TAB and CHD. One could determine the extent to which mortality bias is a problem by testing whether the sensitivity between TAB and CHD decreases in populations in which a higher percentage of individuals have incurred a fatal MI. That is, researchers should compare the sensitivities across different subgroups of individuals or individuals in different studies with varying mortality rates (Ransohoff & Feinstein, 1978). One would have evidence of mortality bias if the sensitivities decrease in samples with higher mortality rates.

Investigating other types of spectrum biases. This dissertation only discussed two types of spectrum biases that may contribute to confusing and contradictory findings in epidemiological research--DBS bias and mortality bias. The DT model can be used to describe other types of spectrum biases. Similarly, there is no reason to consider only cases where the spectrum of disease is reduced. Spectrum biases may be concerned with any aspect of the pathology, clinical features or co-morbidity of the study sample (Ransohoff & Feinstein, 1978).

### Research to assess the prevalence of risk factors.

Establishing healthy population estimates of the specificity of a risk factor for different populations is an important aspect of research because healthy population estimates are useful for ascertaining the true magnitude of the association between a risk factor and a disease across the entire spectrum of the disease.

### Planning sample sizes for future research.

Researchers who aspire to conduct high risk studies should attempt to estimate a priori the extent to which DBS bias attenuates risk ratios in their high risk studies. Appendix B of this manuscript presents a derivation of a formula that can be used for calculating the expected reduction in the RR and/or the odds ratio for a high risk studies. Researchers should use this formula to calculate expected risk ratios and to estimate the sample size required to obtain a statistically significant result in high risk studies.

The expected power analyses may be quite discouraging to those interested in conducting high risk studies. In many cases, the estimated reduction in risk can be substantial. For example, 2.3% of all subjects in the WCGS at the 4.5 year follow-up were found to have a symptomatic MI, and the RR reported in the healthy population prospective study was 2.5 (see Table 2). Among those subjects who incurred an initial MI, 16% incurred a recurrent CHD. Applying these estimates to the formulas



presented in Appendix B, the estimated RR is 1.2 for the WCGS recurrent CHD results. This result is quite similar to the actual results (RR = 1.1) of the recurrent CHD of the WCGS.

The difference between the healthy population findings and the high risk study findings is substantial; varying from 2.5 to 1.1. This difference is almost entirely predicted by a formula that estimates the degree to which DBS bias reduces statistical associations in high risk studies. The research reported in the results section of this dissertation found that similar DBS bias formulas for  $r$ 's predicted the small associations between TAB and CHD reported in other recurrent CHD studies. All of these findings suggest that DBS bias is a serious problem in research on TAB and CHD. DBS bias may also be a problem in other research domains.

One can estimate the sample sizes for the healthy population and high risk studies by calculating the minimum sample size required to obtain a chi-squared value that would be significant at some level of significance (e.g.,  $p < .05$ ). These power analyses are useful for understanding how much of an increase in sample size is required to obtain a statistically significant result in high risk studies. The estimated sample size required to obtain a statistically significant result from the recurrent CHD study using an estimated RR of 1.2 is 2,378. In comparison, the minimum

required sample size to obtain a statistically significant result in the healthy population study that reported a RR of 2.5 would be 1,092. These results suggest much larger sample sizes may be required for research conducted in recurrent CHD populations than for healthy population studies. These findings are consistent with the research presented in the results section of this dissertation. These results suggested that DBS attenuates correlations in recurrent CHD studies.

Another possibility before conducting new studies that researchers should consider is to estimate the influence of DBS bias in previous high risk studies. For example, biased specificities for TAB found in high risk studies could have been replaced with a healthy population estimate of the specificity and new adjusted odds ratios can be estimated (Miller et al., 1988). The healthy population estimate of the specificity can be obtained by a review of the literature of healthy population studies such as the one conducted in this dissertation. The difference between the actual and adjusted odds ratio can be used as an indicator of the extent to which DBS bias has attenuated associations between TAB and CHD in previous high risk studies<sup>7</sup>.

Using previous research from other sciences. Although the concept of spectrum bias has only recently been discussed in epidemiology (Feinstein, 1986; Miller et al., 1988; Philbrick, Horwitz, Feinstein, Langou & Chandler,

1982; Ransohoff & Feinstein, 1978), spectrum bias has a long history in other scientific endeavors. There are several relevant areas of research the interested reader may consult. In industrial psychology (Lord & Novick, 1968; Thorndike, 1949) and statistics (Little & Rubin, 1987; Rydberg, 1963), spectrum biases have been referred to as range restriction. In economics, the term sample selection bias has been used (Heckman, 1979).

## References

- Alexander, R. A., Carson, K. P., Alliger, G. M., & Barrett, G. V. (1985). Further considerations of the power to detect nonzero validity coefficients under range restriction. Journal of Applied Psychology, 70(3), 451-460.
- Appels, A., & Mulder, P. (1985). Type A behavior and myocardial infarction: A 9.5 year follow-up of a small cohort. International Journal of Cardiology, 8, 465-470.
- Belmaker, R. H., Pollin, W., Jenkins, C. D., & Brensike, J. (1976). Coronary prone behavior pattern in a sample of type II hypercholesteremic patients. Journal of psychosomatic research, 20, 591-594.
- Booth-Kewley, S., & Friedman, H. S. (1987). Psychological predictors of heart disease: A quantitative review. Psychological Bulletin, 101, 343-362.
- Brackett, C. D. & Powell, L. H. (1988). Psychosocial and physiological predictors of sudden cardiac death after healing of acute myocardial infarction. American Journal of Cardiology, 61, 979-983.
- Cohen, J., & Cohen, P. (1984). Applied multiple regression/ correlation for the behavioral sciences. (2nd Ed.), Hillsdale, NJ: Erlbaum.
- Correspondence. (1988). Correspondence. New England Journal of Medicine, 319(2), 114-118.

- Dimsdale, J. E. (1988). A perspective on Type A behavior and coronary disease. New England Journal of Medicine, 318, 110-112.
- Einarson, T. R., Leeder J. S., & Koren, G. (1988). A method for meta-analysis of epidemiological studies. Drug Intelligence and Clinical Pharmacy, 22, 813-824.
- Feinstein, A. R. (1986). On blind men, elephants, spectrums, and controversies: Lessons from rheumatic fever revisited. Journal of chronic diseases, 39(5), 337-342.
- Fried, L. P., & Pearson, T. A. (1987). The association of risk factors with arteriographically defined artery disease: What is the appropriate control group. American Journal of Epidemiology, 125, 844-853.
- Friedman, M., & Rosenman, R. H. (1959). Association of specific overt behavior pattern with blood and cardiovascular findings. Journal of the American Medical Association, 96, 1286-1296.
- Friedman, M., Thoresen, C. E., Gill, J. J., Ulmer, D., Powell, L. H., Price, V. A., Brown, B., Thompson, L., Rabin, D. D., Breall, W. S., Bourg, E., Levy, R., Dixon, T. (1986). Alteration of type A behavior and its effect on cardiac recurrences in post myocardial infarction patients: Summary results of the recurrent coronary prevention project. American Heart Journal, 112(4), 653-665.

- Glass, G. V., McGraw B., & Smith, M. L. (1981). Meta-analysis in social research. Beverly Hills, CA: Sage Publications.
- Heckman, J. J. (1979). Sample selection bias as a specification error. Econometrica, 47, 153-161.
- Hedges, L. V., & Olkin, I. (1985). Statistical methods for meta-analysis. New York: Academic Press.
- Hunter, J. E., Schmidt, F. L., & Jackson, G. B. (1982). Meta-analysis: Cumulating research findings across studies. Beverly Hill: CA: Sage.
- Jenkins, C. D., Zyzanski, S. J., & Rosenman, R. H. (1971). Progress toward validation of a computer-scored test for the type A coronary-prone behavior pattern. Psychosomatic Medicine, 33(3), 193-202.
- Keefe, F. J., Castell, P. J., & Blumenthal, J. A. (1986). Angina pectoris in type A and type B cardiac patients. Pain, 27, 211-218.
- Kemsey, E. R., Dunlap, W. P., & Griffeth, R. W. (1988). Correction for variance restriction in point-biserial correlations. Journal of Applied Psychology, 73(4), 688-91.
- Kittel, F. (1986). Type A behavior and other psychological factors in relation to coronary heart disease. In T. H. Schmidt, T. M. Dembrowski & G. Blumchen. Biological and psychological factors in cardiovascular disease. Berlin Heidelberg: Springer-Verlag.

- Knaus, W. A., Wagner, D. P., Draper, E. A. (1984). The value of measuring severity of disease in clinical research on acutely ill patients. Journal of Chronic Diseases, 37(6), 455-463.
- Koskenvuo, M., Kaprio, Langinvainio, H., & Romo, M. (1983). Mortality in relation to coronary-prone behavior: a six year follow-up of the Bortner scale in middle-aged Finnish men. Activa Nervosa, Supplement (Praha), 25(2), 107-109.
- Little, R. J. A., & Rubin, D. B. (1987). Statistical analysis with missing data. New York: Wiley.
- Lord, F. M., & Novick, M. R. (1968). Statistical theories of mental test scores. Reading MA: Addison-Wesley.
- Matthews, K. A. (1988). Coronary heart disease and Type A behaviors: Update on and alternative to the Booth-Kewley and Friedman (1987) quantitative review. Psychological Bulletin, 104(3), 373-380.
- Miller, T. Q., Turner, C. W., Tindale, R. S., & Posavac, E. J. (1988). Disease based spectrum bias in referred samples and the relationship between Type A behavior and arteriosclerosis. Journal of Clinical Epidemiology, 41(12), 1139-1149.
- Moss, G. E., Dielman, T. E., Campanelli, P. C., Leech, S. L., Harlan, W. R., van Harrison, R., Horvath, W. J. (1986). Demographic correlates of SI assessments of type A behavior. Psychosomatic Medicine, 48, 564-574.

- Philbrick, J. T., Horiwitz, R. I., Feinstein, A. R., Langou, R. A., Chandler, J. P. (1982). The limited spectrum of patients studied in exercise test research: Analyzing the tip of the iceberg. Journal of the American Medical Association, 248(19), 2467-2470.
- Ragland, D. R., & Brand, R. J. (1988a). Type A behavior and mortality from coronary heart disease. New England Journal of Medicine, 318, 65-69.
- Ragland, D. R., & Brand, R. J. (1988b). Coronary heart disease mortality in the Western Collaborative Groups Study: follow-up experience of 22 years. American Journal of Epidemiology, 127(3), 462-475.
- Ransohoff, D. F., & Feinstein, A. R. (1978). Problems of spectrum and bias in evaluating the efficacy of diagnostic tests. New England Journal of Medicine, 299(17), 926-930.
- Rorer, L. G., Hoffman, P. J., & Hsieh, K. C. (1966). Tables of optimum cutting scores to discriminate groups of unequal size and variance. Journal of Applied Psychology, 50 153-164.
- Rosenman, R. H. (1978). The interview method of assessment of the coronary-prone behavior pattern. In T. M. Dembrowski, S. M. Weiss, J. L. Shields, S. G. Haynes & M. Feinleib (eds.) Coronary-prone behavior. New York: Springer-Verlag.



- Rosenman, R. H. (1986). Current and past history of type A behavior pattern. In T. H. Schmidt, T. M. Dembrowski & G. Blumchen. Biological and psychological factors in cardiovascular disease. Berlin Heidelberg: Springer-Verlag.
- Rosenman, R. H., Brand, R., Jenkins, D., Friedman, M., Straus, Wurm, M. (1975). Coronary heart disease in the Western Collaborative Group Study: Final follow-up of 8.5 years. Journal of the American Medical Association, 233(8), 872-877.
- Rosenman, R. H., & Friedman, M. (1961). Association of specific behavior pattern in women with blood and cardiovascular findings. Circulation, 24, 1173-1184.
- Rydberg, S. (1963). Bias in prediction: On correction methods. Stockholm studies in educational psychology, 7, Stockholm: Almqvist and Wiksells.
- Sackett, D. L. (1979). Bias in analytic research. Journal of chronic diseases, 32, 51-63.
- Schwertner, H. A., Troxler, R. G., Uhl, G. S., Jackson, W. G., & (1984). Relationship between cortisol and cholesterol in men with coronary artery disease and type A behavior. Arteriosclerosis, 4(1), 59-64.
- Shekelle, R. B., Hulley, S. B., Neaton, J. D., Billings, J. H., Borhani, N. D., Gerace, T. A., Jacobs, D. R., Lasser, N. L., Mittlemark, M. B., & Stambler, J. (1985). The MRFIT behavior pattern study II. Type A

- behavior and incidence of coronary heart disease,  
American Journal of Epidemiology, 122(4), 559-570.
- Sondik, E. J., Brown Jr., B Wm., Silvers, A. (1974). High risk subjects and the cost of large field trials.  
Journal of Chronic Diseases, 27, 177-187.
- Staff. (1987, July). Is Type A Passe? University of California, Berkeley Wellness Letter, p. 1.
- The MRFIT group. (1977). Statistical design considerations in the NHLBI multiple factor intervention trial (MRFIT). Journal of Chronic Diseases, 30, 261-275.
- The Review Panel on coronary-prone behavior and coronary heart disease: Coronary-prone behavior and coronary heart disease: A critical review. (1978).  
Circulation, 65, 1199-1215.
- Thorndike, R. L. (1949). Personnel selection. New York: Wiley.
- Wielgosz, A. T., Wielgosz, M., Biro, E., Nicholls, MacWilliam, L., Haney, T. (1988). Risk factors for myocardial infarction: The importance of relaxation.  
British Journal of Medical Psychology, 61, 209-217.
- Williams, Jr., R. B., Haney, T. L., Lee, K. L., Kong, Y., Blumenthal, J. A., Whalen, R. E. (1988). Type A behavior, hostility, and coronary atherosclerosis.  
Psychosomatic Medicine, 42, 539-549.
- Williams, Jr., R. B., Barefoot, J. C., Haney, T. L., Harrell, Jr., F. E., Blumenthal, J. A., Pryor, D. B.,

Peterson, B. (1988). Type A behavior and angiographically documented coronary atherosclerosis in a sample of 2,289 patients. Psychosomatic Medicine, 50, 139-152.

**APPENDIX A**

## LIST OF STUDIES NOT INCLUDED IN PREVIOUS REVIEWS

Note. This appendix lists studies used for reanalyses included in neither Booth-Kewley and Friedman's (1987), Miller et al.'s (1988) nor Matthews (1988) reviews. Some of these studies represent updates but most are additional studies that were required for the detailed reanalyses (e.g., estimates of percentages of Type A's) presented in the current paper.

Appels, A., & Mulder, P. (1985). Type A behavior and myocardial infarction. A 9.5-year follow-up of a small cohort. International Journal of Cardiology, 8, 465-470.

Blumenthal, J. A., Burg, M. M., Barefoot, J., Williams, R. B. Jr., Haney, T., & Zimet, G. (1987). Social support, type A behavior and coronary artery disease. Psychosomatic Medicine, 49, 331-340.

Bryne, D. G., Rosenman, R. H., Schiller, E. & Chesney, M. A. (1985). Consistency and variation among instruments purporting to measure the type A behavior pattern. Psychosomatic Medicine, 47(3), 242-261.

Caffrey, B. (1969). Behavior patterns and personality characteristics related to prevalence rates of coronary heart disease in American monks. Journal of Chronic Diseases, 22, 93-103.

- Chesney, M. A., Balck, G. W., Chadwick, & Rosenman, R. H. (1981). Psychological correlates of the type A behavior pattern. Journal of Behavioral Medicine, 4(2), 217-229.
- Dimsdale, J. E., Gilbert, J., Hutter, Jr., A. M., Hackett, T. P., & Block, P. C. (1981). Predicting cardiac morbidity based on risk factors and coronary angiographic findings. The American Journal of Cardiology, 47, 73-76.
- Eaker, E. D., & Castelli, W. P. (1988). Type A behavior and mortality from coronary disease in the Framingham study. New England Journal of Medicine, 319(22), 1480-1481.
- French-Belgian pooling project. (1984). Assessment of type A behavior by the bortner scale and ischaemic heart disease. European Heart Journal, 5, 440-446.
- Friedman, G. D., Ury, H. K., Klatsky, A. L., & Siegelau, A. B. (1974). A psychological questionnaire predictive of myocardial infarction: Results from the Kaiser-permanente epidemiologic study of myocardial infarction. Psychosomatic Medicine, 36(4), 327-343.
- Friedman, H. S., & Booth-Kewley, S. (1987). Personality, type A behavior, and coronary heart disease: The role of emotional expression. Journal of Personality and Social Psychology, 53(4), 783-792.

- Gallacher, J. E. J., Yarnell, J. W. G., Elwood, P. C., & Phillips, K. M. (1984). Type A behavior and heart disease prevalent in men in the Caerphilly study. British Medical Journal, 289, 732-733.
- Haynes, S. G., Feinleib, M., Levine, S., Scotch, N., & Kannel, W. B. (1978). The relationship of psychological factors to coronary heart disease in the Framingham study II. Prevalence of coronary heart disease. American Journal of Epidemiology, 107(5), 384-402.
- Jenkins, C. D., Zyzanski, S. J., & Rosenman, R. H. (1976). Risk of new myocardial infarction in middle-aged men with manifest coronary heart disease. Circulation, 53(2), 342
- Kittel, F., Kornitzer, M., Zyzanski, S. J., Jenkins, C. D., Rustin, R. M., & Degre, C. (1978). Two methods of assessing the type A coronary-prone behavior pattern in Belgium. Journal of Chronic Diseases, 31, 147-155.
- Koskenvuo, M., Kaprio, Langinvainio, H., & Romo, M. (1983). Mortality in relation to coronary-prone behavior: a six year follow-up of the Bortner scale in middle-aged Finnish men. Activa Nervosa, Supplement (Praha), 25(2), 107-109.

- Koskenvuo, M., Kaprio, Langinvainio, H., Romo, M., & Sarna, S. (1981). Psychosocial and environmental correlates of coronary-prone behavior in Finland. Journal of Chronic Diseases, 34, 331-340.
- Langeluddecke, P., Fulcher, G., Jones, M., & Tennant, C. (1988). Type A behavior and coronary atherosclerosis. Journal of Psychosomatic Research, 32(1), 77-84.
- Mann, A. H., & Brennan, A. J. (1987). Type A behavior score and the incidence of cardiovascular disease: A failure to replicate the claimed associations. Journal of Psychosomatic Research, 31(6), 685-692.
- Moss, G. E., Dielman, T. E., Campanelli, P. C., Leech, S. L., Harlan, W. R., van Harrison, R., & Horvath, W. J. (1986). Demographic correlates of SI assessments of type A behavior. Psychosomatic Medicine, 48, 564-574.
- Pearson, T. A., Achuff, S. C., Kwiterovich, P. O., et al. (1982). Type A personality and coronary atherosclerosis: an indirect association? Circulation, 66(Suppl II), 114.
- Ray, J. J. (1984). A-B personality, authoritarianism and coronary heart disease: A correction. British Journal of Medical Psychology, 57, 386.
- Ragland, D. R., & Brand, R. J. (1988b). Coronary heart disease mortality in the Western Collaborative Groups Study: follow-up experience of 22 years. American Journal of Epidemiology, 127(3), 462-475.



- Rechnitzer, P. A., Cunningham, D. A., Donner, A. P., Andrew, G. M., Buck, C. W., Jones, N. L., Kavanaugh, T., Oldridge, N. B., Parker, J. O., Shephard, R. J., & Sutton, J. R. (1983). Characteristics that predicted recurrence of infarction within 3 years in the Ontario exercise-heart collaborative study. Canadian Medical Association Journal, 128, 1287-1290.
- Reunanen, A. (1988). Type A behavior pattern and ischaemic heart disease. Annals of Clinical Research, 20, 137-142.
- Reunanen, A., Hanses, O., Maatela, J., Impivaara, O., & Aromaa, A. (1987). Type A behavior pattern in Finland. Annals of Clinical Research, 19, 144-199.
- Shoham-Yakubovich, I., Ragland, D., Brand, R. J., & Syme, S. L. (1988). Type A behavior pattern and health status after 22 years of follow-up in the Western Collaborative Group Study. American Journal of Epidemiology, 128(3), 579-588.
- Sieglman, A. W., Feldstein, S., Tomasso, C. T., Ringel, N., & Lating, J. (1987). Expressive vocal behavior and the severity of coronary artery disease. Psychosomatic Medicine, 49, 545-561.
- Wielgosz, A. T., Wielgosz, M., Biro, E., Nicholls, MacWilliam, L., & Haney, T. (1988). Risk factors for myocardial infarction: The importance of relaxation. British Journal of Medical Psychology, 61, 209-217.

**APPENDIX B**

## ESTIMATING REDUCTION IN RISK

This appendix illustrates a technique for estimating the reduction in risk produced by DBS bias in a high risk study from the results of healthy population studies. All one requires are values for the statistical parameters that describe the DT model presented in Figure 1(b). These parameters are (a) the probability of possessing the risk factor (e.g., being a Type A) in the study sample (b) the magnitude of the  $\underline{d}'$ , (c) the location of  $\underline{c}$  and  $\underline{c}'$ , and (d) the shape of the distributions of the risk factors. For the equations presented in the current paper, the shape of the frequency distributions for different values of the risk factor are assumed to be normal and symmetric with respect to disease status.

To illustrate the estimation procedure, an example from research on TAB and CHD is used. The RR associated with recurrent CHD is obtained from an estimate of the probability of incurring a MI in a healthy individual ( $p(d)$ ). The probability of recurrent CHD in the future for a healthy individual (the probability of recurrent disease in a healthy individual =  $p(rd_h)$ ) is described by the following equation

$$p(rd_h) = p(d)p(rd) \quad (1)$$

where  $p(rd)$  equals the probability of recurrent CHD in a sample of individuals with a history of MI. In some

cases, an estimate of  $p(\text{rdh})$  may be obtained directly from the results of a healthy population prospective study. In other cases, estimates of  $p(\text{rd})$  and  $(d)$  can be obtained from prior research and these values can be used to calculate  $p(\text{rdh})$ .

The next step is to obtain  $z$  scores that satisfy the following equations.

$$z_{A,h} - z_{B,h} = \underline{d}' \quad (2)$$

$$w_1 p(\text{rd}_{A,h}) + w_2 p(\text{rd}_{B,h}) = p(\text{rdh}) \quad (3)$$

where "zh" equals the z score associated with  $p(\text{rdh})$ , the A and B subscripts indicate Type A and Type B behavior and "w" indicates the weights associated with the probability of being Type A or Type B in the healthy population study sample. The 'A' and 'B' subscripts for the A and B distributions indicate which distribution--Type A or B--the z score or probability is estimated from. For example,  $p(\text{rd}_{A,h})$  indicates the probability of recurrent CHD in healthy Type A's. Equation (2) is the standard equation for the effect size  $\underline{d}'$  (Glass, McGraw, & Smith, 1981). Equation (3) indicates that the sum of the number of Type A's and B's who develop recurrent CHD equals  $p(\text{rdh})$ .

The simplest way to calculate the zh scores is to use a standard  $z$  score conversion table. One simply finds the pair of zh scores with probability values that sum to  $p(\text{rdh})$  and that satisfy equation (2). The zh scores can be used to

estimate the RR. The formula for the RR is

$$p(z_Ah)p(z_B)/p(z_A)p(z_Bh) \quad (4)$$

where each  $p(z_)$  equals the probability value associated with a  $z$  score, and a  $z$  without a "h" sign equals the  $z$  score associated with the probability that a healthy individual will incur a MI. The formula for the odds ratio is

$$(p(z_A)-p(z_Ah))(p(rd_Bh)/(p(z_B)-p(z_Bh))(z_Ah)) \quad (5)$$

Furthermore,

$$p(z_Ah)/(p(z_Bh) + p(z_Ah)) \quad (6)$$

equals the sensitivity and

$$(p(z_B)-p(z_Bh))/(p(z_B)) \quad (7)$$

equals the specificity.

The following is an example of how one can use the preceding equations to estimate the RR between TAB and CHD in the recurrent CHD of the WCGS from the healthy population results. In order to estimate the RR, one first obtains an estimate of  $p(rd)$ . For example, the WCGS reported that approximately 2.3% of the subjects developed symptomatic MI's over the 4.5 year follow-up period. Thus,  $p(rd)$  equals 2.3%.

Second, one obtains the  $z$  scores associated with the percentage of Type A's and B's who develop CHD from the healthy population results. The  $z$  scores associated with the percentages of Type A's and B's that incur a MI can be obtained from a standard score conversion table. These  $z$

scores would both be located at  $\underline{c}$  in Figure 1(b). One  $\underline{z}$  score is associated with the Type B distribution and one  $\underline{z}$  score is associated with the Type A distribution.

Subtracting the Type B  $\underline{z}$  score from the Type A  $\underline{z}$  score provides an estimate of  $\underline{d}'$ . For example, the  $\underline{z}$  score associated with the percentage of Type A's that incurred a MI in the WCGS is 1.85. The  $\underline{z}$  score is 2.22 for the percentage of Type B's. The difference between the Type A and B  $\underline{z}$  scores is 0.37. Therefore, the  $\underline{d}'$  for the WCGS is 0.37.

The next step is to obtain  $\underline{z}$  scores for  $\underline{c}'$ . For recurrent CHD studies,  $\underline{c}'$  is the point at which individuals develop recurrent CHD. To obtain these estimates, one only requires an estimate of  $p(\text{rd}')$ . In the case of the WCGS, the recurrent CHD rate was 16%. Using equations 2 and 3, the estimated result for the recurrent CHD study is 1.2.

Table 2 presents the results for the actual and estimated recurrent CHD study of the WCGS. In no cases are the estimated results significantly different from actual results. The only discrepancy between the estimated and actual results occurs in the sensitivity (this also accounts for the lower  $\underline{d}'$ ). For a discussion of possible reasons why the sensitivity in high risk studies is lower than the estimates, see the section of the current paper entitled age bias.

The range restriction formulas derived in the current paper are based on the assumption that the Type A and B distributions are symmetric with respect to CHD status and that the relationship between TAB and CHD is linear. In many cases, this may be an unreasonable assumption. The need for range restriction formulas based on less restrictive distributional assumptions is area of future research. Research using quasi-continuous measures of TAB and CHD would be useful to determine that the relationship between TAB and CHD is linear and bivariate normal.

## Endnotes

1. Most research has treated CHD as a dichotomous variable (e.g., MI versus no MI, angina versus no angina). The current research defines CHD as a continuum in which MI or angina are discrete points along a spectrum of disease severity. Many researchers (e.g., Knaus, Wagner & Draper, 1984; Miller, Turner, Tindale & Posavac, 1988) have noted that defining CHD as a continuous variable is a useful way of conceptualizing CHD. When CHD is conceptualized as occurring along a continuum, one can describe high risk studies as a sample in which only individuals from the diseased range of CHD are selected for study.

2. Previous research suggests the relationship between TAB and CHD is linear (Booth-Kewley and Friedman, 1987) and that the distributions of Type A's and B's are normally distributed (Kittel et al., 1978). However, the distribution of TAB with respect to CHD status remains unknown because researchers have relied on statistical analyses that treated CHD and TAB as dichotomous variables.

3. Hedges and Olkin (1985) have recommended correcting  $r$ 's for unreliability to obtain a more accurate estimate of the theoretical relation between the variables of interest. Previous research (Booth-Kewley & Friedman, 1987) has addressed this question. Correction for unreliability was



not undertaken in the current research because the focus was to identify study characteristics that are associated with positive and null findings.

A chi-squared test (see Hedges & Olkin, 1985) was used to determine if the amount of inter-study variance not attributable to sampling variability as indicated by the IVI was statistically significant. The chi-squared test was almost always significant. This finding is not surprising considering the large sizes examined in the current research. However, the magnitude of the IVI in most cases suggests that the actual percentage of variance attributable to inter-study variation is quite small. Therefore, the results of the chi-squared tests are not reported.

4. The range restriction formula that is presented in Alexander et al. (1985) is

$$E(r) = r_{xy} \left| \frac{\begin{array}{c} - \\ X \\ \hline Y \\ - \end{array}}{\begin{array}{c} - \\ \\ - \end{array}} \right|^{1/2}$$

Where  $E(r)$  equals the expected value of  $\underline{r}$ ,  $r_{xy}$  is the value or  $r$  observed in the healthy population study, and  $X$  and  $Y$  are equal to

$$X = 1 - (h^2/\phi^2) + (h/\phi)(Z_{xc})$$

$$Y = 1 - (r_{xy}^2)(h^2/\phi^2) + r_{xy}^2(h/\phi)(Z_{xc})$$

Where  $\phi$  is the selection ratio,  $h$  is the height of the normal curve on  $X$  corresponding to the observed  $\phi$ , and  $Z_{xc}$  is the standard score cutoff point on  $X$  for  $\phi$ .

5. The substantial inter-study variation (IVI = .132) in the case control studies that assessed TAB by the SI was produced entirely by one study (Wielgosz et al., 1988;  $r = -.04$ ). The WMR for case-control studies that assessed TAB by the SI excluding this study (WMR = .41,  $N = 505$ ; IVI = .009) is much higher and consistent with the results for case control studies using self-report measures of TAB (see Table 2).

The large WMR's associated with case control studies are inconsistent with the results of other more methodologically rigorous designs. This finding is consistent with other meta-analyses (Einarson, Leeder & Koren, 1988). Therefore, the results of case-control studies should probably be treated with more caution than other type of designs.

6. Another frequently mentioned explanation for the null findings in high risk studies has been that a person's TAB status may change after incurring CHD. However, this hypothesis appears to be incorrect because the prevalence of TAB in subjects with CHD is similar in both prospective and cross-sectional studies.

For the studies reported in the current review, subjects' TAB status was assessed soon after the coronary event. Therefore, TAB status may change after the first few months following a MI.

7. Unfortunately, replacing healthy population estimates of the sensitivity or specificity to correct for biased estimates may frequently be impractical. One reason is that healthy population estimates of individuals with similar characteristics to the diseased sample are often unavailable. Furthermore, all the characteristics that may be important are often unknown. Thus, the omitted variable problem may produce misleading results when adjusted risk ratios are used.

Another problem with replacing a study sample estimate with a healthy population estimate is that this reduces the study design to essentially a case-control design even if the original design was prospective.

APPROVAL SHEET

The dissertation submitted by Todd Q. Miller has been read and approved by the following committee:

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The final copies have been examined by the director of the dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the dissertation is now given final approval by the Committee with reference to content and form.

The dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

12/4/89  
Date

B. Scott Tindale  
Director's Signature