### MODIFICATION OF CORONARY HEART DISEASE RISK FACTOR LEVELS IN MEXICAN AMERICANS

A Thesis

Presented to

the Faculty of Department of Psychology

# University of Houston

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

Ву

David Russell Rubovits

October, 1988

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### Abstract

Mexican Americans are at greater risk for several coronary heart disease (CHD) risk factors, including hyperlipidemia, hyperglycemia, hypertension, and obesity than is the general population. Yet there is little research on the distribution of CHD risk factors and the effectiveness of modifying those risk factors in the Mexican American population. The present study represents an analysis of CHD risk factors in a Mexican American population participating in a longitudinal program of weight A total of 118 women were followed for a six loss. month period while they participated in the program as members of one of three intervention groups. Group 1 served as a comparison group receiving the weight loss program through the weight loss manual. Group 2, the individual group, received the same booklet and also attended classes for its instruction. Group 3, the family group, received the booklet and was also encouraged to include their spouses in class attendance. Group 3's classes included additional information on how to incorporate dietary and lifestyle changes into their families. The purpose of

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this study was to investigate the effectiveness of this program for modifying lipid, blood sugar, blood pressure, and body mass index (BMI) levels in this population. It was hypothesized that subjects in the family group intervention would be more successful than the individual intervention and comparison groups. Multivariate and univariate repeated measures analyses were conducted on the dependent measures. There was a significant Group X Time interaction, however BMI was the only dependent measure significantly modified. The family and individual group interventions had a significantly greater reduction in BMI than the comparison group, but did not significantly differ from each other. There was, however, a significant linear trend in BMI across all three groups. Exploratory analyses yielded a much greater proportion of family group subjects losing weight and BMI than the other two groups. Therefore, there is strong evidence that this intervention succeeded in reducing at least one CHD risk factor The success of the intervention relates well level. to a systems theory framework, and may generalize to other samples and populations.

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#### CHAPTER I

#### INTRODUCTION

Despite modern medicine's pharmaceuticals and technical expertise, coronary heart disease (CHD) continues to be the number one cause of death in most Northern European, North American and other industrialized societies (Castelli, 1984). Even though there has been a decrease in CHD incidence over the past decade in the United States, it still accounts for 37% of all deaths in the U.S. The Mexican American population in the U.S. is considered at increased risk for the development of CHD as a result of their increased levels of CHD risk factors (Stern, Gaskill, Allen, Garza, Gonzalez, & Waldrop, 1981; Stern, Haskell, Wood, Osann, King, & Farguhar, 1975; Stern, Pugh, Gaskill, & Hazuda, 1982). Unfortunately, few intervention studies have been designed to reduce this risk. This study was part of a larger program, that had as its primary objective, to measure weight loss in Mexican American families by modifying dietary and life-style behaviors. Three groups of subjects were constructed: 1) a comparison group; 2) a group in which the focus of the intervention was the target individual; and 3) a group in which the focus of the intervention was the target

individual and her family. The purpose of this study was to examine the effectiveness of the larger program for the reduction of CHD risk factor levels (a secondary objective of the program). This study also addresses the heuristic utility of a systems theoretic framework for such interventions.

The following sections discuss CHD and the relationships between its various risk factors and its prevention. That information is incorporated in a description of the Mexican American population at risk for CHD. Finally, the assimilation of the cultural and CHD risk factor information is applied to a systems theory framework and design of the study. Coronary Heart Disease

Castelli (1984) reported that by age 60, one in five males, and one in 17 females will have some form of CHD sufficiently advanced to cause health problems. Until recently, the study of coronary heart disease was traditionally limited to adult populations at high risk for the disease. However, recent research indicates that atherosclerosis begins in youth and even as early as childhood (Newman, Freedman, & Voors, 1986; McGill, 1980). As early as the 1930's

physicians discussed etiologies of atherosclerosis in "young" people (Gertler & White, 1954; Glendy, Levine, & White, 1937). However, it was difficult to justify studies investigating atherogenesis in children and adolescents without objective diagnoses of the disease in those populations. Still, it was suggested that "... the time has come to emphasize that coronary arteriosclerosis may occur, literally, from infancy onward and is not particularly uncommon after the age of 30" (Marvin, 1937, p. 1781). In fact, autopsies performed on American casualties from the Korean and Vietnam wars showed that coronary plaques do develop by late teenage/early adult years in American society as evidenced by the discovery of plaques in 77.3% of Korean servicemen (Mean CA = 22.1 years) and 45% of Vietnam servicemen (Mean CA = 22.1 years) (Enos, Holmes, & Beyer, 1953; McNamara, Malot, Stremple, & Cutting, 1971).

Recognizing CHD clinically involves diagnostic measures of the medical profession. However, epidemiological studies are able to predict potential victims of CHD, from risk factors, whether adults or children. These risk factors are developed using

multiple regression techniques on large samples of populations with and without clinical CHD. This permits the investigation of the development of CHD in normal populations as well as those at increased risk. Many such studies are widely cited in the popular literature: Framingham (Castelli, 1984); Seven Countries (Keys, 1980); MRFIT (Multiple Risk Factor Intervention Trial Group, 1982); the Stanford Five Cities Project (Farguhar et al., 1984); and Western Collaborative Group Study (Ragland & Brand, 1988). The Framingham study, for example, has been operating since 1949, following 5,209 men and women 30-62 years old, randomly selected from the 10,000 residents of Framingham, Massachusetts. The subjects are "measured" biennially on a long list of variables including physical examination to determine any evidence of CHD. Currently the CHD risk factors assembled from these studies include: total to high density lipoprotein cholesterol ratio, hypertension, smoking, excess weight, hyperglycemia, sedentary lifestyle, stress (both physical and psychological, including coronary prone behavior), gender, heredity,

and age. In the next section, I discuss such CHD risk factors.

#### Coronary Heart Disease Risk Factors

A curvilinear relationship between the concentration of plasma cholesterol and risk for CHD is due to the atherogenic effect of plasma levels of cholesterol (Grundy, 1986). Briefly, atherogenesis involves the formation and development of fatty streaks within the coronary arteries. Over time, these streaks become plagues that can narrow the lumen of these vessels. Sufficient closure of the arteries will lead to an infarct or heart attack. The greater the size and extent of the plaques, the higher the risk of infarct from this cause. "Hiah Cholesterol", then, not only represents values at the upper end of the distribution, but an exponential increase in CHD risk.

Defining hypercholesterolemia with a value of 240 mg/dl or greater will categorize 15-20% of American adults with that increased risk (Grundy, 1986). Hypercholesterolemia can be thought of as a disease itself, and thus may have several etiologies, including genetic and dietary influences.

Brown and Goldstein (1983) received a Nobel Prize for their work in defining the genetic roles of cholesterol and CHD. They found that genetic contributions to cholesterol levels are found predominantly in one of two inherited syndromes: 1) heterozygous familial hypercholesterolemia, affecting 1 in 500 people with twice the normal levels of cholesterol, and 2) homozygous familial hypercholesterolemia (much less common) with four times the normal levels of cholesterol. Although the most dramatic forms of high cholesterol are genetic in origin, this condition is responsible for less than 2% of all the individuals with cholesterol levels greater than 240 mg/dl. The high cholesterol levels in the remaining 98% are either a function of diet alone, or, most likely, a combination of dietary and genetic contributions (Grundy, 1986).

Dietary effects on cholesterol levels are caused primarily by dietary cholesterol and saturated fat (Grundy, 1986). While dietary cholesterol is found only in animal products including their greases and oils, saturated fat is found in both animal and vegetable foods. Polyunsaturated and monounsaturated

fats have a less distinct contribution to serum cholesterol levels.

The total cholesterol levels themselves can also be broken down into components. The high density (HDL) and low density (LDL) lipoprotein fractions are the ones associated most directly with CHD. The HDL fraction is generally considered the "good cholesterol" because it contains a higher proportion of protein to fat, while the LDL has a lower density of protein and thus is labeled the "bad cholesterol". Due to the lack of data showing a relationship between diet and HDL levels, LDL is the primary dietresponsive variable and pathogen (Blackburn, 1983). Whether diet has a long-term effect on cholesterol levels remains to be demonstrated, but if it does, then diet could indeed play a major role in its modification (Grundy, 1986).

Data from large cross cultural studies (such as the Seven Countries Study) suggest that different diets (between the various cultures) predict different cholesterol levels and a "... diet high in saturated fat and cholesterol would be a necessary and possibly sufficient factor for mass atherosclerosis"

(Blackburn, 1983, p. 2; Keys, 1980). Results such as these have provided the impetuous for nutrition modification on a large scale.

The American Heart Association, among others, has developed detailed dietary prescriptions for whole segments of our society. The justification of these diet (and subsequent serum cholesterol, glucose, and blood pressure levels) recommendations are a result of convergence between the epidemiological data and intervention data for lowering CHD risk and incidence (Blackburn, 1983). However, the recommendations are not necessarily the last word:

The relation between plasma cholesterol level and CHD is incontrovertible. Our understanding of the mechanisms for control of the plasma cholesterol concentration and the means to alter the levels of lipoproteins has advanced greatly. We now have strong evidence that lowering LDL concentrations will reduce the risk of CHD and we are on the verge of possessing the therapeutic modalities required to effectively reduce LDL levels. Although these advances are exciting and encouraging, the medical community is faced with

major decisions about how to apply them for the control of high plasma cholesterol levels in the American public (Grundy, 1986, p. 2854).

In addition to the curvilinear relationship between cholesterol and CHD risk, there is a similar relationship between blood pressure and subsequent development of CHD. Weight is also a strong predictor of CHD for both men and women (Castelli, 1984) and some studies are now designating it a direct, as opposed to an indirect risk factor (Foreyt, 1987; Hubert, Feinleib, McNamara, & Castelli, 1983). Castelli (1984) has also found a marked increase in CHD incidence with blood glucose levels above 119 mg/dl in males, and above 125 mg/dl in females.

The relationships between the individual risk factors and the incidence of CHD suggest that reducing the levels of one risk factor will in turn result in a corresponding reduction in the incidence of CHD. In general, this may be true. However, the major population studies reveal at least an additive, and probably a multiplicative interaction, of major CHD risk factors, cholesterol level, hypertension, and smoking (Castelli, 1984; Grundy, 1986). Therefore, although it is useful to conceptualize etiologies and incidence levels from individual risk factors, a true picture of CHD risk can not be assessed without consideration of all applicable risk factors and their interactions. In fact:

"Isolated management of one risk factor is poor medicine, and attention must be focused on all the major factors if the short- and long-term goals are to be kept firmly in mind, or if coronary heart disease is to be kept significantly reduced" (Castelli, 1984, p. 11).

Interventions must be carefully planned so as not to increase one risk factor while attempting to control another. (Evidence of this can be seen in the relationship between antihypertensive drugs possibly raising cholesterol levels)

### Interventions of CHD Prevention

The most positive evidence for reducing the incidence of CHD comes from intervention trials which have generally shown that lowering risk factors reduces the rate of CHD incidence (Castelli, 1984; Lipid Research Clinics Program, 1984a&b). While the intervention trials attempted to modify and measure several risk factors, the majority of their efforts were concentrated on the modification of serum cholesterol levels. There are three important developments that allowed the prevention of CHD by modifying high cholesterol levels: 1) Brown and Goldstein's Nobel Prize winning discovery of cell surface receptors for LDL, allowing greater understanding of how cholesterol levels can be controlled; 2) the development of new drugs that act as cholesterol inhibitors; and 3) the Lipid Research Clinics findings that lowering cholesterol levels lowers CHD incidence (Grundy, 1986).

The Lipid Research Clinics - Coronary Primary Prevention Trial (Lipid Research Clinics Program, 1984a&b) was a double blind, placebo controlled clinical trial that tested the efficacy of lowering cholesterol levels for the primary prevention of CHD. Twelve clinics recruited 3,806 middle-aged men with primary hypercholesterolemia (an excess of lipoproteins in the blood), but free of CHD. The men were randomized into two groups: 1) treatment group received cholestyramine (a drug which serves to bind cholesterol), and a moderate cholesterol lowering

diet; 2) the control group received a placebo drug and the same diet. The treatment group experienced significant reductions of 8.5% in total serum cholesterol levels and 12.6% in LDL levels over the control group. The treatment group had a significant reduction of 19% in risk as measured by the endpoint of definite CHD death or definite non-fatal myocardial infarction over the control group.

This type of intervention involves economic and labor intensive commitments. The high risk individuals that are served by such an approach no doubt are in need of it, but are a very small proportion of the population that does need to lower their risk of CHD. Even assuming a standard distribution of cholesterol levels (i.e., total CHD risk), a small percentage of a population is served with interventions for high-risk individuals. Because a high risk strategy approach of CHD risk reduction is inefficient for a population, a public health strategy based on public education and dietary modification might serve the general population better, and lower the total incidence of CHD (Grundy, 1986). A public health view of diet, atherosclerosis, and coronary disease is based on an extension of the host-environment concept to the idea that mass disease, such as atherosclerosis and hypertension, is very likely the action of powerful cultural factors on widespread human susceptibility (Blackburn, 1983, p. 2).

In fact, population diet and cholesterol levels are highly correlated; whereas individual diet and cholesterol relationships indicate an importance for genetic factors for cholesterol levels and dietary responses. Cultural factors may also play a role in a predisposition to obesity and diabetes (Stern, Pugh, Gaskill, & Hazuda, 1982). Without debating the "nature versus nurture" issue, it is plausible to argue that there are cultural influences on health behaviors such as diet as well as on biological boundaries of lipoprotein, blood glucose, and blood pressure levels.

The United States contains many different cultures, each expressing its own set of behavioral and disease characteristics. While research in the medical and psychological fields often attempts to generalize findings from white middle class males to the entire population, minority considerations should not be overlooked.

#### Mexican Americans and CHD

Mexican Americans are the second largest minority group in the U.S. (approximately 7 million people) (Stern, Gaskill, Allen, Garza, Gonzalez, & Waldrop, 1981; U.S. Department of Commerce, Bureau of the Census, 1987). The number of Mexican Americans in the United States increased 93% between 1970 and 1980, and almost one-fifth the population of Texas is of Mexican origin (U.S. Department of Commerce, Bureau of the Census, 1982). Even though the Mexican American population is large and continues to increase, there are relatively few scientific investigations of their health (Stern, Gaskill, Allen, Garza, Gonzalez, & Waldrop, 1981).

Studies designed to assess both the nature of CHD risk and behavioral dimensions of Mexican Americans include the San Antonio Heart Study, the Laredo, Texas project, and the Stanford Project. The San Antonio Heart Study is a comprehensive epidemiological investigation of lifestyles as they relate to obesity, diabetes, and CHD risk factors in Mexican Americans and Anglos in San Antonio, Texas (Stern, Pugh, Gaskill, & Hazuda, 1982). The Laredo project was designed to study the distribution of CHD risk factors in a low-income Mexican American population from Laredo, Texas (Stern, Gaskill, Allen, Garza, Gonzalez, & Waldrop, 1981). The Stanford project compared 119 Mexican American males, and 180 Mexican American females to a non-Mexican American, white, population in California of 743 males and 923 females (Stern, Haskell, Wood, Osann, King, & Farguhar, 1975).

Descriptive data on the risk of CHD in Mexican Americans is sparse but consistent. Hanis, Ferrell, and Schull (1985) report four to six times the prevalence of non-insulin dependent diabetes, obesity, and hypertension in Mexican Americans of South Texas than the rest of the U.S. This is an alarming increase in CHD risk for an ethnic group. The etiologies of the increase may be found in culture specific behaviors and attitudes. For instance, Mexican Americans have a lesser degree of "sugar avoidance" and researchers have reported that Mexican American women tend to favor an attitude of fatalism for obesity. In addition, both Mexican American men and women view Anglo-Americans as too concerned about weight loss (Stern, Pugh, Gaskill, & Hazuda, 1982). Obesity in Mexican Americans

Obesity occurs with greater frequency in Mexican Americans than in the general U.S. population, regardless of SES (Stern, Haskell, Wood, Osann, King, & Farquhar, 1975; Stern, Pugh, Gaskill, & Hazuda, 1982). The Laredo, Texas project reported age adjusted rates of 25.8% for male and 44.8% for female Mexican Americans more than 20% over ideal body weight. This compares to a rate of 15.6% of male and 29.0% of white female subjects in the HANES study of the U.S. population (Stern, Gaskill, Allen, Garza, Gonzalez, & Waldrop, 1981). Since obesity is often positively correlated with high blood pressure, one might expect a higher prevalence of hypertension in Mexican Americans than in the general U.S. population (Franco, Stern, Rosenthal, Haffner, Hazuda, & Comeaux, 1985).

### Hypertension in Mexican Americans

Although the San Antonio study revealed roughly the same prevalence of hypertension in Mexican Americans and other whites, the Mexican Americans lagged behind in being aware of having, being treated for, and being in good control of their hypertension. This supported the researchers' hypothesis of cultural barriers to information and health care (Franco, Stern, Rosenthal, Haffner, Hazuda, & Comeaux, 1985). Other studies including the Laredo study found the prevalence of hypertension in Mexican American males inversely related to SES. In addition, the blood pressure levels of Mexican American males were found to fall below the levels of blacks and above those of whites (Kraus, Borhani, & Frant, 1980; Stern, Gaskill, Allen, Garza, Gonzalez, & Waldrop, 1981).

A study in Starr County, Texas found a 133% higher rate of hypertension in Mexican Americans than in other white populations (Hanis, Ferrel, & Schull, 1985). However, a longitudinal study of blood pressures in Mexican American adolescents found no differences in levels between whites, blacks,or Mexican Americans under 20 years old (Baron, Freyer, & Fixler, 1986).

#### High Cholesterol in Mexican Americans

Serum cholesterol levels collected in women from Laredo, Texas were significantly higher than U.S. averages across all ages (Stern, Gaskill, Allen, Garza, Gonzalez, & Waldrop, 1981). Mexican American women's triglyceride levels were also significantly higher than those of other whites in the Stanford study (Means of 142 mg/dl & 116 mg/dl, respectively; Stern, Haskell, Wood, Osann, King, & Farguhar, 1975). Although there were no significant differences between Mexican Americans and other whites on serum cholesterol levels during the Stanford study, the more recent data of the Laredo project have shown a slight increase in those levels from 1975 to 1981 for Mexican Americans and a decrease for other whites. This suggests that changes occurred in the general U.S. population that did not occur in the Mexican American population. Those changes could be related to cultural differences in the modification of health behavior during the past decade.

## Hyperglycemia in Mexican Americans

There is also evidence that environmental factors exert a major influence on obesity in Mexican Americans, which in turn influences the prevalence of diabetes (Stern, Gaskill, Allen, Garza, & Gonzalez, 1981). This environmental or cultural influence could interact with a genetic predisposition through Mexican Americans' common ancestry with Native Americans, who have a documented high prevalence of diabetes (Stern, Gaskill, Allen, Garza, & Gonzalez, 1981; Stern, Pugh, Gaskill, & Hazuda, 1982). Stern, Gaskill and colleagues (1981) reported that hyperglycemia is more than twice as prevalent in Mexican American men and more than three times as prevalent in Mexican American women than in the general U.S. population. In addition, only a minority of diabetic Mexican Americans were controlling their blood sugar levels adequately (good control), with less than 1/3 of the young females being in good control. The age-adjusted mortality for diabetes has also been reported higher for Mexican Americans than other whites.

### Individual Versus Community Interventions

Given the previous discussion of CHD risk factors in the Mexican American population, one can readily hypothesize that the population is at increased risk for developing coronary heart disease over the general population. In addition, it seems that the increased risk may be due in part to cultural differences between Mexican Americans and other whites. As discussed above, an individual approach to reducing this risk would require a great deal of effort and might still fall short of its goal. Interventions designed to prevent CHD need to consider the nature of the disease, the people who have it, the tools available for its prevention, and the change agents (Nader, Taras, Sallis, & Patterson, 1987).

The medical model of an individual patient-doctor approach to risk assessment and prophylactic therapy is insufficient when the disease is so ubiquitous and insidious, when the causes are predominantly sociocultural and when therapy is nontraditional. Under these circumstances the high risk medical model of care requires the support of a broader, community-wide educational system. This is necessary for the true primary prevention of atherosclerosis (Blackburn, 1983, p. 3).

#### Behavioral Change and Systems Theory

Systems theory can provide the basis for a framework more capable of doing justice to the complexities and dynamic properties of the sociocultural system than can the traditional, individual designs (Buckley, 1967). General systems theory postulates organized interrelationships among, and predictive influences between, elements of a system which work in such ways as to promote the system's functioning (Ackoff & Emery, 1972; von Bertalanffy, 1968). General systems theory evolved from the insufficiency of isolated causal chains and mechanistic thinking, to explain the burgeoning technology of the 1940's and 1950's in the natural and social sciences. It is generally believed that the "systems concepts" arose out of the engineering and physics disciplines. And while it is true that the fields of power engineering gave way to selfcontrolling machines, it is important to realize that the impetus of general systems theory was that it be general to all scientific disciplines (von Bertalanffy, 1968).

General systems theory (GST) proposed a way of thinking that was radically different from that of most other scientific paradigms. GST challenged the idea that the whole is merely the sum of its parts. The systems ideas suggested that the whole is not only greater than the sum of its parts, but different in design, function, and scope. Traditional scientific conceptions were explicitly reductionistic, removing the subject of study from the context (the system) in which it occurred. GST attempted to recentralize the subject into its "natural" setting for study (von Bertalanffy, 1968; Minuchin, 1985; Novikoff, 1945).

Recently, the term "behavioral systems framework" has been coined to describe the relationships between context, variable interdependence, and multilevel analysis of principles from learning, social psychology, cognitive processes, environmental psychology, and marketing (Winett, 1986). The behavioral systems framework reduces to two assumptions:

1. Behavior and environment are best studied as reciprocal systems. That is, person-centered

variables must always be studied within an environmental context.

2. Reciprocity implies that behavior-environment influence is bi-directional. To be sure, the environment shapes, maintains, and constrains behavior. However, people are not passive in this process. They are architects of their environments, although their architectural plans are influenced by the prevailing environment, and the environment that is constructed, in turn, influences their behavior. (Winett, 1986, p.10)

The development of systems is frequently associated with increased complexity and differentiating processes, as in any other developing entity (Minuchin, 1985). The pairing of developmental and social constructs is not unique. However, Minuchin (1985) describes some basic principles of behavioral systems theory, (derived from GST) with a distinct developmental flavor:

(1) Any system is an organized whole, and elements within the system are necessarily interdependent. (2) Patterns in a system are circular rather than linear.

(3) Systems have homeostatic features that maintain the stability of their patterns.

(4) Evolution and change are inherent in open systems.

(5) Complex systems are composed of subsystems.
(6) The subsystems within a larger system are separated by boundaries, and interactions across boundaries are governed by implicit rules and patterns.

It is obvious from the above list of theoretical propositions that to test <u>anything</u> empirically within a systems framework is not a simple task. This blend of constructs and individuals adds a complexity to the situation that can intimidate research operationalizations. However, it is also possible to take advantage of the connectedness, and interdependency in a family, to better influence the behavior of the individual as well as the group. Systems-theoretic approaches have been considered and tested in areas such as problem solving strategies and communication patterns within the family (see Bateson, Jackson, Haley, & Weakland, 1956; Napier & Whitaker, 1978; Reiss, 1971).

Few studies have attempted to bring together a systems theory framework for both family interaction and family development. Kreppner, Paulsen, and Schuetze (1982) investigated the changes in the family system upon the arrival of, and subsequent two-year developmental period of a second child. Thev postulated the occurrence of three stages within these two years, based on major developmental milestones typically crossed by an infant. Their results parallel Minuchin's description of systems development. It is clear that the researchers observed the family as an organized whole with very interdependent parts. Minuchin (1985) and Combrinck-Graham (1985) blend the principles of developmental psychology with a family systems perspective. The results are new directions for conceptualizations, research investigations, and interventions all directed "... to regard the family as an organized system and the individual as a contributing member, part of the process that creates and maintains the patterns that regulate behavior"

(Minuchin, 1985, p. 289). Thus, not only do children and individuals develop, but families progress through stages of adaptation, diversification, and growth.

These systems conceptions can be used as a heuristic for understanding concepts of interdependency, cultural context, and communication within intervention modalities of multi-group research designs. For example and exploration, an elaborate program involving different levels of a social system was developed as part of the National Research and Demonstration Center of Atherosclerosis at Baylor College of Medicine.

### Objectives of the Current Study

The current study was part of a larger program in progress -- Cuidando El Corazon (CEC) -- to study weight loss in young Mexican American families by modifying their diet and exercise behavior patterns. The objectives of the CEC program were to: 1) promote achievement of ideal body weight, 2) promote decreases in cardiovascular risk factors, and 3) promote prevention of obesity and cardiovascular disease in children.

Three intervention groups were compared; the primary target subjects were mothers in Mexican American families. Mothers in a booklet-only group (the "comparison" group) received the intervention in a manual with information on diet, exercise, and behavior modification along with recipes for "heart healthy" Mexican dishes. Mothers in the "individualoriented" group received the same manual and also attended a total of 30 classes over the treatment year to emphasize the material in the intervention. Mothers in a "family-oriented" group received the same intervention as the individual-oriented group but in addition, received information in the booklet and in class pertaining to parenting strategies, partner communication, and effective skills for family behavior modification. Spouses of family-oriented group subjects were also encouraged to attend the classes, while in the individual-oriented and bookletonly groups they were not. Preschool-aged children also attended classes in which nutrition, exercise, and cardiovascular health were taught in ageappropriate ways.

Subjects (i.e. target mothers), spouses, and children of the respective groups were required to attend three measurement sessions during the treatment year and yearly follow-ups for the remaining three years. Adult measures included: weight, height, skinfold thicknesses, submaximal graded exercises test, plasma lipids, plasma glucose, blood pressure, pulse, 24-hour diet recall, food frequency checklist, and demographic variables such as acculturation, family structure, and social support.

In the larger program (CEC), it was hypothesized that subjects in both the family-oriented and the individual-oriented groups would be more successful in losing weight than subjects in the comparison group; with the family-oriented group losing the most weight. The current study hypothesized that there would be a greater reduction in CHD risk factor levels (as measured by total to high density lipoprotein ratio, glucose tolerance, body mass index, and blood pressure) over the initial six months of the program within the family-oriented group than in either the individual-oriented or comparison groups.

One way to conceptualize increased reductions in CHD risk factor levels in the family-oriented group is through the systems concepts mentioned above. Increased risk level reduction in the subjects with increased family involvement would be compatible with a family systems framework.

The inclusion of Mexican American cultural components such as: personalismo, fatalismo, and familismo (Suris-Rangel, 1987) also are compatible with the family systems framework. Personalismo refers to the particular emphasis that the Mexican American culture places on individualized attention. Fatalismo describes Mexican Americans as having a fatalistic view of life, not being able to control their future (Garcia-Preto, 1982; Ruiz & Padilla, 1977). The increased attention that families can bring to these issues, such as support and encouragement focussed the intervention where it was most likely to have succeeded. Probably the most relevant component to this intervention, and its strongest link to family systems, was <u>familismo</u>, or a high level of extended familism.

Mexican Americans exhibit higher levels of family cohesiveness than Anglos or Blacks (Mindel, 1980). The value placed on family includes the nuclear family as well as an extended network of grandparents, aunts and uncles, in-laws, and godparents (Arce, 1978; Vasquez-Nuttal, Avilas-Vivas, Morales-Barreto, 1984). The family's unity can not be dismissed in a program of this nature as it also stresses family priorities before individual needs, as well as complete family loyalty (Falicov, 1982).

To summarize, mothers in the family-oriented group are expected to reduce their CHD risk factors levels more than either the individual-oriented or comparison groups during the initial six months of the program. The nature of a family systems framework, combining increased family involvement and cultural appropriateness, is in keeping with the design of the family-oriented group.

#### CHAPTER II

#### METHOD

## Subjects

One hundred and seventy three families from Fort Bend County, Texas, were recruited into three one-year cohorts for the existing program, CEC. The families were recruited through media promotion and personal contacts in the local community primarily through churches and health agencies. Initial screenings were conducted to ensure that the families met the following <u>inclusion</u> criteria: 1) they were residents of Fort Bend County, Texas; 2) one or both parents were of Mexican origin; 3) they had at least one child between three and six years of age; 4) the mother was between 18 and 45 years of age; 5) the mother was at least 20% over ideal body weight.

Potential families were excluded from the study if the mother met one or more of the following <u>exclusion</u> criteria: 1) having a chronic illness which had dietary and/or exercise recommendations different from those proposed in the study, 2) being greater than 100% over ideal body weight, 3) having diastolic blood pressure measurements of 115 mm Hg or greater, or 4) being a diagnosed diabetic or having a fasting plasma glucose value greater than or equal to 140 mg/dl. The present study used only the 118 mothers from this subject pool for which CHD risk data were available from the baseline and six month measurements.

#### Procedures

Subjects from the CEC program were stratified according to weight and then randomized into one of the three treatment groups.

While the larger program involved many different questionnaire and observation procedures, the present study used only the following measurements: 1) body mass index (Weight in Kg / (Height in Meters)<sup>2</sup>), 2) total to high-density lipoprotein ratio, 3) fasting plasma glucose levels, and 4) blood pressures (systolic and diastolic).

Weight was obtained in street clothing without shoes, measured on a balance scale, and height was obtained using a secured CDC height stadiometer. BMI was used because it represents a better measure of obesity and "overweight" by taking into consideration subjects' heights (Bray, 1986).

Lipid levels were calculated from a 12-14 hour fasting blood sample collected from the subjects and analyzed for total plasma cholesterol, triglycerides, and HDL cholesterol. The cholesterol ratio was obtained by dividing the HDL by the total cholesterol value. These ratios are sensitive measures of cholesterol risk reduction. As LDL values (bad cholesterol) are reduced the HDL values (good cholesterol) increase their proportion to the total values.

A glucose tolerance test, a measure of the body's ability to metabolize glucose, was calculated by administering a standard oral glucose tolerance test to the subjects according to guidelines issued by the National Diabetes Data Group (1979). A fasting blood sample was collected, and used in the current analysis, after which a glucose dose of 75 g in a concentration no greater than 25 g/dl of flavored water was administered. A second blood sample was collected after two additional hours.

Subjects' blood pressures were taken while they sat in comfortable chairs in a quiet environment. Two consecutive blood pressures were obtained from each

subject using a standard sphygmomanometer. The mean systolic and mean diastolic blood pressures were used in the analyses.

#### CHAPTER III

#### RESULTS

### Analyses of Subject Attrition

A total of 173 women and their families were recruited in three one-year cohort groups. Fifty-five of these subjects dropped out of the study, or stopped attending measurement sessions, before the six month measurement. The attrition rates in the comparison, individual-oriented, and family-oriented groups were 27.8%, 26.2%, and 41.2% respectively. The overall attrition rate during the initial six months of the study was 31.8%.

Analyses comparing subjects who remained in the study and attended measurement sessions with those who did not found some significant differences between these two groups. The drop-outs did have significantly higher weights at baseline than those who remained in the study (<u>M</u> Difference = 14.52 lbs.), <u>F</u> (1,170) = 9.34, p < .01), however, they were also significantly taller (<u>M</u> Difference = 1.31 inches), <u>F</u> (1,170) = 15.15, p < .001). Therefore it was not surprising that baseline BMI's were not significantly different between drop-outs and those who remained in the study. Although there were differences between drop-outs and remaining subjects, those differences were comparable across the three subject groups. That is, there were no dependent measure interactions between drop-out or remaining subjects and intervention group.

#### Analysis of the Current Study

Group means and standard deviations at baseline and six months for each dependent measure are presented as follows: Table 1 - weight and BMI; Table 2 - total cholesterol, HDL, and cholesterol ratio; Table 3 - systolic and diastolic blood pressures; and Table 4 - fasting glucose.

An overall MANOVA was performed on the baseline dependent measures by each cohort year; no significant effects were found (<u>Wilk's Lambda</u> = .9243, <u>p</u> > .05) allowing the three cohorts to be collapsed into one sample, <u>N</u> = 118. To test for the degree of equality of dependent measure values in the comparison, individual-oriented, and family-oriented groups at baseline, MANOVA was performed on the combined baseline data and revealed no significant differences between groups (<u>Wilk's Lambda</u> = .8949, <u>p</u> > .05).

These results allowed the continuation of the analyses with a high degree of confidence.

The multivariate repeated measures analysis of the baseline to six months data yielded a significant Group X Time interaction (Hotteling's T = .2701, p < .01) supporting the hypothesis of an intervention effect over time.

## <u>Table 1</u>

# Group Means and Standard Deviations at Baseline and Six Months for Weight and BMI

	Weig	nt (1bs.)	BMI	
	<u>baseline</u>	<u>6 mos.</u>	<u>baseline</u>	<u>6 mos.</u>
Comparison Group	163.1	162.5	31.5	31.4
	(23.9)	(25.4)	(4.7)	<b>(4.</b> 9)
Individual-Oriented Group	171.3	165.7	33.1	32.0
Group	(34.3)	(35.1)	(5.9)	(6.0)
Family-Oriented Group	161.7	151.8	31.1	29.3
Group	(28.9)	(27.1)	(5.2)	(5.0)
All Groups Combined	165.8	160.6	32.0	31.0
Jown Theat	(29.7)	(30.2)	(5.4)	(5.5)

## <u>Table 2</u>

# Group Values and Standard Deviations at Baseline and Six Months for Total Cholesterol, HDL, and Cholesterol Ratio

	Chole	tal sterol /dl			Choles Rat _Hdl/	io
ba	seline	<u>6 mos.</u>	<u>bl.</u>	6 mos.	<u>bl.</u>	<u>6 mos.</u>
Comparison Group	186.6	184.9	46.9	46.5	.253	.254
	(24.6)	(23.9)	(10.2)	(11.0)	(.052)	(.060)
Individual-		183.8	45.3	47.1	.254	.260
Oriented Group		(10.3)	(11.7)	(27.1)	(.058)	<b>(.</b> 075)
Family-Oriented	182.2	187.0	47.7	50.3	.267	.272
Group	(29.3)	(30.4)	(12.6)	(13.6)	(.080)	(.076)
All Groups Combined	185.5	185.1	46.5	47.8	.254	.262
COMDINED	(28.2)	(26.5)	(11.5)	(12.3)	(.064)	(.071)

## <u>Table 3</u>

# Group Means and Standard Deviations at Baseline and Six Months for Systolic and Diastolic Blood Pressures

<u></u>				
	Systol	ic BP	<u>Diasto</u> ]	ic BP.
	<u>baseline</u>	6 mos.	<u>baseline</u>	<u>6 mos.</u>
Comparison Group	112.1	110.8	73.9	73.0
	(11.4)	(10.3)	(8.3)	(8.0)
Individual-	109.9	110.3	73.2	72.7
Oriented Group	(11.4)	(10.3)	(10.5)	(8.5)
Family-Oriented	111.6	111.0	72.6	71.9
Group	(13.4)	(13.7)	(9.4)	(10.8)
All Groups Combined	111.1	110.7	73.3	72.6
Comprised	(11.5)	(11.3)	(9.4)	(9.0)

# <u>Table 4</u>

# Group Means and Standard Deviations at Baseline and Six Months for Fasting Glucose

	Fasting Glucose	
	<u>baseline</u>	<u>6 mos.</u>
Comparison Group	99.9	100.2
	(15.0)	(20.1)
Individual-Oriented	94.7	93.2
Individual-Oriented Group	(15.6)	(12.8)
Family-Oriented Group	100.0	93.9
Group	(12.4)	(16.2)
All Groups	98.0	95.7
Combined	(14.7)	(16.6)
	( - 4 • / )	(10.0)

Univariate analyses revealed that significant change over time (baseline to six months) occurred only for the dependent measure of BMI (M = 1.02, SD = 1.84), <u>F</u> (2,115) = 12.616, p < .001. A Scheffe' test of simple effects (p = .05) for the mean differences in BMI over six months supported the hypotheses that the individual-oriented and family-oriented groups would show a greater reduction in a CHD risk factor level than the comparison group. Although the familyoriented group did not differ significantly from the individual-oriented group, a trend analysis revealed a significant linear trend in the BMI data, F(1,113) =24.437, p < .001). The change in BMI was progressively greater from the comparison group ( $\underline{M}$  = .145, <u>SD</u> = 1.12) to the individual-oriented group (<u>M</u> = 1.079, <u>SD</u> = 1.49) to the family-oriented group (<u>M</u> =  $1.885, \underline{SD} = 1.84$ ).

## Exploratory Analyses

Several categorization schemes were designed to investigate the nature of BMI and weight differences between the three groups over time. The decision to categorize levels of BMI and weight change was made following a review of similar schemes in practice (see

Bray [1986]; Van Itallie [1985]) and consultation with experts (J. P. Foreyt, personal communication, October 13, 1988). Criterion reference points were determined based on clinical (as opposed to statistical) significance in order to aid in interpretation. The BMI strategy used reference points of plus and minus 1.0. This procedure categorized subjects with BMI gains above 1.0 or more as gainers, subjects with BMI losses of more than 1.0 as losers, and those in between as maintainers. A BMI change of 1.0 for a five-foot four-inch person weighing 130 pounds is analogous to an eight pound difference in their weight. The weight change scheme is similar in design to the BMI strategy and uses reference points of plus and minus ten pounds. The weight system was used to increase interpretability.

Cross-tabulations were performed by familyoriented, individual-oriented, and comparison groups, and Chi-square statistics computed for each of the categorical systems. Differences in BMI yielded a significant Chi-square,  $\underline{X}^2(4, \underline{N} = 118) = 10.70, \underline{p} < .05$ . In order to interpret these results in light of unequal <u>N</u> in the three groups, percentages of subjects in each group who were gainers, maintainers, and losers are reported in Table 5. While 13% of subjects in the comparison group were "gainers", only 3% of the subjects in the family-oriented group gained in BMI. Similarly, 23% of subjects in the comparison group were losers, while 59% of subjects in the family-oriented group lowered their BMI.

Differences in the weight scheme yielded similar results and effects. The weight strategy yielded a significant Chi-square,  $\underline{X}^2(4, \underline{N} = 118) = 18.11, \underline{p} <$ .01. Percentages of subjects in each group who were gainers, maintainers, and losers are reported in Table 6. Only two subjects gained more than ten pounds (one each in the individual-oriented and comparison groups). However, 2% of subjects in the comparison group were categorized as losers, while 29% of subjects in the individual-oriented group and 44% of subjects in the family-oriented group were categorized as such.

The exploratory analyses reveal a very definite tendency for subjects in each of the three groups to change in their predicted directions. Tables 5 and 6 are clear in showing a much greater percentage of subjects in the family-oriented group losing a clinically significant level of BMI (1.0) and weight (10.0 lbs.) than the other two groups. They also display the percentages of category membership for the individual-oriented group subjects as in between those of the comparison and family-oriented groups. And, whereas some subjects in the comparison group lost or gained weight, many more of them were in the maintenance categories for both BMI and weight than subjects in the individual-oriented or family-oriented groups.

### <u>Table 5</u>

# Percentages of Group Membership Categorized as Gainers,

## Maintainers, or Losers of BMI

	<u>Gainers</u>	<u>Maintainers</u>	Losers
Comparison Group	13%	64%	23%
Individual-Oriented Group	9%	44%	47%
Family-Oriented Group	38	38%	59%
All Groups Combined	98	49%	42%

Note. Categories are defined as follows: Gainers, increase of 1.0 or more in BMI; Maintainers, any change within 1.0 (+ or -) in BMI; Losers, decrease of more than 1.0 in BMI.

## <u>Table 6</u>

# Percentages of Group Membership Categorized as Gainers,

## Maintainers, or Losers of Weight

··		
<u>Gainers</u>	<u>Maintainers</u>	<u>Losers</u>
38	95%	2%
2%	69%	29%
0%	56%	44%
2%	74%	24%
	3% 2% 0%	3% 95% 2% 69% 0% 56%

<u>Note</u>. Categories are defined as follows: Gainers, increase of 10 lbs. or more in weight; Maintainers, any change within 10 lbs. (+ or -) in weight; Losers, decrease of more than 10 lbs.in weight.

#### CHAPTER IV

#### DISCUSSION

#### Major Findings

The purpose of this study was to test the efficacy of a behavioral intervention for lowering CHD risk factor levels in Mexican American women. It was hypothesized that subjects in the family-oriented and individual-oriented interventions would reduce their levels of BMI, cholesterol ratio, systolic and diastolic blood pressure, and blood glucose more during the initial six months of the program than subjects in the comparison group. While there was a significant Group X Time interaction, suggesting a greater reduction in risk factor levels for increased level of intervention (comparison to family-oriented group), further analysis revealed a significant reduction only in BMI.

The individual-oriented and family-oriented group subjects reduced their BMI levels significantly more than the comparison group. Although the hypothesis that subjects in the family-oriented group would reduce their risk levels significantly more than those in the individual-oriented group was not supported, there was a significant linear trend in the BMI measure, revealing a linear increase in BMI reduction from comparison group to family-oriented group subjects. The results of the exploratory analyses aided substantially in the understanding and interpretability of the multivariate and univariate analyses of variance results.

Using either the BMI or weight categories of "gain", "loss", and "maintain" the results indicate that many more subjects in the family-oriented group indeed reduced their levels of CHD risk from obesity than either the individual-oriented or comparison groups. Almost half (44%) of the subjects in the family-oriented group lost more than 10 pounds during the initial six months of the program, in contrast to only 29% of subjects in the individual-oriented group and 3% of subjects in the comparison group who lost more than 10 pounds. Together with the results of the MANOVA's and ANOVA's, these results indicate a clear trend; there is a greater reduction of one CHD risk factor level in the family-oriented group than the individual-oriented group or comparison group.

### Contradictions in CHD Risk Factor Levels

Before discussing interpretations of interventions and generalizablity of these results, it is important to discuss reasons why the other CHD risk factors (other than BMI) were not significantly effected by the interventions. In the introduction to this paper, Mexican Americans were described as a population at increased risk for developing CHD as a result of their association with higher mean levels of serum cholesterol and blood pressure, as well as being at increased risk for diabetes and obesity. Except for the BMI data, the baseline data of this study do not support this premise.

Neither baseline total serum cholesterol levels nor HDL levels were considered "high" (see Table 2). For each group, in fact, the mean total cholesterol levels (186.6 md/dl, 186.9 mg/dl, and 182.2 mg/dl) are well below U.S. national "high blood cholesterol" levels, 240 mg/dl (National Cholesterol Education Program, 1988). The greatest change in those levels over the six month period was seen in the familyoriented group, which, contrary to prediction, <u>increased</u> an average of 4.8 mg/dl (NS). No group mean at any time was above 190 mg/dl, still 10 mg/dl below the beginning of the "borderline high" category proposed by the National Cholesterol Education Program (National Cholesterol Education Program, 1988).

Similarly, both mean baseline systolic and diastolic blood pressure levels are well below an "at risk" cut off (Castelli, 1984). Mean changes for these levels within groups are negligible and probably represent variability in measurement rather than changes in blood pressure.

The fasting glucose levels were, for the most part, lower than levels associated with noninsulindependent diabetes mellitus (NIDDM) (National Diabetes Data Group, 1979), due to the fact that diabetic subjects were not allowed into the study. In addition, even a more sensitive diabetic disease categorization procedure involving both the fasting glucose levels and a glucose tolerance test, described above, revealed no significant changes in NIDDM classification between groups (National Diabetes Data Group, 1979).

The implications of these cholesterol, blood pressure, and blood glucose levels suggest that this particular sample was not at increased risk for CHD from these risk factors and, therefore, would probably not respond to a nonpharmacological intervention.

Weight and BMI were the only risk factors consistently above an "at risk level" (Foreyt, 1987; Hubert, Feinleib, McNamara, & Castelli, 1983) and also showed the greatest changes over the six month period.

Results of subjects with BMI levels indicating "obesity" ( > 30.0) who also have blood pressure, blood glucose, and cholesterol levels below "risk" cut-offs are counterintuitive as well as contradictory to previous findings. In general, previous research has conceptualized these CHD risk factors as occurring together with obesity (National Institutes of Health Consensus Development Panel on the Health Implications of Obesity, 1985). However, the present results suggest that the obese Mexican American women in this study, while at increased risk for CHD from obesity, are at no increased risk for hypertension, hyperglycemia, and hypercholesterolemia. This could be an important finding for obesity research and must be replicated before conclusions can be drawn.

It should be noted that subjects in this study were selected to be obese, non-diabetic, and not taking any medication that would be interfered with by this program (e.g., certain medications for hypertension). Therefore, subjects should not have had very high levels of fasting glucose or blood pressure. However, it was still hypothesized that their levels would be high enough to warrant modification.

In addition, subjects in this study were primarily in their late twenties or early thirties. Although CHD risk associated with cholesterol levels is now calculated independently of age, hypertension and hyperglycemia still hold very strong positive relationships with age and severity. Therefore, while these subjects may currently not be at risk for CHD through hyperglycemia or hypertension, their obesity may raise their CHD risk from these factors as they age.

Although these results do not suggest strong relationships among the changes in CHD risk factors, it is still important to continue to conceptualize, test, and analyze these types of CHD risk studies using a multivariate approach. While the risk factors may be unrelated as measured, their physiological effects are multiplicative (Castelli, 1984; Grundy, 1986).

#### The Intervention and Systems Theory

The increased reduction of BMI found in the family-oriented group as compared to the individualoriented and comparison groups supports a discussion of the merits and demerits of using a systems framework in this type of field research. The positive results of the trend analysis and exploratory analyses supporting the family-oriented intervention indicate something different going on in that group than the others. As designed, the family-oriented intervention was only to differ from the individualoriented intervention on the inclusion of methods of family communication, spouse support and attendance at the classes, and child class instruction. From a research perspective, these are difficult manipulations to check. From a theoretical

perspective, they represent a more "real" concept of the most prominent social organization, the family.

By including concepts of communication and support within a family framework the challenge, effort, and reward of weight loss can be diffused throughout the family. In this way, husbands and children can help the mother achieve her goals, and benefit themselves, through better exercise and eating habits.

In contrast, the individual-oriented group subjects were placed in the position of a mother in a traditional weight loss program (Foreyt & Cousins, 1987). The nutrition and behavior change instructions were the same as in the family-oriented group, but the mother had to go home and do it herself. The comparison group provided additional contrast to the family-oriented group by just receiving the program materials and not benefitting from the instruction. Neither of these two interventions produced as great a reduction in BMI reduction as the full family-oriented intervention. However, in order to interpret these results correctly, it is necessary to discuss what

actually happened in the interventions as well as what was planned.

While the ideas of greater family involvement for the family-oriented group were laid out in the proposals for this program, the program was implemented by the class instructors -- dieticians not acquainted with these concepts. Despite continued training and a manual with explicit directions, instructors often fell back to their traditional vocational roles instead of following strict methodological guidelines. Some of the instructors were required to teach both individual-oriented and family-oriented groups and this could have posed an additional threat by placing the instructors in the position of feeling compelled to give both groups the same information (see discussions of, compensatory equalization of treatments by Cook & Campbell, 1979).

In addition there were cultural barriers to structuring planned group comparisons for family vs. individual-oriented involvement. The Mexican American family culture, as described above, does not support individualism over familism. It is possible that the fathers felt that the issues of weight loss are their wives' problems, as cooking, meal planning, and health are all female issues and responsibilities in traditional Mexican American families. Therefore, although the family group was designed for the full inclusion of the entire family, the changes were directed primarily at the mother and may have alienated the father causing his reluctance to participate. There are few documented reports supporting or refuting these ideas.

It is difficult to assess quantitative and qualitative changes in systemic involvement in this study, however, Suris-Rangel (1987), investigating the effects of social support on weight loss in the CEC program, found high amounts of general social support across all groups, although not related to weightloss. A possible indirect measure of spousal support within the family group are the attendance levels of the spouses at the classes. It can be inferred from project reports that spousal attendance at the "family" classes was far less than expected and, therefore, spousal support may have resembled that of the individual-oriented group. Other indirect measures are the reports by research assistants in the field suggesting that the family-oriented group members acted similarly to the individual-oriented group members regarding class participation and attendance. No evaluation component was built into the program, so further analyses of this situation may be difficult.

Despite some evidence that the family systems framework may not actually fit with the resulting intervention, it is encouraging that several strong results, primarily the trend analysis and exploratory analyses, indicate a tendency in CHD risk lowering in the predicted directions.

#### Generalizablity of the Results

Analyses of the drop-out rates between groups yielded an interesting finding; the rate of attrition in the family-oriented group (41.2%) was approximately 50% greater than the comparison (27.8%) or individualoriented group (26.2%). This result is surprising given that studies designed like the current one traditionally produce higher attrition rates in the comparison groups rather than the treatment groups. The CEC program is currently planning several investigations into this effect including hypotheses that: the family-oriented group subjects "had" to work harder and had increased responsibility than the other subjects, prompting those less committed to withdraw; and teachers of family-oriented group classes may not have been equivalent in their presentations and thus produced different drop-out rates between classes.

The high attrition rate and the significant differences between those who dropped out and those who remained in the study may question the validity of the statistical conclusions drawn from this sample. However, as there were no significant interactions of attrition with intervention groups, there still remains a good deal of confidence in the results. There is, though, a question as to the representativeness of this sample for generalizing back to the original Mexican American population.

Further analyses of this program may be able to tease out additional differences between the drop-outs and those who remained. There is also the possibility that strong recruitment procedures achieved a biased initial sample by recruiting a group of people that dropped out when the recruitment was over. These

factors make it difficult to generalize these results directly to the Mexican Americans in Fort Bend County, Texas.

Comparison of the dependent measures' values in this study to similar measurements of other Mexican American populations reveals a generally lower risk factor profile for subjects in this study. Values of serum cholesterol, systolic and diastolic blood pressures, and fasting glucose levels were lower for subjects in this study than comparison samples, even though subjects in this study were obese (Stern, Haskell, Wood, Osann, King, & Farguhar, 1975). It does not seem plausible that the intervention was only successful because the other CHD risk factor levels Therefore, the interventions of this study were low. could be disseminated to other Mexican American samples and populations for analysis. However, there is still a question of these results generalizing to a general "at risk" population.

The major findings in this study support a culturally appropriate, behavioral, and familyoriented intervention for reducing body mass index and obesity in obese Mexican American women. There is some evidence that obesity makes an independent contribution to CHD risk (Hubert, Feinlab, McNamara, & Castelli, 1983; Kannel & Dawber, 1972; Miller & Shekelle, 1976). Therefore, a reduction in obesity (as measured by BMI) is also likely to reduce the risk for CHD.

In light of this, the current study suggests that the intervention may also be helpful for lowering risk levels of CHD attributable to obesity in other populations as well. The main components of the intervention were cultural specificity, behavioral direction, and family orientation. Cultural specificity should be an important consideration for any ethnic population, not just Mexican Americans. Similarly, behavioral interventions are generally accepted as more productive than traditional techniques (e.g., basic dieting) (Foreyt & Cousins, 1987).

The inclusion of the family and family systems theory concepts have been shown to increase health behaviors in other studies of Mexican Americans (Sallis, Patterson, Buono, Atkins, & Nader, 1988). It seems possible that the family approach might prove to be efficacious, and essential, for providing CHD risk factor interventions with clinical significance for other populations as well.

### <u>Conclusions</u>

The strength of this study lies in its support of the CEC program for demonstrating the effectiveness of the family-oriented intervention for lowering CHD risk levels attributable to obesity in this Mexican American sample. A greater proportion of the familyoriented group subjects lost a clinically significant amount of weight than subjects in the either the individual-oriented or comparison groups. There was also a linear trend in BMI reduction, greater BMI reduction in the family-oriented group than the individual-oriented and comparison groups.

Although the subjects in this study were clearly obese, they did not have elevated risk levels of serum cholesterol, glucose, or blood pressure. This contradicts much of the previous knowledge on obesity and CHD risk factors and requires further study to fully understand.

The success of the family-oriented group can be explained in terms of a family systems conception. Greater involvement of family members in the ongoing processes of diet, exercise, and life-style modification takes advantage of the naturally occurring interdependencies and connectedness of a family. The inclusion of familismo in Mexican American family culture is even more important than in average American homes.

Continued support of family and family systems conceptions in CHD risk factor level reduction for average risk, as well as increased risk, populations should not only increase the efficacy of interventions, but also the generalizablity and expandability of the results.

#### REFERENCES

- Ackoff, R. L. & Emery, F. E. (1972). <u>On purposeful</u> <u>systems</u>, Chicago: Aldine-Atherton.
- Arce, C. H. (1978, October). <u>Dimensions of familism</u> <u>and familial identification.</u> Paper presented at the C.O.S.S.M.H.O. Conference on the Hispanic Family, Houston, Texas.
- Baron, A. E., Freyer, B., & Fixler, D. E. (1986). Longitudinal blood pressures in blacks, whites, and Mexican Americans during adolescence and early adulthood. <u>American Journal of</u> Epidemiology, 123, 809-817.
- Bateson, G., Jackson, D. D., Haley, J., & Weakland, J. (1956). Toward a theory of schizophrenia. <u>Behavioral Science</u>, <u>1</u>, 251-264.
- von Bertalanffy, L. (1968). General systems theory: <u>Foundations, development, applications</u>, New York: George Braziller.
- Blackburn, H. (1975). Coronary risk factors: How to evaluate and manage them. <u>European Journal of</u> <u>Cardiology</u>, <u>2</u>, 249-283.

Blackburn, H. (1983). Diet and atherosclerosis:

Epidemiologic evidence and public health implications. <u>Preventive Medicine, 12</u>, 2-10.

- Bray, G. A. (1986). Effects of obesity on health and happiness. In K. D. Brownell & J. P. Foreyt (Eds.), <u>Handbook of eating disorders</u> (pp. 3-44). New York: Basic Books.
- Brown, M. S. & Goldstein, J. L. (1983). Lipoprotein receptors in the liver: Control sign for plasma cholesterol traffic. <u>Journal of the Clinical</u> <u>Investigator</u>, <u>72</u>, 743-747.

Buckley, W. (1967). <u>Sociology and modern systems</u> <u>theory</u>, Englewood Cliffs, NJ. : Prentice-Hall. Castelli, W. P. (1984). Epidemiology of coronary heart disease: The Framingham study. <u>The</u> <u>American Journal of Medicine</u>, <u>Feb. 27</u>, 4-12. Combrinck-Graham, L. (1985). A developmental model for family systems. <u>Family Process</u>, <u>24</u>, 139-150. Cook, T. D. & Campbell, D. T. (1979). <u>Quasi-</u>

<u>experimentation -- Design & analysis issues for</u> <u>field settings.</u> Boston: Houghton Mifflin Company.

- Enos, W. F., Holmes, R. H., & Beyer, J. (1953). Coronary disease among United States soldiers killed in action in Korea: Preliminary report. Journal of the American Medical Association, 152, 1090-1093.
- Falicov, C. (1982). Mexican families. In M. McGoldrick, J. Pearce, & J. Giordana (Eds.), <u>Ethnicity and family therapy</u>. New York: Guilford Press.
- Farquhar, J. W., Fortmann, S. P., Maccoby, N., Wood, P. D., Haskell, W. L., Taylor, C. B., Flura, J. A., Solomon, D. S., Rogers, T., Adler, E., Breitrose, P., & Weiner, L. (1984). The Stanford five-city project: An overview. In J. O. Matarazzo, J. A. Herd, N. E. Miller, & S. M. Weiss (Eds.), <u>Behavioral health: A handbook of health</u> <u>enhancement and disease prevention</u> (pp. 1154-1165). New York: Wiley.
- Foreyt, J. P. (1987). Issues in the assessment and treatment of obesity. Journal of Consulting and <u>Clinical Psychology</u>, <u>55</u>, 677-684.

- Foreyt, J. P. & Cousins, J. H. (1987). Obesity. In M. Hersen & V. B. Van Hasselt (Eds.), <u>Behavior</u> <u>therapy with children and adolescents: A clinical</u> <u>approach</u>, (485-511). New York: John Wiley & Sons.
- Franco, L. J., Stern, M. P., Rosenthal, M., Haffner, S. M., Hazuda, H. P., & Comeaux, P. J. (1985). Prevalence, detection, and control of hypertension in a biethnic community. <u>American</u> <u>Journal of Epidemiology</u>, 121, 684-696.
- Garcia-Preto, N. (1982). Puerto Rican families. In M. McGoldrick, J. Pearce, & J. Giordano (Eds.), <u>Ethnicity and family therapy</u>. New York: Guilford Press.
- Gertler, M. M. & White. P. D. (1954). <u>Coronary heart</u> <u>disease in young adults: A multidisciplinary</u>

study. Cambridge MA: Harvard University Press.

- Glendy, R. E., Levine, S. A., & White, P. D. (1937). Coronary disease in youth. <u>Journal of the</u> <u>American Medical Association</u>, <u>109</u>, 1775-1781.
- Grundy, S. M. (1986). Cholesterol and coronary heart disease -- A new era. Journal of the American <u>Medical Association</u>, 256, 2849-2858.

- Hanis, C. L., Ferrell, R. E., & Schull, W. T. (1985). Hypertension and sources of blood pressure variability among Mexican Americans in Starr County, Texas. <u>International Journal of</u> Epidemiology, 14, 231-238.
- Hubert, H. B., Feinlab, M., McNamara, P. M., & Castelli, W. P. (1983). Obesity as an independent risk factor for cardiovascular disease: A 26-year follow-up of participants in the Framingham heart study. <u>Circulation</u>, <u>67</u>, 968-977.
- Keys, A. (1980). <u>Seven countries: A multivariate</u> <u>analysis of death an coronary heart disease</u>. Cambridge, MA: Harvard University Press.
- Kannel, W. B. & Dawber, T. R. (1972).
  Atherosclerosis: A pediatric problem. Journal of
  Pediatrics, 80, 544-554.
- Kraus, J. F., Borhani, N. O., & Frant, C. E. (1980). Socioeconomic status, ethnicity, and risk of Coronary heart disease. <u>American Journal of</u> <u>Epidemiology</u>, <u>111</u>, 407-414.

- Kreppner, K., Paulsen, S., & Schuetze, Y. (1982). Infant and family development: From triads to tetrads. <u>Human Development</u>, <u>25</u>, 373-391.
- Levy, R. I. (1981). Review: Declining mortality in coronary heart disease. <u>Arteriosclerosis</u>, <u>1</u>, 312-325.
- Lipid Research Clinics Program. (1984a). The lipid research clinics coronary primary prevention trial results I. Reduction in incidence of coronary heart disease. Journal of the American <u>Medical Association</u>, 251, 351-364.
- Lipid Research Clinics Program. (1984b). The lipid research clinics coronary primary prevention trial results II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. Journal of the American Medical Association, 251, 365-374.
- Marvin, H. M. (1937). Abstract of discussion to Gertler, Levine, and White. Journal of the American Medical Association, 109, 1781.

- McGill, H. C. (1980). Morphologic development of the atherosclerotic plaque. In R. M. Laurer & R. B. Shekelle (Eds.), <u>Childhood prevention of</u> <u>atherosclerosis and hypertension</u> (41-50), New York: Raven Press.
- McNamara, J. J., Malot, M. A., Stremple, J. F., & Cutting, R. I. (1971). Coronary artery disease in combat casualties in Vietnam. <u>Journal of the</u> <u>American Medical Association</u>, <u>216</u>, 1185-1187.
- Miller, R. A. & Shekelle, R. B. (1976). Blood pressure in tenth-grade students. <u>Circulation</u>, <u>54</u>, 993-1000.
- Mindel, C. H. (1980). Extended familism among urban Mexican Americans, Anglos, and Blacks. <u>Hispanic</u> <u>Journal of Behavioral Sciences</u>, <u>2</u>, 21-34.
- Minuchin, P. (1985). Families and individual development: Provocations from the field of family therapy. <u>Child Development</u>, <u>56</u>, 289-302. Multiple Risk Factor Intervention Trial Research Group. (1982). Multiple Risk Factor Intervention Trial. Journal of the American Medical Association, <u>248</u>,

1465-1477.

Nader, P. R., Taras, H. L., Sallis, J. F., & Patterson, T. L. (1987). Adult heart disease prevention in childhood: A national survey of pediatricians' practices and attitudes. <u>Pediatrics</u>, <u>79</u>, 843-850. Napier, A. Y. & Whitaker, C. A. (1978). <u>The family</u>

crucible, New York: Harper & Row.

National Cholesterol Education Program. (1988).

Report of the Expert Panel on detection,

evaluation, and treatment of high blood

<u>cholesterol in adults.</u> (NIH Publication No. 88-2925). Washington, DC: U.S. Government Printing Office.

- National Diabetes Data Group. (1979). Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. <u>Diabetes</u>, <u>28</u>, 1039-1057.
- National Institutes of Health Consensus Development Panel on the Health Implications of Obesity. (1985). Health implications of obesity: National Institutes of Health consensus development conference statement. <u>Annals of</u> <u>Internal Medicine</u>, <u>103</u>, 1073-1077.

Novikoff, A. B. (1945). The concept of integrative levels of biology. Science, 101, 209-215.

- Ragland, D. R. & Brand, R. J. (1988). Coronary heart disease mortality in the Western Collaborative Group Study: A follow-up experience of 22 years. <u>American Journal of Epidemiology</u>, <u>127</u>, 462-475.
- Reiss, D. (1971). Varieties of consensual experience.
  I: A theory for relating family interaction to individual thinking. <u>Family Process</u>, <u>10</u>, 1-28.
- Ruiz, R. & Padilla, A. (1977). Counseling Latinos. Personnel and Guidance Journal, <u>55</u>, 213-231.
- Sallis, J. F., Patterson, T. L., Buono, M. J., Atkins, C. J., & Nader, P. R. (1988). Aggregation of physical activity habits in Mexican-American and Anglo families. <u>Journal of Behavioral Medicine</u>, <u>11</u>, 31-41.
- Stern, M. P., Gaskill, S. P., Allen, C. R., Garza, V., Gonzalez, J. L., & Waldrop, R. H. (1981). Cardiovascular risk factors in Mexican Americans in Laredo, Texas I. Prevalence of overweight and diabetes and distributions of serum lipids. American Journal of Epidemiology, 113, 546-555.

- Stern, M. P., Gaskill, S. P., Allen, C. R., Garza, V., Gonzalez, J. L., & Waldrop, R. H. (1981). Cardiovascular risk factors in Mexican Americans in Laredo, Texas II. Prevalence and control of hypertension. <u>American Journal of Epidemiology</u>, <u>113</u>, 556-562.
- Stern, M. P., Haskell, W. L., Wood, P. D. S., Osann, K. E, King, A. B., & Farquhar, J. W. (1975). Affluence and cardiovascular risk factors in Mexican Americans and other whites in three Norther California communities. Journal of Chronic Disease, 28, 623-636.
- Stern, M. P., Pugh, J. A., Gaskill, S. P., & Hazuda, H. (1982). Knowledge, attitudes, and behavior related to obesity and dieting in Mexican Americans and Anglos: The San Antonio heart study. <u>American Journal of Epidemiology</u>, <u>115</u>, 917-928.
- Suris-Rangel, A. (1987). Weight loss in Mexican Americans as related to acculturation and social support. Unpublished master's thesis, University of Houston, Houston, TX.

- U. S. Department od Commerce, Bureau of the Census. (1987). <u>Statistical Abstract of the United</u> <u>States</u>. Washington, D.C.: U.S. Government Printing Office.
- Van Itallie, T. B. (1985). Health implications of overweight and obesity in the United States. <u>Annals of Internal Medicine</u>, <u>103</u>, 983-988.
- Vasquez-Nuttal, E., Avilas-Vivas, Z., & Morales-Barreto, G. (1984). Working with Latin American families. In J. C. Hansen (Ed.), <u>Family therapy</u> with school related problems, (pp. 74-90). Rockville, MD: Aspen Systems Corporation.
- Winett, R. (1986). <u>Information and behavior: Systems</u> <u>of influence</u>, Hillsdale, NJ: Lawrence Erlbaum Associates.