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PSYCHOSOCIAL VARIABLES AND THEIR RELATIONSHIP TO DIABETIC  
OUTCOME AMONG THE STRONG HEART STUDY COHORT

by

Brian D. O'Leary

A dissertation submitted in partial fulfillment  
of the requirements for the degree

of

DOCTOR OF PHILOSOPHY

in

Psychology

Approved:

UTAH STATE UNIVERSITY  
Logan, Utah

2007

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## ABSTRACT

Psychosocial Variables and their Relationship to Diabetic  
Outcome Among the Strong Heart Study Cohort

by

Brian D. O'Leary, Doctor of Philosophy

Utah State University, 2007

Major Professor: Dr. Kevin S. Masters  
Department: Psychology

Diabetes mellitus is one of the leading causes of death and disability in the United States. Certain Native American groups have been harder hit than the mainstream population, both in prevalence of the disease and in rates of related complications. The highest known prevalence in the world is found among a Southwestern U.S. Tribe, and other Native American Tribes have demonstrated similar prevalence rates. It has been shown that certain psychological factors such as depression and hostility impact both the occurrence and outcome of certain diseases, including diabetes.

This study examined whether those individuals who have not met the criteria for diabetes mellitus were more prone to develop diabetes mellitus if they reported signs of depression, cynical hostility, or anger that is either expressed or not expressed. It also examined the impact of depression, cynical hostility, and anger on glucose control among individuals who were diagnosed with diabetes mellitus. Finally, an aim was to determine if "psychological distress," rather than specific psychosocial variables, was related to poorer diabetic outcomes among a specific Native American population.

Participants for this study were part of the Strong Heart Study and were examined at two different points in time (1992-1994 and 1997-1999).

The current study found that psychosocial variables did not predict the incidence of diabetes mellitus. Depression was found to impact glucose control among individuals without diabetes or impaired glucose tolerance, but psychosocial variables did not appear to have any measurable influence on glucose control among those individuals with diabetes mellitus or impaired glucose tolerance. Overall, it appears that psychosocial variables do not play as large of role in both incidence and outcome among certain Native American tribes as has been shown among the mainstream population.

(139 pages)

DEDICATION

For my very patient wife.

## ACKNOWLEDGMENTS

I would like to take this opportunity to thank both the investigators and participants of the Strong Heart Study, without whom this study would not have been possible. In particular, I would like to acknowledge Dr. Barbara Howard and Dr. Tom Welty, whose encouragement, interest, and mentorship have had a huge impact on my development as a health professional. I would also like to express appreciation to my committee members, Kevin Masters, Ph.D., Carolyn Barcus, Ed.D., Scott DeBerard, Ph.D., Edward Heath, Ph.D., and Christopher Corcoran, Ph.D. Their unyielding persistence and guidance were essential for the completion of this project.

Brian D. O'Leary

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## CHAPTER I

### PROBLEM STATEMENT

“Sadness or long sorrow, as likewise convulsions, and other depressions and disorders of the animal spirits are used to generate or foment this morbid disposition.” This is how Thomas Willis described the relationship between diabetes and depression in 1684 (as cited in Geringer, 1990, p. 239).

As the quality of life and the standard of medical care have continued to increase in the United States, the nature of the diseases that impact our population has changed. At the turn of the 20<sup>th</sup> century, infectious diseases were the leading cause of illness and mortality. At the beginning of the 21<sup>st</sup> century, the diseases that impact our population have distinct behavioral and psychological components. The probability of contracting heart disease, cancer, or diabetes mellitus is influenced by the behaviors that people either practice or abstain from throughout their life. Other behaviors such as eating habits, substance abuse, activity levels, and unsafe sexual practices have been shown to be highly related to long-term morbidity and mortality rates as well as lowering a person's overall quality of life. New pathways have been discovered and are currently being researched, finding that a person's overall mental health, both acute and chronic, can directly impact the course of disease. Understanding psychosocial variables that influence individual choices and the course of disease is critical if we are to continue improving standards of care and the overall quality of life of people.

Diabetes mellitus has become a leading cause of death and disability. In 2005, the Centers for Disease Control and Prevention (CDC) estimated that diabetes affects 20.8 million people in the U.S., or about 7.0% of the population. About half of those individuals with diabetes may be unaware of their disorder and thus do not take steps to treat it (Bellenir, 1999). According to the CDC (1999), there has been a 19% increase in

the number of known diabetes cases from 1980 to 1996, of which only one third can be accounted for by an increase in age. According to the American Diabetes Association (ADA; 2000), 2,200 individuals are diagnosed with diabetes every day in the U.S.

Diabetes costs those who suffer from it and their families in many ways. It is a slow killer, with its complications taking years to manifest themselves. Diabetic individuals who do not, or are unable to, take control of their glucose levels can expect increased risk for cardiovascular disease, stroke, renal failure, nerve damage, blindness, and lower limb amputation. There is also an emotional cost to diabetes, both to the diabetic who suffers and the family members who have to sit by and watch their loved one deteriorate. As the U.S. population continues to age, we can expect more families to be affected by diabetes.

Certain Native American tribes have been harder hit by diabetes than have other groups. The highest known prevalence of diabetes in the world is found among Southwestern U.S. tribes. What is more alarming is that before 1940, diabetes was almost unknown among Native Americans (West, 1974). Not only is the overall rate of diabetes high among native populations, but complications associated with diabetes appear to occur at a higher rate in this population than in the general diabetic population. For example, the rate of diabetic end-stage renal disease (kidney failure) is six times higher among Native Americans (ADA, 2000). Native Americans also have a 3-4 times higher rate of lower limb amputations compared to the general population (ADA, 2000). Thus far, there has been limited research examining how psychosocial factors such as depression, hostility, anger, and general psychological distress impact diabetes among Native American populations.

This study (a) examined whether those individuals who have not met the criteria for diabetes mellitus are more prone to develop diabetes mellitus if they report signs of

depression, cynical hostility, or anger that is either expressed or not expressed; (b) examined the impact of depression, cynical hostility, and anger on glucose control among those individuals who are diagnosed with diabetes mellitus; and (c) determined if “psychological distress,” rather than specific psychosocial variables, is related to poorer diabetic outcome among a specific Native American population.

### Purpose and Objectives

It has been suggested that psychosocial factors play a role in disease outcome. The purpose of this study is to examine three related but distinct objectives among a specific Native American population over a period of 5 years.

1. Examine whether those individuals who have not met the criteria for diabetes mellitus are more prone to develop diabetes mellitus if they show signs of depression, cynical hostility, or report anger that is either expressed or unexpressed. The selection of psychosocial variables has been based on past research with this population, which demonstrated that depression, cynical hostility, and anger demonstrated a statically significant relationship with diabetic outcome variables, while other psychosocial variables did not, when observed in a cross-sectional model (O’Leary, 2001).

2. Examine the impact of depression, cynical hostility, and anger on glucose control among those individuals who are diagnosed with diabetes mellitus. Fasting glucose, a commonly used measure of glucose control, will be used and is available for all participants in the Strong Heart Study.

3. Determine if “psychological distress,” rather than specific psychosocial variables, is related to poorer diabetic outcome among a specific Native American population.

## Hypotheses

### *Hypothesis 1*

There is a positive relationship between psychosocial factors, such as depression, hostility, and anger, and the incidence of new cases of diabetes mellitus among a Native American population. It is hypothesized that those individuals with scores on psychosocial instruments that indicate depressive symptoms, cynical hostility, or anger will be more likely to develop diabetes mellitus over a 5-year period than those individuals with lower scores on the same instruments.

### *Hypothesis 2*

There is a negative relationship between psychosocial factors such as depression, hostility, and anger, and glucose control among individuals with either abnormal glucose tolerance or diabetes mellitus. It is hypothesized that those individuals who have higher scores on psychosocial instruments at time 1 will show greater increases in glucose levels over a 5-year period of time than those individuals who have lower scores on the same instruments.

### *Hypothesis 3*

There is a positive relationship between psychological distress, as measured by the Rand Short Form-36 mental health composite score, and diabetic-related outcome variables such as glucose control, perceived physical health, peripheral nerve damage, and foot ulcers over a 5-year period. It is hypothesized that those individuals who score lower on the mental health composite variable at time 1 will have poorer diabetic outcomes.

## CHAPTER II

### LITERATURE REVIEW

The search of the literature for this review, utilized the ERIC, Psychlit, Pubmed, and Medline computer databases. The key words used were: *Native American, American Indian, Indian, Sioux, Cheyenne River, Diabetes, Diabetes Mellitus, Psychosocial, Stress, Depression, Hostility, Anger, Obesity, Outcome, Quality of Life, Glycemic Control, Social Support, Strong Heart Study, type 2 diabetes, and Non Insulin Dependent Diabetes Mellitus*. The reference lists included with each article were also searched to find relevant materials.

#### Introduction to Diabetes

Diabetes as an illness has been recognized for over 3,000 years, with the side effect of “urinating honey” being recognized by early healers. It was first documented by Aretaeus, over 2,000 years ago, who named it diabetes, meaning “to run through a siphon.” In the 1700s, the term mellitus was added, which means “honey” (as cited in Gatchel & Oordt, 2003). By the 19<sup>th</sup> century, physicians had identified two different types of diabetes. One appeared in childhood wherein the patient would die shortly after the onset of symptoms; today we identify that as type 1 diabetes. The second appeared in obese people and could be treated with weight loss and an improved diet, which we now identify as type 2 diabetes (Surwit & Schneider, 1993).

Diabetes is a family of metabolic disorders, in which insulin is either not produced, produced in inadequate quantities, or the cells manifest resistance to the insulin that decreases the effectiveness of insulin in transferring glucose across the cell membrane. Most food is broken down into a simple sugar called glucose, which is the



primary source of fuel for the body. After food is broken down into glucose, it enters the bloodstream to be carried to individual cells. Insulin is a hormone that allows glucose to be transported across the cell membrane. In individuals without diabetes, glucose metabolism is closely regulated. If glucose levels start to rise above the normal range, the pancreas releases insulin. This lowers glucose in the bloodstream by inducing glucose utilization or storage in the liver or fat cells. When a nondiabetic person's glucose level falls below the normal range, the pancreas releases glucagon that induces the release of stored glucose out of the liver and fat cells for use by the body (Peyrot, McMurry, & Kruger, 1999).

There are three cardinal signs of diabetes mellitus. The first is polyuria, which is an excessive urine output that leads to decreased blood volume and dehydration. This is caused by the excess glucose in the kidney filtrate that inhibits water reabsorption by the kidney tubules. The second is polydipsia, or excessive thirst. Electrolyte loss due to dehydration results in sodium and potassium ion loss, which, in turn, causes abdominal pain and possible vomiting that causes the body's stress reaction to become even higher. The final sign is polyphagia, or excessive hunger and food consumption. Even though plenty of glucose is available the body cannot use it and, instead, starts to utilize its fat and protein stores as sources of metabolism, leading to what has been described as "starving in the land of plenty" (Marieb, 2004). In both types of diabetes, the lack of insulin will cause a person's glucose levels to fluctuate and rise to dangerous levels. The diagnostic criteria for diabetes are an impaired fasting plasma glucose level of  $\geq 126$  mg/dl or nonfasting glucose level of  $\geq 200$  mg/dl. The results of a positive test need to be replicated a second time on another day to confirm the diagnosis (ADA, 2003a).

The CDC (Center for Disease Control and Prevention, 2005) estimates that there are 20.8 million people in the U.S. with diagnosed diabetes. Minority populations seem

to be showing a greater increase in prevalence of diabetes (CDC, 1998). The prevalence of diabetes is higher among certain ethnic and racial minority groups and increases with age among all groups (ADA, 2003a). In certain minority groups, the rate of diabetes is estimated to be increased two- to five-fold (Culpepper, 2002). Diabetes is the leading cause of blindness, and accounts for 35% of end stage renal disease cases in the U.S. A large proportion of patients do not achieve tight control of glucose levels and many are simply not treated. Tight control of glucose levels delays the onset or progression of end organ diseases including renal disease, blindness, cerebrovascular disease, and coronary artery disease (Culpepper). Glasgow, Toobert, Hampson, and Wilson (1995) predicted that because one of the risk factors for type 2 diabetes is age, we can expect diabetes to become an even greater public health concern as the U.S. population ages.

### Type 1 Diabetes

In some people, the pancreas produces either too much or too little insulin. Individuals who have little or no insulin produced are designated as having type 1 diabetes. According to a CDC diabetes fact sheet (Center for Disease Control and Prevention, 1997), type 1 diabetes, also called insulin-dependent diabetes mellitus, accounts for only 5-10% of diagnosed cases of diabetes in the U.S. with approximately 750,000 Americans having this disease (Marieb, 2004). Type 1 diabetes is considered an autoimmune disease (Bellenir, 1999) in that the body's immune system attacks the beta cells in the pancreas, which are the cells that produce insulin, and destroys them. The result of the beta cell destruction is insufficient or no insulin being produced to metabolize carbohydrates. Before insulin was discovered in 1921, individuals with type 1 diabetes died shortly after the appearance of the disease (Bellenir). Type 1 diabetes typically onsets between the ages of 11 and 14 years (Chessler & Lernmark, 2000) with

symptoms appearing suddenly. Predating these symptoms is a long asymptomatic period during which beta cells are continually being destroyed by an autoimmune response (Marieb). It is thought that the cause of this type of diabetes lies in a combination of many variables, including: genetics, environmental factors such as chemicals, pharmaceuticals, and viral infections, and possibly an insult to the fetus in utero (Chessler & Lernmark, 2000). Some investigators now believe that molecular mimicry is at least a part of the process. They believe that some foreign substance, a virus for example, has entered the body and is so similar to certain beta cell proteins that the immune system attacks the beta cells as well as the invading substance (Marieb).

### Type 2 Diabetes

Sometimes the problem is not a lack of insulin, but the development of insulin resistance, so that the insulin induces the transport of either little or no glucose across the cell membrane. This is called type 2 diabetes (Peyrot et al., 1999). While the true etiology of type 2 diabetes is unknown, it is thought to be a disorder in which both environmental and genetic factors play a role (Gohdes & Acton, 2000).

There are two defects that can occur in type 2 diabetes. The first is similar to type 1 diabetes in that the pancreas' ability to produce insulin is impaired. Over the course of the disease, the pancreas compensates for the insulin resistance by releasing increasing amounts of insulin. At some point, the pancreas cannot keep up with the body's demands and the pancreas simply wears out, making it necessary for type 2 diabetics to supplement their treatment with insulin (Peyrot et al., 1999).

The second is insulin resistance. The insulin that is available in the bloodstream is not able to stimulate glucose transport across the cell membrane (Gohdes & Acton, 2000). Most people with diabetes have type 2, which usually develops in people over the

age of 40 years (Bellenir, 1999), and accounts for 90-95% of all diagnosed cases of diabetes (Center for Disease Control and Prevention, 1997). One of the primary risk factors for developing type 2 diabetes is obesity. Most individuals with type 2 diabetes are obese or have been at some point in their lives (Gohdes & Acton). Apparently adipose cells of obese, sedentary individuals overproduce a hormone-like chemical that depresses synthesis of glucose translocation protein. This protein enables glucose to pass through insulin-primed plasma membranes. Without glucose translocation protein cells cannot take up glucose (Marieb, 2004). Some of the symptoms of type 2 diabetes include fatigue, increased and unusual thirst, weight loss, blurred vision, increased urination, frequent infections, and sores that are slow to heal (Bellenir; Gohdes & Acton). The early symptoms of type 2 diabetes are typically less noticeable than type 1 diabetes (Bellenir).

Unlike type 1 diabetes, many with type 2 diabetes are able to manage their illness without the aid of exogeneous insulin. Often before type 2 diabetes symptoms become noticeable insulin production is unimpaired, but the cells are resistant to the action of insulin (Peyrot et al., 1999), and the insulin resistance is usually a precursor to insulin secretory defects (Lillioja, Mott, & Spraul, 1993). Some individuals with type 2 diabetes do require insulin when either the pancreas wears out or they need assistance to better manage their blood glucose levels (Gohdes & Acton, 2000). Insulin is also needed temporarily for some individuals for glucose control in stress-induced hyperglycemia (Gohdes & Acton).

### Gestational Diabetes

Gestational diabetes develops while a woman is pregnant, but usually disappears when the pregnancy comes to term. Women with marked obesity, abnormal

glucose tolerance, a family history of diabetes, or who are members of an ethnic group with high prevalence rates of diabetes are all considered to have risk factors associated with gestational diabetes. It is recommended that pregnant women who meet any of these factors be screened and treated during their pregnancy (ADA, 2003a). Gestational diabetes is found in 2-5% of all pregnancies. Nearly 40% of women that experienced gestational diabetes eventually develop type 2 diabetes (Center for Disease Control and Prevention, 1997), which makes this a significant risk factor.

#### Impaired Fasting Glucose

When individuals have a fasting glucose level of 110-125 mg/dL, they are considered to be in the impaired glucose range. It is estimated that 7% of the U.S. population fall within this range (ADA, 1997). This range has been termed "pre-diabetes" by the ADA (2003a) due to increased risk for eventually developing type 2 diabetes.

#### Cost of Diabetes

Diabetes imposes a substantial cost burden to society and imposes a specific burden to those with the disease and their families. According to the ADA (2003b), the cost of diabetes in 2002 was estimated at \$132 billion in medical expenditures and lost productivity. This amount is significantly higher than the \$98 billion estimated cost of diabetes in 1997. Direct medical expenditures for the 12.2 million Americans with diabetes are more than double the expenditures for similar people who do not have diabetes. More than \$1 in every \$10 spent on health care components in the U.S. is attributable to diabetes. And \$1 of every \$5 spent on health care itself is for a person with diabetes. The greatest cost is for inpatient stays, followed by nursing home care, and physician office visits. Diabetes is responsible for 18% of home health care

expenditures, 14% of hospice care expenditures, and 15% of nursing home expenditures (ADA). These amounts verify the huge economic burden of diabetes, but they do not account for pain and suffering of people with diabetes or their families and friends. As of January, 2002, there were 122,000 people receiving disability benefits from social security who listed diabetes as the primary basis of disability, and 109,000 receiving disability who listed diabetes as the secondary basis of disability. Each case of permanent disability results in an average lost earnings of \$42,462 per year (ADA). Increasing the productivity in the U.S. economy and increasing the quality of life for people who suffer from diabetes and their families can be achieved by providing better access to preventative care, having more intensive disease management, more widespread diagnosis, and the advent of new medical technologies (ADA).

### Complications

Acute complications can arise from the extreme blood glucose fluctuations associated with diabetes. If blood sugar levels drop too low, symptoms of hypoglycemia can develop including nervousness, shaking, confusion, and impaired judgment (Bellenir, 1999). Hypoglycemic episodes have been reported to make individuals with diabetes feel vulnerable and out of control (Jacobson, 1996). When blood sugar levels become too high (hyperglycemia), during which mental status changes can be significant, a person can enter a diabetic coma, and finally death (Bellenir), a complication experienced much more often by individuals with type 1 than type 2 diabetes.

Typically, diabetes does not kill a person suffering from it, rather the complications of diabetes do. The National Diabetes Data Group (1995) found mortality rates to be twice as high among middle-aged individuals with diabetes when compared

to middle-aged individuals without diabetes. This group also noted that diabetes contributed to 187,000 deaths in 1995 and was the seventh leading cause of death listed on U.S. death certificates. In 2002, 186,000 deaths were attributed to diabetes. The Diabetes Control and Complications Trials research group found a direct relationship between the degree of hyperglycemia and the risk of microvascular complications (cited in Jacobson, 1996). It was demonstrated that complication progression was far more rapid in those individuals with poor glycemic control. Microvascular damage can result in renal failure, peripheral nerve damage, and blindness (Peyrot et al., 1999). These complications have been shown to lower the overall quality of life and create significantly higher levels of impairment (Smith, 2004).

Microalbuminuria is one test that is used to assess the level of damage being done to the kidneys (Indian Health Service, 1999). If the body does not produce sufficient insulin (type 1 diabetes), it may begin to utilize proteins and fat as a source of energy. This may produce large amounts of ketoacids that may lead to a diabetic coma, which may result in death (Goetsch & Wiebe, 1998). Another complication that is often overlooked is erectile dysfunction. It afflicts 50-75% of diabetic men and usually has an earlier onset than in the general population (Vinik & Richardson, 1998). De Berardis and associates (2002) conducted a study of 1,620 males with diabetes to assess the prevalence of self-reported erectile dysfunction and its impact on quality of life. They found that 34% reported frequent erectile dysfunction, and 24% reported occasional erectile dysfunction. These patients showed worse psychological adaptation to diabetes and a less satisfactory sexual life, as well as significantly higher levels of diabetes-specific health distress. Of those with erectile dysfunction, 50% regarded diabetes as having a great impact on their sexual life. They also showed higher levels of discouragement and frustration, had a lower acceptance of diabetes, worse metabolic

control, and higher levels of depressive symptomology. De Berardis and associates (2005) continued to track this group for 3 years, finding a significant reduction in quality of life scores preceded the development of erectile dysfunction. They attributed the lower quality of life scores to the higher rates and severity of diabetic complications. The authors suggested that sexual function should be considered an integral part of the overall health in diabetic patients.

The most common complication, however, is macrovascular disease. Individuals with diabetes mellitus have a two- to six-fold increased risk of myocardial infarction, stroke, congestive heart failure, and peripheral vascular disease (Gatchel & Oordt, 2003).

People with diabetes are at a greater risk of cardiovascular disease, peripheral vascular disease, renal disease, ophthalmic disease, endocrine/metabolic complications, and other chronic complications compared to those without diabetes (ADA, 2003a; Folsom, Chambless, Duncan, Gilbert, & Pankow, 2003). Of these complications, cardiovascular disease has the highest proportion of health care use attributable to diabetes.

In 2002, 183,000 deaths were attributable to diabetes. An estimated 19% of all deaths where cardiovascular disease was listed as the primary cause are attributable to diabetes; this accounts for 58% of all deaths from diabetes (ADA, 2003a). This supports the findings of DeStefano and Newman (1993), who found that coronary heart disease is the leading cause of death among those with diabetes, and those with diabetes had a 13 times greater risk of coronary heart disease compared to people without diabetes.



## Risk Factors

There are several demographic risk factors that have been associated with diabetes. In most populations, the base rate for diabetes is relatively low before the age of 30 years, with the rates increasing rapidly as people age (Rewers & Hamman, 1995). This tends to be more true for individuals in the majority population, with some minority groups developing diabetes at far younger ages. For example, the Pima Indians of the Southwestern U.S. have the same prevalence rate for diabetes at the ages of 25-29 years that non-Hispanic Whites experience in the 60-65 year age range (Rewers & Hamman). Obviously, ethnicity can be another significant risk factor. Generally, being a member of certain Native American tribes, or being of Hispanic or African American origin is considered a risk factor for developing type 2 diabetes after adjusting for age, gender, obesity, education, and income (ADA, 2004; Rewers & Hamman). Gender does not appear to be a risk factor for developing type 2 diabetes (Rewers & Hamman).

While we cannot control most of the demographic risk variables, other risk factors for diabetes can be controlled and are generally linked to lifestyle choices or variables that can be changed. These include obesity, diet, physical activity, alcohol use, and socioeconomic status (SES).

Type 2 diabetes accounts for 90-95% of all cases of diabetes, and 90% of those with type 2 diabetes are overweight. According to an overview of the literature on the treatment of diabetes, Fabricatore and Wadden (2003) explain that 64% of American adults are either overweight, having a body mass index (BMI) of 25.0-29.9 kg/m<sup>2</sup>, or are obese with a BMI of  $\geq 30$  kg/m<sup>2</sup>. These rates are double what they were in 1980. Longer duration of obesity increases the risk of developing type 2 diabetes (Rewers & Hamman, 1995). How individuals carry their body fat is considered a risk factor as well, with those

individuals who carry their weight in their upper body appearing more prone to develop diabetes (Rewers & Hamman). Diet and activity level are highly correlated with obesity, thus intervention typically involves targeting these areas. Significant improvements in glycemic control, blood pressure, and cholesterol levels have been associated with small weight losses of 10-15% (Rewers & Hamman). Researchers have also found that there is an excess accumulation of intracellular triglyceride in both liver and muscle in individuals with type 2 diabetes, which appears to predate the onset of the disease by several years (Taylor, 2004). It has been suggested that this is caused by mitochondrial dysfunction, which can be influenced by genetic, age and behavioral factors (Taylor). Finally, alcohol consumption has been suggested as a potential risk factor through a variety of pathways, including direct physiological changes to the liver and pancreas, lower adherence rates to treatment, and additional calories (Rewers & Hamman).

#### Treatment/Prevention

According to the ADA (2003a), the primary means of treating diabetes and slowing the onset of its complications is lifestyle modification. The primary goal in the management of diabetes is keeping blood glucose levels within the normal range as much of the time as possible (Bellenir, 1999). To do so, people with diabetes are encouraged to monitor their glucose levels several times a day, taking drops of blood from their finger tips and measuring glucose levels with portable electronic meters. The diabetic individual can then take the appropriate measures to control glucose levels. Diet and exercise are considered the foundation for treatment of type 2 diabetes (Gohdes & Acton, 2000). High levels of physical activity and low fat diets have been demonstrated to decrease insulin resistance and reduce the body's demand for insulin secretion. The

current U.S. lifestyle of low physical activity and high fat diets has the opposite effect (Gohdes & Acton).

Long-term treatment of obesity recognizes that obesity is a chronic condition similar to diabetes or hypertension and includes continued patient-provider contact, either in person or by mail. This type of treatment has been shown to be more successful than short-term treatment (Fabricatore & Wadden, 2003). Very low calorie diets have been shown ineffective in maintaining weight loss over time and are not recommended as a lifestyle intervention. Increased physical activity has been linked to maintaining weight loss. Those who exercise regularly are more successful than those who use diet alone. Lifestyle activity, for example, using the stairs rather than escalators or elevators and choosing a distant parking lot, has been shown to be more effective in maintaining weight loss than programmed or planned aerobic activity. Lifestyle activity may be a good alternative to programmed activity for those obese individuals who say they hate to exercise (Fabricatore & Wadden).

Several medications have been approved for treatment of obesity by the Food and Drug Administration (FDA). Sibutramine (Meridia), a combined serotonin-norepinephrine reuptake inhibitor, is associated with reports of increased satiation. When this drug was used with a low calorie diet, an initial weight loss was found to be higher than with a low calorie diet and placebo (7% vs. 2%; Fabricatore & Wadden, 2003). The drawback to using this drug is that it is not recommended for those taking antidepressant medication, or those with a history of coronary artery disease, arrhythmia, stroke, congestive heart failure, or a history of hypertension. Unfortunately, obese individuals are at a greater risk for these conditions. The second approved medication is orlistat (Xenical), which is a gastric lipase inhibitor that blocks the absorptions of about one third of the fat contained in a meal. Use of this drug decreases

calorie intake by 150-180 kcal per day. While using this drug patients are negatively reinforced to eat a low fat diet. If they consume more than 20 grams of fat per meal or more than 70 grams of fat per day, they suffer adverse gastrointestinal events (Fabricatore & Wadden).

Surgical interventions have proved successful for obese individuals (Sjostrom et al., 2004). Bariatric surgery produces average reductions of initial weight between 25-30%. This surgery significantly improves asthma, hypertension, sleep apnea, and diabetes (Kral, 1992). Patients treated with gastric bypass surgery maintain a 50% weight loss for as long as 14 years following the surgery. Bariatric surgery represents a potential cure for type 2 diabetes for patients who are extremely obese (Fabricatore & Wadden, 2003). Pories and Albrecht (2001) found that 14 years after undergoing gastric bypass surgery 82.9% of patients with diabetes and 98.7% of those with impaired glucose tolerance had maintained normal levels of blood glucose and hemoglobin A<sub>1c</sub> (H<sub>g</sub>A<sub>1c</sub>).

Tuomilehto and associates (2001) conducted a study in Finland that included 522 middle age (mean age 55) obese (mean BMI 31kg/m<sup>2</sup>) individuals who were given either brief diet and exercise counseling (control group) or intensive individualized instruction on food intake, weight reduction, and guidance on increasing physical activity (intervention group). After a 3.2 year follow-up, they found there was a 58% reduction in the incidence of diabetes in those who received the intensive instruction compared to the control group. A strong correlation was also found between the degree to which subjects were able to lose weight (5% reduction), reduce fat intake (10% of calories), increase fiber intake (.15g /1,000 kcal), and exercise (>150 min /week), and the ability to stop the progression to diabetes (Tuomilehto et al.).

The Diabetes Control and Complications Trial (DCCT; Diabetes Control and Complications Trial Research Team, 1993a, 1993b) demonstrated that the lower the average HgA<sub>1c</sub> levels were, the less likely a diabetic individual was to develop diabetic-related complications. The 3,234 enrolled subjects of the Diabetes Prevention Program, who had a mean age of 51 and a mean BMI of 34kg/m<sup>2</sup>, were randomized into three different intervention groups. The three groups were an intensive nutrition and exercise counseling group (lifestyle group), a biguanide metformin (Glucophage) group, or a placebo group. After a 2.8 year follow-up for the lifestyle group, there was found to be a 58% reduction in the progression to diabetes. There was also a 31% reduction in diabetes in the metformin group (Diabetes Prevention Research Group, 2002; The Diabetes Prevention Program, 1999; The Diabetes Prevention Program Research Group, 2000). This group (DCCT/EDIC, 2005) has also shown that intensive diabetes treatment lowers the risk of nonfatal myocardial infarction, stroke and death by cardiovascular disease by 57%.

According to the American Diabetes Association and National Institute of Diabetes (2002), type 2 diabetes can be prevented or delayed, and individuals who are at high risk of developing the disease can be easily identified. Prevention policies that focus on lifestyle modification, including weight loss and increased physical activity are the key to delay and prevention. Screening in health care settings to detect impaired fasting glucose and impaired glucose tolerance should be considered in those older than 45 years, and is strongly recommend for those over 45 years and overweight if they have another risk factor such as being an ethnicity other than Caucasian, if they have hypertension or dyslipidemia, or if they have a first- degree relative with diabetes or previous gestational diabetes. Those who are identified as being at-risk for developing diabetes should be given counseling on weight loss and instructed on increasing

physical activity. Follow-up counseling has been shown to be important for success, and monitoring for the development of diabetes should be conducted every 1-2 years (ADA and National Institute of Diabetes and Digestive and Kidney Diseases).

The ADA (2003a) has several recommendations for management of diabetes. They recommend that individuals receive care from a physician-coordinated team. This team should include physicians, dietitians, nurses, mental health professionals with special interest and expertise in the field of diabetes, and pharmacists. It is also essential that diabetic individuals play an active role in their treatment. Individual management plans should be formulated with all the above members taking into account the diabetic's age, work, or school schedule, eating patterns, physical activity, cultural factors, social situation and personality, presence of other medical conditions, and presence of other diabetes complications.

Individuals with diabetes may find facing the disease and its treatment daunting (Gatchel & Oordt, 2003; Jacobson, 1996). One factor that makes the diabetic regimen so challenging is that it is lifelong (Glasgow et al., 1995). Lifestyle changes are more difficult than most people tend to believe, one indicator being the number of people who choose invasive medical procedures rather than making a lifestyle change. For example, bariatric surgery is thought of by some as forced behavior modification (Van Hout, Van Oudensden, & Van Heck, 2004). Some patients request the surgery as a "mechanical" solution to their problem and believe that having surgery will make them feel less hungry, which will lower their need for will power when it comes to stopping eating. Particular to behavior change with diabetes, Rosenstock (1985) examined 60 diabetic patients and found that 60% did not administer their insulin correctly, 73% did not follow their prescribed diets, and 50% did not properly care for their feet. Only 7% of this group complied with all the steps of the diabetic regimen. Further, individuals experiencing

psychological stress often alter their behavior in ways that negatively affect glucose control (Jacobson).

### Psychosocial Variables

The idea that psychological variables can and do impact health is relatively well-accepted. Over the last three decades, epidemiological studies have examined multiple psychosocial variables and their impact on various diseases. Diabetes is no exception. McVeigh, Mostashari, and Thorpe (2005) examined individuals with "serious psychological distress" and diabetes in New York City. They found individuals with comorbid serious psychological distress and diabetes were more likely to be divorced, have low income, have poorer health care utilization, be less compliant with their medical regimen, and report poorer health outcome than individuals with only diabetes.

#### *Depression*

Depression appears to be the most widely studied psychosocial variable with regard to health. The World Health Organization (WHO) found major depression to be the fourth leading cause of worldwide disability in 1990, causing more disability than heart disease or stroke (Murray & Lopez, 1997).

Depression has been shown to be related to diabetes in many ways. The initial diagnosis of a chronic disease such as diabetes often leads to a sense of loss and time of bereavement, regardless of the age of onset (Jacobson, 1996). These feelings can potentially turn into a major depressive episode and interfere with an individual making appropriate changes in his or her life to manage diabetes. Depression can reduce an individual's ability to adhere to a regimen (Ciechanowski, Katon, & Russo; 2000, Lin et al., 2004; Park, Hong, Lee, Ha, & Sung, 2004). Further, individuals with poorer glycemic

control demonstrate a higher prevalence of depression and eating disorders and are more apt to live in families with less social support (Lustman, Griffith, Clouse, & Cryer, 1986). Leedom, Meehan, Procci, and Ziedler (1991) found that individuals diagnosed with diabetes were more likely to score in the clinical range on both the Zung Self-Rated Depression Scale and the Beck Depression Scale, than a demographically matched sample of people without diabetes.

Marcus, Wing, Guare, Blair, and Jawad (1992) examined the relationship between a history of major depression disorder and those individuals seeking an obesity intervention. In this study, a history of depression was not related to pretreatment glycemic control, but was associated with higher rates of attrition from the intervention. Egede, Zheng, and Simpson (2002) concluded that individuals with diabetes were more likely to develop depression than nondiabetic individuals, after examining outcomes of 825 adults with diabetes and 20,688 adults without diabetes using the 1996 Medical Expenditure Panel Survey. They also concluded that individuals with diabetes who were depressed had health care expenditures that were 4.5 times higher than individuals with diabetes who were not depressed.

Dalahanty, Meigs, Hayden, Williamson, and Nathan (2002), found no significant relation between anxiety scores or depression and baseline BMI. They did find, however, that there was significant interaction by ethnic group for the relation between BMI and anxiety, low fat diet self-efficacy, and weight loss self-efficacy, indicating that the correlations between BMI and these variables differed by ethnic group. Depression, anxiety, and perceived stress were positively correlated with each other. This study excluded those on greater than the minimal therapeutic dose of selective serotonin reuptake inhibitors and those with major psychiatric illness. The low levels of depression and anxiety in this group may explain why neither depression nor anxiety was correlated



with baseline BMI, as had been seen in other studies (Musante, Costanzo, & Friedman, 1998).

Eaton, Armenian, Gallo, Pratt, and Ford (1996) concluded that the presence of a major mood disorder substantially increased the risk of diabetes. In their 13-year prospective follow-up reports from a community-based study, major depression was associated with a 2.2 fold increase in the development of diabetes after they adjusted for other risk factors. No similar increase was found associated with milder forms of depressive symptomology.

In order to clarify the relationship between psychosocial factors and depression, Egede and Zheng (2003) examined a sample of 1,810 individuals over age 18 years from the 1999 National Health Interview Survey (NHIS) with diabetes, while controlling for multiple confounding factors. Among individuals with diabetes, they found those who were younger, poorer, unmarried, and female, and who reported worsening health status and had diabetes for less than 5 years, were more likely to have major depressive disorder. These individuals were also more likely to have major complications, to be smokers, and to have visits to emergency rooms, psychiatrists and/or mental health professionals than those with diabetes who did not have major depressive disorder.

Less than 30% of depressed individuals, regardless of diabetes status, reported visiting a psychiatrist. It is likely that primary care providers treated more patients with depression, and that the stigma of seeing a mental health professional played a role in decreased visits to a psychiatrist (Egede & Zheng, 2003).

After controlling for potential confounds, psychosocial factors such as perceived health status, income, and education remained independently associated with depression in individuals with diabetes. Perceived worsening of health status was independently associated with depression. Longer duration of disease, using insulin or

medications, and having major complications were not associated with depression (Egede & Zheng, 2003). This suggested that psychosocial factors, perceptions about the effect of diabetes on overall health, illness, severity, or type of treatment were likely to play a role in the etiology of depression in those with diabetes. In 2005, Egede, Nietert, and Zheng reported that the coexistence of depression and diabetes increased the risk of mortality from all causes, compared to having either depression or diabetes alone.

Nichols and Brown (2003) examined 16,180 full-year health maintenance organization group members in 1999 who had been diagnosed with type 2 diabetes, and 16,180 comparison members, who were matched for age and sex, and did not have diabetes. They found depression was more common in individuals with type 2 diabetes than among matched control subjects. Women were twice as likely as men to have depression; for those women, body weight was a stronger predictor of depression than diabetes; for men, cardiovascular disease was the strongest predictor of depression. Subjects with diabetes had 1.5 times greater prevalence of depression than the control subjects. Diabetic individuals were nearly 50% more likely to have diagnosed depression than nondiabetic individuals. This study did not use self-report measures to diagnose depression in the subjects in this study. The data came from clinicians, who had charted them as depressed after seeing them in an ambulatory visit or by evidence of antidepressant drug therapy. The authors of this study concluded that the relationship between diabetes and depression may be bidirectional.

Kawakami, Takatsuka, Shimizue, and Ishibashi (1999) followed 2,380 Japanese men for 8 years who worked for an electrical company in Japan. They completed a self-administered questionnaire including the Zung Self-Rating Depression Scale. At the follow-up survey, the occurrence of diabetes was determined by WHO criteria. The authors found that depressive symptoms may be associated with the onset of type 2

diabetes. This study indicated that moderate or severe levels of depressive symptoms were associated with later occurrence of type 2 diabetes. This study also demonstrated that the association between depressive symptoms and the occurrence of type 2 diabetes was independent of smoking, obesity, drinking, chronic medical conditions at baseline, family history of diabetes, and leisure time physical activity.

Illness intrusiveness, as discussed by Talbot, Nouwen, Girgas, Belanger, and Audet (1999), is described as the disruptions of valued activities and interests as a result of restraints imposed by an illness. Talbot and colleagues found that illness intrusiveness explained over half of the variance in depressive symptomology among their diabetic participants. They also found individuals who had significant psychosocial stressors in their lives seemed to have a more difficult time coping with their illness. It has been suggested that there is a direct link between painful diabetic neuropathy and depression. Pain is often a component with peripheral neuropathy and tricyclic antidepressants have been shown to be useful in treating this syndrome (Geringer, 1990).

Talbot and Nouwen (2000) conducted an extensive literature review in order to determine the relationship between depression and diabetes. They looked at depression as a result of biochemical factors and found that the development of major depressive disorder preceded the diagnosis of type 2 diabetes and that major depressive disorder and depressive symptomology may increase the risk of developing type 2 diabetes. As for type 1 diabetes, major depressive disorder usually followed the diagnosis. Their review found that the initial onset of major depressive disorder in individuals with type 1 diabetes was 22.1 years, which was lower than that of the general population, but later than the age at which type 1 diabetes was generally diagnosed (late childhood to early adolescence). They also found evidence that in individuals with diabetes, blood glucose

levels improved with remission of depression. In both type 1 and type 2 diabetic individuals, hyperglycemia resolved with the treatment of clinical depression (Talbot & Nouwen). Their review also showed that major depressive disorder increased the risk of complications in diabetes, not that diabetes increased the risk of major depressive disorder. Another hypothesis Talbot and Nouwen studied was whether depression was the result of the psychological demand imposed by diabetes. Individuals with diabetes may develop depression due to the increased strain of having a chronic medical condition rather than from the disease itself. Depressed mood is related to difficulties in adapting to diabetic complications. The authors found that negative mood and disease variables were mediated by illness intrusiveness (Talbot & Nouwen). For those with three or more complications from diabetes, the risk of depressive symptomology was greater than for those with fewer complications. Higher levels of social support also helped reduce depressive symptomology. Individuals with poor health status had a limited ability to develop and maintain social relations, which contributed to emotional distress. The diabetic's coping skills also influenced depressive symptomology (Talbot & Nouwen). Those who might feel overwhelmed by their disease were more likely to use passive coping skills such as wishful thinking and avoidant behaviors, and have poorer psychological adjustment. Some individuals with diabetes may reach learned helplessness, and subsequent depression, while trying to control their diabetes, because strict adherence does not guarantee avoidance of complications or glucose control. Lower levels of adherence and more demanding regimens were associated with depressive symptomology (Talbot & Nouwen).

Lustman and associates (2000) conducted a meta-analysis of 24 studies that measured the association of depression with glycemic control. They concluded that subclinical and clinical expressions of depression were found in >25% of patients with

type 1 or type 2 diabetes. Depression had adverse effects on functioning and quality of life. Depression was associated with hyperglycemia in patients with type 1 and 2 diabetes. They indicated that they could not determine the direction of the interaction, indicating that hyperglycemia and depression may impact each other differently, depending on individuals and time (Lustman et al.).

DiMatteo, Lepper, and Croghan (2000) conducted a meta-analysis of the effects of anxiety and depression on patient adherence, which included 12 articles on depression and 13 on anxiety, and found the association between anxiety and adherence was small and nonsignificant. The association between depression and noncompliance, however, had an odds ratio of 3.03 (95% confidence interval, 1.96-4.89) that was substantial and significant. The odds that depressed patients would be noncompliant with medical treatment recommendations were three times higher than in patients without depression (DiMatteo et al.). Depression was associated with severe limitations of daily functioning as well as with high rates of health care utilization. Mood disorders such as anxiety and depression that impair cognitive focus, energy and motivation might affect patients' willingness and ability to follow through with medical treatment. Nearly half of all medical patients in the U.S. do not follow the recommendations of their physicians. Among every 100 noncompliant patients, 63.5 can be expected to be depressed. The authors concluded that a "feedback loop" existed in that depression caused noncompliance and noncompliance further exacerbated depression, and also that poor health caused both depression and noncompliance (DiMatteo et al.).

Anderson, Freedland, Clouse, and Lustman (2001) conducted a meta-analysis of 42 studies and found that the presence of diabetes doubled the odds of comorbid depression. Compared to control subjects, the odds of depression were twice as high for

those with diabetes. Major depressive disorder was present in 11.4% of patients with diabetes. Elevations in depressive symptoms were present in 31% of patients with diabetes. Lifetime prevalence of depression was higher in those with diabetes compared to control groups. They also found the odds of depression were higher in women than men (Anderson et al.), which is consistent with U.S. prevalence rates for major depressive disorder. As many as one in three individuals with diabetes has depression at a level that interferes with adherence to medical treatment, quality of life, functioning, glycemic control, and increases the risk of diabetes complications.

A review conducted by Fabricatore and Wadden (2003) did not support the assumption that obesity is related to depression and other psychopathology. Depression in obese people should not be attributed to weight alone. Although losing weight does improve mood it does not necessarily improve depressive symptomology. Treatment for mood disorders should be concluded before a weight loss regimen is introduced. This should lower the patients' psychological distress and improve their ability to adhere to a weight loss regime. Obesity has, however, been associated with unfavorable characteristics. Those who are obese are discriminated against and others show prejudice towards them. Prejudice and discrimination can be seen as chronic stressors that can have deleterious effects on the obese individual's emotional well-being (Fabricatore & Wadden).

In a study of 187 Latino and European American participants and their partners aged 25-62, who had been diagnosed with diabetes for 1-9 years and who had been cohabitating with their partners for a minimum of 3 years, Fisher, Chesla, Skaff, Mullan, and Kanter (2002) found that levels of depressive affect and anxiety and rates of depression were as high for partners of those with diabetes as they were for the patients themselves. Most of the variance was accounted for by family level variables, such as

life stressors, not just disease. Marital discord, conflict, and hostility are positively correlated with depressed affect, which, in turn, decreases disease-related problem solving and marital satisfaction. Negative affect, mostly criticalness, among partners of patients with depression or chronic illness can be linked to patients not following medical regimens and may lead to complications or patient relapse. Negative affect among partners can affect disease progression and management. This study demonstrated that effects of chronic disease were not limited to the patient alone, and that the assessment of family and couple relationship issues should be addressed as part of diabetes care. In order to reduce distress and improve disease-management, interventions should include helping couples resolve diabetes-related conflicts, enhance spousal collaboration, support, and closeness, as well as joint problem-solving (Fisher et al.). Another interesting finding of this study was that female partners demonstrated a higher level of psychological distress than male partners.

Not all of the studies reviewed suggested a relationship between diabetes and depression. Researchers in Norway (Engum, Mykletun, Midthjess, Holen, & Dahl, 2005) examined 60,869 individuals with and without diabetes. They found that those individuals with type 2 diabetes had the same odds of developing depression as individuals without diabetes. They did find those individuals with type 2 diabetes and comorbid chronic somatic were at higher risk. They also concluded that hyperglycemia was not associated with depression and either type 1 or 2 diabetes. Another study (Trief et al., 2006) found that the presence of depression did not predict glycemic control.

Turk and Rudy (1985) reported a relationship between diabetic perception of diabetes as being disabling and dysphoric mood. In a review of the depression literature, Geringer (1990) found that individuals with diabetes tended to have higher rates of depression than both the general population and patients with other medical illnesses.

The prevalence of depression is increased in adults with type 1 diabetes (Gavard, Lustman, & Clouse, 1993).

Glycosylated hemoglobin (H<sub>gA</sub><sub>1c</sub>) is considered to be one of the best measures of long-term glycemic control. Glucose attaches itself to circulating hemoglobin molecules within the red blood cell. The rate that glucose attaches itself, or glycation, is related to the glucose levels in the body. Lustman, Harper, Griffith, and Clouse (1986) found that depressed patients show significantly increased levels of glycated hemoglobin. In the same study, the researchers found the duration the individual had been experiencing the depressive symptoms was related to the severity of the increase in H<sub>gA</sub><sub>1c</sub> levels. Hoey and associates (2002) examined the impact of levels of H<sub>gA</sub><sub>1c</sub> on individuals diagnosed with diabetes and their quality of life. Their study concluded that lower H<sub>gA</sub><sub>1c</sub> was significantly associated with better reported quality of life as measured by the Diabetes Quality of Life questionnaire. Lawrence and associates (2006) also found no significant relationship between depressive symptoms and H<sub>gA</sub><sub>1c</sub>.

It has long been speculated that psychopathology, specifically depression, is related to diabetes onset and the course the disease will take. There have been numerous studies conducted to document this speculation. The relationship between depression and diabetes could arise in several ways. There could be a common neuroendocrine basis that underlies both disorders. Causality could either be from depression to diabetes or from diabetes to depression or coincident with both (Barlow, Hatcher, Edidin, & Sload-Rossite, 1984). It may be that a depressive episode produces a bodily change that leaves the individual at a higher risk for diabetes (Herbert & Cohen, 1993). The stress of having diabetes, as well as for other severe medical conditions, may cause a psychological reaction due to the threat of loss of life (Jacobson, 1993). Finally, depression may be related to a number of confounding factors that include, but



are not limited to, obesity, exercise, lifestyle, and even use of medication, which may all increase the risk of diabetes and for poor control after onset of the disease (Eaton, Mengel, Larson, Campbell, & Monague, 1992; Goldney, Phillips, Fisher, & Wilson, 2004, Ludman et al., 2004). It should be noted, however, that not all reviewed studies suggest this relationship between depression and diabetes (Engum et al., 2005; Trief et al., 2006).

### *Social Support, Stress, Anger, and Anxiety*

Not all psychological variables that influence health are considered a disease or disability such as depression. It has long been recognized that other internal and external factors such as how we perceive the amount and quality of support we receive from those around us, how efficiently we handle day-to-day stressors, or how we handle perceived threats all impact our health.

Although social support can facilitate diabetes management, it also may have a "dark side" in some cases. Nouwen, Gingras, Talbot, and Bouchard (1997) developed a psychosocial classification system in which a spousal over-involvement group was identified. Individuals in this category tended to perceive a loss of self-efficacy, which could lead to nonadherence. Another study found that individuals with diabetes did not benefit from emotional support when they perceived that they did not need it. Too much involvement may cause feelings of helplessness and dependency, lowering the diabetic's mental health quality of life (Penninx et al., 1998).

Bjorntorp (1997) formulated a theory that activation of the hypothalamic-pituitary-adrenal axis can come about from perceived psychological stress when the person has a helplessness or defeatist reaction to the perceived stress. This can result in several endocrine system abnormalities, including low sex steroid levels and high cortisol levels

that provoke the actions of insulin in the body. This hormonal imbalance causes visceral adiposity, which contributes to the development of insulin resistance. Animal studies have shown that stressful situations can induce hyperglycemia (Surwit, Schneider, & Feinglos, 1992).

Studies have also shown that in individuals with diabetes, psychological stress can induce hyperglycemia (Wales, 1995). Based on this theory Mooy, de Vries, Grootenhuis, Bouter, and Heine (2000) conducted a study to see if psychological stress could be one of the factors related to the development of type 2 diabetes. They used a cross-sectional study of 2,380 people. One hundred eighteen participants were excluded due to missing information, leaving 2,262 individuals without a history of diabetes. Individuals were tested to determine whether chronic psychological stress was positively associated with the incidence of type 2 diabetes, and to see if the relationship between diabetes and stress was mediated by visceral adiposity. They found the more stressful events that were reported within the last 5 years, the higher the incidence of previously undetected diabetes (Mooy et al.).

Stress can impact glycemic control by increasing liver production of glucose in response to the release of stress hormones such as epinephrine and cortisol (Goetsch & Wiebe, 1998). Major life events, both good and bad, that produce stress have been found to result in poorer control of glucose levels (Schwartz, Springer, Flaherty, & Kiana, 1986). Herschbach and associates (1997) found that individuals with diabetes with more long-term complications reported higher levels of stress than individuals with diabetes without as severe complications. Diabetes control can be affected by very small variations in stress or self-management behavior.

Physical and/or psychological stress elicits a stereotyped "fight or flight" reaction. These reactions involve the activation of the autonomic nervous system and the release

of counter-regulatory "stress-hormones," namely cortisol, epinephrine, growth hormone, and glucagons that increase glucose levels and affect glucose homeostasis (Skosnik, Chatterton, Swisher, & Park, 2000). It has been demonstrated that acute and chronic psychological stress results in a deterioration of metabolic control (Violettes et al., 1992). Patients have reported new diabetes onset after an extremely stressful situation.

Consequently, psychological stress may play a role in the development of diabetes. In order to better evaluate the relation between glucose control and psychological stress by examining the effect of acute severe psychotic stress reaction on insulin sensitivity and beta-cell function in individuals without diabetes, Shiloah and associates (2003) examined 39 nondiabetic adults who were admitted through the psychiatric emergency ward with acute psychotic stress. Patients with depressive disorders, presence of any endocrine or concomitant acute disease, those on medications that could affect insulin secretion or activity or cause hyperglycemia, those with increased fasting glucose, and those who could not understand or sign an informed consent form were excluded. The study found that stress had an adverse effect of both glucose and insulin levels as well as  $\beta$ -Cell function. The findings showed mixed results for insulin sensitivity depending on the overall stress level upon admission. Those individuals with extreme stress behave differently than those with lower stress levels. For those individuals with this highest level of reported stress, insulin sensitivity was low and glucose and insulin levels were higher. But the overall mean of insulin sensitivity for the entire group was directly correlated with stress, showing increased insulin sensitivity with increased stress, so long as the stress did not reach a critical level (Shiloah et al.).

Expression of anger and the personality trait of "cynical hostility" have been associated with cardiovascular and cerebrovascular disease outcomes and have been demonstrated as a risk factor for diabetes (Clay, 2001; Eaker, Sullivan, Kelly-Hayes,

D'Agostino, & Benjamin, 2004; Everson et al., 1999). Impatience and anger were also associated with negative health outcomes by Booth-Kewley and Friedman (1987). Golden and associates (2006) found that those individuals who were in the top tertile or trait anger temperament scores had a 34% increased risk for developing type 2 diabetes. But this finding was nonsignificant once BMI and waist-to-hip ratio were controlled. Surwit and associates (2002) examined the impact of hostility using the Cook-Medley hostility scale on fasting glucose levels among healthy African American and Caucasian individuals. In both ethnic groups, hostility appeared to impact fasting glucose, but through different means. Among the Caucasian group, the relationship of hostility to insulin sensitivity was partially dependent on BMI, while such a relationship was not found among the African American group. Psychosocial variables can affect metabolic control directly through psychophysiological processes, and, indirectly, by disrupting regimen adherence (Peyrot et al., 1999).

In a study of 105 adult men and women with type 2 diabetes in a year-long clinical trial on the effects of stress management on glycemic control, Lane and associates (2000) found common personality traits may help to explain variations in glycemic control in these patients. Higher levels of blood glucose (poorer glycemic control) were, surprisingly, associated with lower scores of neuroticism and other personality facets of anxiety, depression, self-consciousness, anger, hostility, and vulnerability. Higher altruism scores were also linked to poorer glycemic control. People with higher neuroticism scores were more prone to experience negative emotions. These emotions included greater tendency to worry, experience guilt, anger, frustration, sadness, and hopelessness. They felt self-conscious and less able to deal with stress. Those with low neuroticism scores had been described as relaxed and even-tempered and were able to face stressful situations without becoming upset or rattled. Those who

had the tendency to worry and experience negative emotions may have had increased motivation to follow a necessary self-care regimen and, therefore, had a better clinical outcome. Patients who had low levels of neuroticism may have lacked the emotional distress that was necessary as a motivator to maintain the proper self-care. They may have minimized the long-term consequences of not maintaining control and the more severe health impact that diabetes will have on their future well-being (Lane et al.). People who have high scores of altruism have an active concern for others and are willing to assist others in need. People who are more concerned with the welfare of others than their own needs may tend to neglect their own self-care in order to assist others' interests and needs. Proper diabetes management may require a degree of self-centeredness to overcome the barriers of regimen adherence. Results of this study suggested that personality assessment techniques may help identify individuals who are at risk for poor glycemic control and that a little worry and self-centeredness may benefit those with diabetes (Lane et al.).

Those individuals with type 1 diabetes have been shown to be more sensitive to psychosocial variables compared to individuals with type 2 diabetes. In type 2 diabetes, insulin is usually still being produced and can respond to short-term fluctuations in glucose levels. Those with type 1 diabetes, on the other hand, are completely dependent on external insulin and their bodies are unable to compensate (Peyrot et al., 1999). Self-management behavior is also linked to stress and other psychosocial variables (Peyrot et al.). Some psychosocial variables such as depression or cynical hostility may have more of a long-term impact on glucose control due to their chronicity.

It has been demonstrated that psychosocial variables, ranging from psychopathology such as depression and anxiety to personality traits such as cynical hostility impact perceived and actual health, as well as disease outcome. Unfortunately,

medical providers often concentrate on the specific disease and miss these variables (Pouwer, Beekman, Lubach, & Snoek, 2006).

### Diabetes and Native Americans

It has been suggested that the epidemic of diabetes facing Native Americans is the result of an interaction between genetics and environment (Gohdes, 1986). The rate of diabetes has been shown to be related to the degree of native heritage one possesses (Lee et al., 1995), and many Native Americans are faced with risk factors such as poverty, poor education, unemployment, acculturation, and a loss of traditional Indian activities (Nelson & Manson, 2000).

Over the last 40 years, Native Americans have been confronted with growing rates of diabetes that have reached epidemic dimensions. Historically, diabetes has not been a problem faced by Native Americans. In 1928, the Prudential Insurance Company published an article indicating that diabetes was thought to be rare in Native Americans (cited in West, 1974). Review of medical reports of physicians serving Native Americans in Oklahoma between 1832 and 1939 does not indicate any prevalence of diabetes (West). The rarity of diabetes prior to 1940 was described among the Plains Indians, Eskimos, and Polynesian peoples (West). Cases of diabetes began to show up in the 1940s among Native people. By 1954, diabetes started to appear moderately among the Pima Indians. At the same time, diabetes was still fairly rare among the Ute and Apache tribes (West). In 1964, West estimated that 25% of Cherokee Indians in North Carolina over the age of 30 had diabetes. Data collected by Indian Health Services (IHS) showed that between 1972-1974, there were 104 diabetes-related deaths per 100,000 among Native Americans in Oklahoma; however, not all Native American tribes were affected to the same extent. For instance, during this same 3-year period, there was not a single

diabetes death among Alaska Indians or Eskimos. Investigation of total visits to IHS medical facilities showed that over one tenth of all visits were diabetes related. In the 45-65 years age group, 60% of visits were diabetes related in 1975. The Aberdeen IHS region, which primarily serves the Lakota and Dakota Sioux Tribes, found that visits for diabetes-related problems were also very high during this period. The number of outpatient diabetes-related visits in IHS facilities rose from 58,901 in 1971 to 156,213 in 1983 (Gohdes, 1986). Deaths related directly to diabetes mellitus among the Cheyenne River Sioux Tribe of South Dakota were 46.1 per 100,000 during 1990-1993. This was higher than the average diabetes-related death rate of the IHS regions as a whole. During the same period, the diabetes-related death rate for all ethnic groups in the U.S. was 11.8 per 100,000 (Huffstetter, 1998).

Acton and associates (2002) conducted a study to determine trends in diabetes prevalence among young American Indians and Alaskan natives. They analyzed IHS outpatient data from 84% of the IHS population for each year from October 1, 1990 to September 30, 1998. Participants were grouped by age as less than 15 years, 15-19, 20-24, and 25-34 years. For all age groups, they found the number of those with diagnosed diabetes who used IHS facilities increased by 71% (from 4,534 to 7,736 persons). The crude prevalence of diabetes increased by 46% (6.4 per 1,000 to 9.3 per 1,000). For those age 15-19 years prevalence of diabetes increased by 68% (3.2 per 1,000 to 5.4 per 1,000). For those age 20-24 years prevalence of diabetes increased by 47% (7.8 per 1,000 to 11.5 per 1,000). For those age 25-34 years diabetes prevalence rose 50% (18.0 per 1,000 to 26.9 per 1,000). For those age 15 years or younger, prevalence did not change (1.2 per 1,000). Among American Indians and Alaskan natives who were younger than 35 years of age prevalence of diabetes rose 46%. Prevalence of diabetes in the U.S. general population who were younger than 45 years

of age only rose 14% from 1990 to 1996 (Acton et al.). According to the National Diabetes Information Clearinghouse (2007), 15.1% of all Alaska Natives and American Indians aged 20 years or older are receiving some type of care for diabetes.

There are several factors that have been suggested as potential causes for the higher prevalence rates of diabetes among Native Americans. Type 1 diabetes remains extremely rare among Native Americans. Almost all cases of diabetes mellitus among Native Americans are type 2. One factor is obesity, which has been known to be a risk factor for developing type 2 diabetes for over 200 years. In 1915, a detailed medical description written on the Southwestern Indians stated that "pathological obesity does not exist." Early photographs taken of different Native American groups also indicated a low prevalence of obesity (West, 1974). This is not true today. The Sioux were nomadic hunters that followed the buffalo before the western U.S. was settled. All food came from either wild game or plant foods native to the plains region (Lang, 1985). When the tribes were confined to reservations, they became dependent on government rations that consisted of green coffee, dried tea, sugar, flour, salt pork, and beef. This demonstrated a shift from a diet high in fiber and unrefined carbohydrates to a diet high in refined carbohydrates and sugar and low in fiber, which was accompanied by increased calorie consumption. In 1985, government "commodity" foods were consumed in 85-90% of the households on one Sioux reservation. Commodities included canned meat, vegetables, fruits packed in syrup, macaroni, rice, vegetable shortening, flour, sugar, and peanut butter (Lang). Healthy foods were often considered not only to be expensive but were also unfamiliar and undesirable. Medical personnel have found the typical Sioux Indian's diet to be high in protein, fat, and carbohydrates, with an underrepresentation of vegetables and fresh fruits. While many individuals from various tribes have attributed diabetes to "White man's food," it is ironic, but not surprising, to find a reluctance to



change current eating habits to conform to a diabetic regimen. Lang found in a Dakota (Sioux) diabetic population that most individuals had received diabetic and dietary education and could repeat it back, in detail, to the interviewer. Yet, most openly stated that they did not follow the prescribed diet. This was consistent with the mainstream population of individuals with diabetes who found changes in lifestyle difficult. Further, the activity level of many Native Americans has been thought to be far less than that of their ancestors, which could also contribute to the current high levels of obesity. A protective effect of lifestyle was found when Ravussin, Vallencia, and Esparanza (1994) examined the differences in Pima Indians who lived a lifestyle that was more traditional versus those who lived a lifestyle closer to the mainstream population.

There seems to be a strong genetic predisposition for type 2 diabetes. Studies show concordance rates as high as 90-100% between monozygotic twins developing diabetes (Goetsch & Wiebe, 1998). Native Americans are the only minority group that has to prove tribal membership (minority status) by their blood degree. Without a documented blood degree, an individual cannot be a tribal member or receive the services they are entitled to by treaty with the U.S. government. This documentation of blood degree has allowed researchers to look at the prevalence rates of individuals with different percentages of Native American ancestry. One theory that has been proposed to explain the high prevalence of diabetes among Native Americans is the "thrifty gene" (Neel, 1962). Under this theory, Native Americans once survived periods of feast or famine, during which the body would store energy during good times for the bad. So in a sense, a Native American's metabolism is not equipped to deal with our current diet of excess. While this theory appears to have face validity, no specific genotype has yet been identified (Gohdes & Acton, 2000). Another study that sampled an adult Cherokee population in North Carolina found an increased prevalence rate of diabetes among

individuals who had a Cherokee blood degree of 50% or more compared to other tribal members of a lesser blood degree. Another study by Lee and associates (1995) also found that the prevalence rate of diabetes significantly increased when participants had a blood degree of 50% or higher in tribes of three different states. Further, among individuals in the Fort Berthold IHS region (North Dakota), those with less than a 50% Native American blood degree had the same prevalence of diabetes as the general U.S. population (Brosseau, Eelkema, Crawford, & Abe, 1979). Similar findings have been found among the Pima and Papago Indians (Brosseau et al.; Knowler, Williams, Pettit, & Stienberg, 1988). These findings suggested a strong hereditary tendency among certain Native American groups for type 2 diabetes.

Adherence barriers have been found among the Sioux that can significantly decrease diabetic adherence. One of the first is obtaining adequate medical care. While many Native Americans have access to free health care, the health care at times may be limited by funding considerations. The physician-patient relationship is also significant when discussing Native American populations. Depending on the individual and the medical doctor, this relationship can vary in effectiveness. Many Native Americans do not feel as if they have a choice in terms of selecting their doctor, because they have to take whoever is available at the IHS facility in order to receive free medical attention. If the doctor is not sensitive to cultural issues, the Native patients may fail to seek medical attention. Individuals of full Native American heritage have been found to make fewer clinical visits than Native Americans of mixed blood or the general U.S. population (Brosseau et al., 1979). Another factor is the load placed on the resources of tribal clinics. It is not uncommon for individuals to wait 8 hours to be seen at a clinic, sometimes having to come back the next day. Men, who tend to not go to the clinics as frequently as women, often give up their place in line in order for women and children to

be seen first. Sioux patients frequently do not ask medical personnel many questions because it is considered disrespectful to do so. This, at times, may lead to misunderstandings, resulting in noncompliance with treatment. The Sioux separate "traditional" and "modern" medicine. While diabetes is often considered to be a "White man's disease" and needs modern medicine, the use of traditional medicine is often preferred over modern medical interventions. Disease is often thought to be a side effect of disharmony among all things, including the body, mind, and spirit. Some medicine men among the Sioux have claimed to be able to treat "sugar," or type 2 diabetes, but have made discontinuing "White medicine" a prerequisite for treatment (Lang, 1985).

Another cultural factor that can influence compliance is that many Indian cultures look at the present and not the future. The issues of today are dealt with first and if these issues continue, little attention is paid to future consequences. This type of cultural feature does not work well with a diabetic regimen when short-term rewards are few and far between. Socioeconomic factors must also be taken into account when treating Sioux patients with diabetes. Some Indian reservations have as high as a 90% unemployment. In 1990, 59.7% of all individuals living on the Cheyenne River Sioux Reservation were living below the poverty line. South Dakota had a statewide unemployment rate of 4.35%, while the Cheyenne River Sioux Reservation had an unemployment rate of 27.9%. During the same period on the same reservation, 24% of Sioux households did not have a vehicle, 44% did not have a telephone, and 7% did not have indoor plumbing (Huffstetter, 1998). These factors, combined with the fact that many members live up to 90 miles from an IHS medical facility, make it difficult to keep follow-up appointments or even return phone calls.

Depression is another adherence barrier that Native Americans face. Native Americans as a group have some of the highest incidences of both suicide attempts and

completions (Huttlinger, 1995). Between 1990 and 1992, deaths due to suicide were 11.4 per 100,000 in the U.S. population, while the rate was 16.2 per 100,000 across all IHS facilities. During the same period, there were 45 suicide-related deaths per 100,000 on the Cheyenne River Sioux Reservation. Alcoholism, which can impair an individual's judgment concerning diabetes care (Zielke, 1999) and create other medical problems, is quite common on certain Indian reservations. In all U.S. races, it has been calculated that there are 6.8 cases per 100,000 of severe alcoholism versus 56.5 per 100,000 on the Cheyenne River Sioux Reservation (Huffstetter, 1998). Depending on how a researcher defines "alcoholism," these rates could be substantially higher.

Certain beliefs about health and body weight may also influence adherence. On the Cheyenne River Sioux Reservation, many elders talk about tuberculosis (TB) and the devastating effect it had on the Sioux population. It was noted that those who were thin seemed to be more susceptible to developing and dying from TB. Losing weight has been associated with sickness. One study looking at Sioux people with diabetes found that 12 out of 19 individuals who received recommendations to lose weight disagreed with the doctor's recommendation, stating their weight was "alright" (Lang, 1985). It has been recently found the diabetes and end stage renal disease (ESRD) put Native Americans on the Pine Ridge Sioux Reservation at a higher risk for developing TB. The rate of TB on this reservation is nine times that of the U.S. population (Mori, Leonardson, & Welty, 1992), ESRD disease, which can be caused by diabetes, has steadily increased on Sioux reservations over the last 20 years. The number of Native Americans with ESRD as a result of diabetes has been found to be 5.8 times higher than among Caucasians (Newman, Marfin, Eggers, & Helgerson, 1990).

IHS has recognized diabetes as a major health problem for Native people and has made efforts to initiate diabetic education and treatment programs on reservations

across the U.S. (Stracqualursi, Rith-Najarian, Hosey, & Lundgren, 1993). These programs have been modeled after guidelines set forth by WHO, which includes three levels of treatment and prevention. The primary stage includes increasing fitness and decreasing obesity within a community. These programs have been in place for several years, but how successful they have been has not been determined. Secondary prevention includes screening members in the community for undiagnosed diabetes and preventing the development of complications in identified diabetic patients. Tertiary prevention attempts to lower the rates of mortality of those individuals with complications. These different levels of treatment and prevention are still ongoing and under refinement. It is unclear what degree of impact they will have in lowering the prevalence of diabetic complications and mortality (Gohdes, Schraer & Rith-Najarian, 1996). Despite these efforts, Native Americans are 2.2 times more likely to have diabetes than non-Hispanic whites (National Diabetes Information Clearinghouse, 2007).

In a population-based cross-sectional study of 206 participants from eight Native American communities in New Mexico, Carter, Gilliland, Perez, Skipper, and Gilliland, (2000) found HgA<sub>1c</sub> levels were highest in the youngest age group studied. This may have been due to the young people in the study having the highest degree of obesity and the highest insulin resistance. Young people who have diabetes may not get treated as vigorously as older people. Survival bias may also explain this, which means that those older people with higher HgA<sub>1c</sub> levels may have died at higher rates. This may also be due to a cohort effect, with the younger cohort having higher levels of HgA<sub>1c</sub>. HgA<sub>1c</sub> levels were highest for those who consumed the most fat and sugar. Those participants who were treated with insulin also had the highest HgA<sub>1c</sub> levels. This suggested that insulin treatment was not producing glycemic control that is tight enough to lower the HgA<sub>1c</sub> levels. The authors concluded that encouraging traditional lifestyles and less high

caloric foods would be beneficial. Also, reducing the risk from in utero exposure to diabetes must have a high priority in this population (Carter et al.).

The Strong Heart Study is one of the largest and longest standing surveillance studies to be conducted among Native Americans (see Methods section for details.) Lee and associates (2002) conducted a study of 3,638 of the original 4,549 Strong Heart Study participants aged 45-74 years from Arizona, Oklahoma, and South and North Dakota, observing them 4 years after baseline examination to estimate incidence rates of diabetes and associated risk factors among these participants. They found that of the original cohort, 413 participants had died during the follow-up period. Those who had died had a higher mean age, higher average degree of Indian blood (93.3% vs. 87.7%), higher rate of albuminuria (51% vs. 29%), higher average fasting plasma glucose (164.7 vs. 151.3 mg/dl), but lower BMI (29.9 vs. 31.1 kg/m<sup>2</sup>) at baseline examination than those who participated in the second evaluation. A total of 1,664 participants were free of diabetes at baseline and were reexamined. During the 4-year follow-up period, 326 (19.6) developed diabetes. A total of 1,132 participants had normal glucose tolerance at baseline; of these 128 (11.3%) developed diabetes; 37% of those who had impaired glucose tolerance at baseline developed diabetes; and, 27.6% of them reverted to normal glucose tolerance. Of those who had diabetes at baseline but were not taking any medication, 2.9% reverted to impaired glucose tolerance, and 2% reverted to normal glucose tolerance. No substantial changes were found in BMI, percentage of body fat, or waist-to-hip ratio in these participants (Lee et al.). Overall, the 4-year incidence rate was 19.7%. Nine percent of men who had normal glucose tolerance developed diabetes during the 4-year period. Women had higher incidence rates than men. Thirty-eight percent of participants who had impaired glucose tolerance at baseline developed diabetes. When those with impaired and normal glucose tolerance at baseline were

combined, 16.2% of men and 22.2% of women developed diabetes during the 4-year follow-up period. Incidence rates of those with diabetes, who had higher waist-to-hip ratio, BMI, fasting glucose, fasting insulin, and albuminuria at baseline, were significantly higher than those who had normal glucose tolerance at baseline. In women, degree of Indian blood, fasting glucose, and albuminuria were significantly associated with the development of diabetes. Total triglyceride levels and percentage of body fat were positively related to the development of diabetes in men, and apoprotein AI and HDL cholesterol were inversely related to the risk of diabetes. In both men and women who had impaired glucose tolerance at baseline, those with higher 2-h glucose, higher HgA<sub>1c</sub>, higher fasting insulin, and 100% Indian blood had higher rates of conversion to diabetes. In men only, BMI and percentage of body fat were associated with the conversion to diabetes (Lee et al.).

The most predictive variables in men with normal glucose tolerance for the development of diabetes were BMI and triglycerides. The odds ratios indicated that for every 0.6-mg/dl increase in triglyceride and every 5-unit increase in BMI, the risk of developing diabetes in men increased 40% and 38%, respectively. In women with normal glucose tolerance, fasting insulin was the only significant variable related to the risk of developing diabetes (Lee et al., 2002). For every 0.7 ( $\mu$ )U/dl increase in insulin, the risk for women developing diabetes increased 91%. Also in women, those with albuminoidal and impaired glucose tolerance had a threefold increased risk for the development of diabetes than those who did not have albuminuria (Lee et al.). Impaired glucose tolerance, percentage of Indian blood, fasting glucose, 2-hr glucose, fasting insulin, and presence of albuminuria were found to be positively related to the development of diabetes, and age was negatively related (Lee et al.). This study confirmed that diabetes is one of the most severe health problems among the American

Indian population. Those with impaired glucose tolerance should be treated with diabetes medication and placed on a weight-reduction program. Increasing physical activity and reducing weight could prevent the development of type 2 diabetes (Lee et al.).

Gilliland, Carter, Skipper, and Acton (2002) conducted a study to determine whether HgA<sub>1c</sub> levels were elevated among younger American Indians/Alaskan native adults nationally and, if so, to determine the relationship between HgA<sub>1c</sub> levels and age due to treatment type, renal disease, BMI, survival, duration of diabetes, or a poor diabetes health care index. A total of 11,419 American Indian/Alaskan natives aged > 18 years with diabetes were included in the National Indian Health Service (NIHS) diabetes care and outcomes audit that was conducted in 1998. Glucose control was assessed by HgA<sub>1c</sub>. Diabetes duration, treatment type, BMI, and proteinuria were assessed from the diabetes care and outcomes audit data. Of the initial cohort, 9,622 had measured HgA<sub>1c</sub> and 1,158 had calculated HgA<sub>1c</sub> levels. They found HgA<sub>1c</sub> level decreased with increasing age. This inverse relationship was not accounted for by differences in health care index, treatment type, duration of diabetes, proteinuria, or BMI. For ages 18-39 years HgA<sub>1c</sub> levels were 9.2; ages 40-49 years had HgA<sub>1c</sub> levels of 8.9; ages 50-59 years had HgA<sub>1c</sub> levels of 8.8; ages 60-69 years had levels of 8.3; and ages 70 years and older had HgA<sub>1c</sub> levels of 7.8. It was also found that younger participants had higher BMI than the older participants. Those individuals age 55 years and younger had the highest levels of HgA<sub>1c</sub>. This suggested an increased risk for diabetes-related complications in the younger age groups and may, on first glance, be surprising because it is expected that HgA<sub>1c</sub> as well as diabetes increase with age. These high levels of HgA<sub>1c</sub> can possibly be explained by survival bias because older people who have poor diabetes control can die at younger ages. This may also be explained by the younger people



having differences in visit frequency due to family/child/work obligations. There may also be differences in treatment strategies. It has been suggested that younger people are less compliant with a treatment plan (Gilliland et al.). This supports the idea that younger Alaskan native/American Indians have worse diabetes control. Factors underlying the elevated levels of HgA<sub>1c</sub> in the younger generation are not completely understood, but they do not appear to be explained by factors such as BMI, treatment type, duration of diabetes, proteinuria, or diabetes health care index. The higher HgA<sub>1c</sub> levels may be related to rapid changes in lifestyle that have occurred in many American Indian/Alaskan native communities. HgA<sub>1c</sub> levels have been shown to be higher in those who consume the most sugar and fat in those ages less than 55 years (Gilliland et al.).

Historically, diabetes mellitus has not been a disease that impacted Native Americans. Unfortunately, because of a combination of genetic and cultural factors, as well as demographic (low SES, poor access to care), nutritional (dietary shift), behavioral (lack of exercise), and psychological (high psychopathology rates) changes, Native Americans now are facing unprecedented prevalence rates of type 2 diabetes (Acton & Burrows, 2003). While local and national health agencies have attempted to curb this epidemic, Native Americans are found to have much poorer glucose control, resulting in diabetes related complications and mortality far exceeding any other group.

#### *Purpose of Study*

The purpose of this study was to examine role of psychosocial variables in both developing type 2 diabetes and the poorer diabetic outcomes among certain Native American groups. We examined whether those individuals who have not met the criteria for diabetes mellitus are more prone to develop diabetes mellitus if they show signs of depression, cynical hostility, or report anger that is either expressed or unexpressed.

The selection of psychosocial variables has been based on past research with this population, which demonstrated that depression, cynical hostility, and anger demonstrated a statically significant relationship with diabetic outcome variables, while other psychosocial variables did not, when observed in a cross-sectional model (O'Leary, 2001). We examined the impact of depression, cynical hostility, and anger on glucose control among those individuals who were diagnosed with diabetes mellitus. Fasting glucose, a commonly used measure of glucose control, was used because the measure is available for all participants in the Strong Heart Study. We attempted to determine if "psychological distress," rather than specific psychosocial variables, was related to poorer diabetic outcome among a specific Native American population.

### Hypotheses

#### *Hypothesis 1*

There is a relationship between psychosocial factors, such as depression, hostility, and anger, and the incidence of new cases of diabetes mellitus among a Native American population. It is hypothesized that those individuals with scores on psychosocial instruments that indicate depressive symptoms, cynical hostility, or anger will be more likely to develop diabetes mellitus over a 5-year period than those individuals with lower scores on the same instruments.

#### *Hypothesis 2*

There is a relationship between psychosocial factors such as depression, hostility, and anger, and glucose control among individuals with either abnormal glucose tolerance or diabetes mellitus. It is hypothesized that those individuals who have higher

scores on psychosocial instruments will show greater increases in glucose levels over a 5-year period than those individuals who have lower scores on the same instruments.

*Hypothesis 3*

There is an impact of psychological distress, as measured by the Rand Short Form-36 mental health composite score, on diabetic-related outcome variables such as glucose control over a 5-year period. It is hypothesized that those individuals who score lower on the mental health composite variable will have poorer diabetic outcomes.

## CHAPTER III

### METHODS

#### The Strong Heart Study

The Strong Heart Study was initiated in 1988 to study cardiovascular disease among Native American groups in three diverse regions after it was determined that there were very little existing data describing these subgroups. The objective of the Strong Heart Study is to:

Employ standardized methodology to estimate cardiovascular disease mortality and morbidity (incidence and prevalence) rates as well as to allow comparison of cardiovascular disease risk factor levels among American Indian groups living in three different areas: central Arizona, Southwestern Oklahoma, and the Aberdeen area of North and South Dakota. (Lee et al., 1990, p. 1143)

The study, using a longitudinal approach, allows for the examination of cardiovascular risk factors and the incidence of heart disease; it also allows for the examination of these same risk factors and diabetes.

The Strong Heart Study has three primary components, a mortality review, a surveillance of the initial cohort of morbidity and mortality, and a clinical examination (Lee et al., 1990). For the purposes of this study, the data from the clinical examination gathered in the second and third waves were utilized. The purpose of the clinical examination was to gather data on the prevalence rates of angina, myocardial infarction, cerebrovascular disease, hypertension, congestive heart failure, diabetes, and abnormal glucose tolerance. This information was compared not only to other studies from different populations, but also within the study across the three centers and measured risk factors of each (Lee et al.). The clinical examination from both the first and second phases consisted of a personal interview and a physical evaluation (Howard et al.,

1998). The personal interview assessed areas such as family health history, dietary information, activity levels, current health status, and demographic information. The physical examination included measures to assess both cardiac and diabetic status as well as overall health. Strong Heart staff were centrally trained and evaluated in data collection, interviewing techniques, and form completions as described in the Strong Heart Study Manual (Lee, Welty, & Howard, 1993). Procedures were taught and demonstrated by an instructor and all staff had sufficient time to practice and demonstrate their competence of the procedures. All personnel with access to data collected for the study were required to sign a confidentiality pledge, and collected data were stored in a secure location (Lee et al.). Thus far, the Strong Heart Study has completed three different exams and is currently expanding into a family-based study (see Table 1), beginning with sibships who were already Strong Heart participants. This phase of the study included several added components: genetic and expanded psychosocial research.

### Participants

The Strong Heart Study population consists of resident tribal members of the following tribes: Pima, Maricopa, and Papage Indians of central Arizona, who live in the Gila River, Salt River, and Ak-Chin Indian communities; the seven tribes of Southwestern Oklahoma (Apache, Caddo, Comanche, Delaware, Fort Sill Apache, Kiowa, and Wichita); the Oglala and Cheyenne River in South Dakota; and the Spirit Lake community in the Fort Totten area of North Dakota (Howard et al., 1998). Communities within tribes were selected because they were considered by the tribe to be representative of the population in lifestyle, employment, education, and other

Table 1

*Strong Heart Study Timeline*

Phase	Years exam conducted	Variables collected for current study
1	1989 - 1991	Demographic variables
2	1993 - 1995	Psychosocial, time one diabetic variables
3	1998 - 1999	Time two diabetic variables
4	2001 - ongoing	

*Note.* Demographic variables include age and diabetic status; psychosocial variables include mental health composite score, Center for Epidemiological Studies-Depression Scale total score, Spielberger's AX total score, anger in score, anger out score and Cook and Medley total score; Diabetic variables include 2-hr glucose, HGA<sub>1c</sub>, fasting glucose, and BMI.

sociodemographic factors, as well as having the facilities to conduct the examination. While the Pima/Maricopa in Arizona and the Sioux tribes in the Dakotas live on reservations in rural environments, the Oklahoma tribes live among the general population. Another difference among the study participants was that many of the individuals in the Oklahoma tribes utilized their own private health care providers (Lee et al., 1990). Other criteria for the first clinical examination included residing in the study communities and being 45-74 years of age between July 1989 and June 1991 when the examination was conducted (Lee et al.). For participants to be eligible for future study participation, they must have been part of the original cohort. Retention rates for Phase II at the second physical examination averaged 89% (Howard et al.). During the second exam, a pilot study was conducted in the Dakota and Oklahoma sites that introduced psychosocial measures that collected data on perceived stress, hostility, depression, social support, and cultural identification. There were 568 participants in the Oklahoma

and South Dakota sites that were administered the psychosocial measures. The Short Form Health Survey (SF-36, version 1) was included in the entire exam 2 protocol for the Oklahoma and South Dakota sites. The Strong Heart Study cohort consists of 4,549 individuals aged 45-74 who were seen at the first (1989-1991) examination. Participants' glucose measures in the psychosocial pilot group were also compared with the cohort as a whole to ensure that they were representative and that findings could be generalized to the rest of the Strong Heart cohort.

#### Data Request Procedure

A formal request was submitted to the Strong Heart Study Steering Committee (SHSSC) for access to the data for the variables in Appendix A. The variables requested were from the data set for the second exam (1992-1994), and the data set for the third exam (1997-1999), which are the most current data sets, ready for external analysis. See Appendix C.

#### Psychosocial Instruments

##### *Center for Epidemiological Studies--Depression Scale*

The Center for Epidemiological Studies-Depression Scale (CES-D) was used to measure depression. The CES-D was developed to represent four dimensions of depression: negative affect, positive affect, psychomotor distress, and interpersonal relations (Beeber, Shea, & McCorkle, 1998). The same instrument has been used in research on healthy, physically ill, and mentally ill populations in past studies (Carpenter, Hall, Rayens, Sachs, & Cunningham, 1998). The CES-D is a self-report instrument that assesses the presence and severity of depressive symptoms occurring over the past week. Respondents rate each item on a 4-point scale: 0 = *rarely or none of the time*, 1 =

some or a little of the time, 2 = occasionally or a moderate amount of the time, and 3 = most of the time. The CES-D takes approximately 5 minutes for a respondent who understands the instrument to complete (Carpenter et al.). An overall score of 16 is generally considered the score at which the symptomatology has reached clinical levels for this instrument (Radloff, 1977). The CES-D has been shown to have adequate test-retest reliability, test-retest  $r = 0.51$  at 2.5 weeks (Radloff). Cronbach's alpha = 0.85 ( $p < 0.001$ ) in a community sample, and 0.907 ( $p < 0.001$ ) in a clinical sample of older adults (Himmelfarb & Murrell, 1983). The internal consistency (Cronbach's alpha) of the CES-D is .89 (Lee et al., 1993). Refer to Appendix E for a copy of the CES-D.

#### *Cook and Medley Hostility Scale*

The Cook and Medley Hostility Scale (Ho) was used to assess hostility. The Ho scale was derived based on items taken from the Minnesota Multiphasic Personality Inventory (Barefoot, Dodge, Peterson, Dahlstrom & Williams, 1989). It consists of eight true or false items that ask questions such as "it is safe to trust nobody," or "most people lie to get ahead." The participants were told that they were going to be asked what they think about other people. The Ho is designed to measure cynical beliefs and mistrust of others, which is a construct that has been found to be a predictor of heart disease in some populations (Vogele, 1997). The Ho scale has an internal consistency of .86 and a test-retest correlation after 1 year of .85 (Lee et al., 1993). Refer to Appendix E for a copy of the Cook and Medley Ho.

#### *Spielberger's Anger Expression Scale*

Spielberger's Anger Expression Scale (AX) was designed to determine how people usually react or behave when they feel angry or furious. It differentiates between experienced and expressed feelings of anger (Lisspers, Nygren, & Soderman, 1998).



The original scale consists of 20 items rated on a 4-point Likert scale (1 = *almost never* to 4 = *almost always*). The AX scale is considered to be one of the standard assessments of anger, being used in numerous studies, including numerous cross-sectional and prospective studies of the role of anger in cardiovascular disease conducted by John Barefoot at the Duke Medical Center and the Stanford Coronary Prevention Program (Lee et al., 1993). Knight, Chriholm, Paulin, and Waal-Manning (1988) supported the validity of the AX scale through factor analysis and concluded that the AX subscales were valid. Internal consistency (Alpha Coefficient) of the 20-item AX scale and the 8-item anger-in and anger-out subscales range from .73 to .84 (Lee et al.). Refer to Appendix E for a copy of the Spielberger's AX.

#### *RAND 36-item Short-Short-Form Health Survey*

The RAND Corporation originally developed the Short Form Health Survey (SF-36) for the Medical Outcome Study (MOS; Ware, 1993). The SF-36 contains 36 questions that cover eight areas: physical functioning, role limitations due to physical problems, social functioning, bodily pain, general mental health, role limitation due to emotional problems, vitality, and general health perception. It was designed as a self-administered questionnaire and usually takes about 10 minutes to complete. One of the limitations of this instrument, when administered in the South Dakota site, was the fact that many of the participants spoke Lakota as their first language and the instrument was translated to them. This may have affected the scale's performance in ways that are not known. The SF-36 is considered by many in the medical community as the standard for measuring perceived health related quality of life and has been widely used in outcome studies. The SF-36 does not yield a total score, but rather weighted subscores for each of its domains. Each subscore has a range of 0-100, with a higher score

indicating a more favorable health status. The SF-36 also yields two composite scores in the areas of physical health (PCS) and mental health (MCS). These scales were designed to better summarize the subscales of the SF-36. They have been shown to have reliability coefficients of .93 for the PCS and .88 for the MCS (Ware, 1994). It is these composite scores that will be used in the current study. This instrument was used to assess each participant's perceived quality of life. Refer to Appendix E for a copy of the SF-36.

### Diabetic Outcome Measures

#### *Hemoglobin A<sub>1c</sub>*

The measure of hemoglobin A<sub>1c</sub> (HbA<sub>1c</sub>) is a widely used laboratory test to determine overall long-term blood glucose control. It reflects the average blood glucose over a 2-3 month period preceding the test. Glycation is defined as glucose that has attached itself to the hemoglobin portion of the red blood cell (South Dakota Diabetes Control Program, 1999). The process is nonenzymatic and irreversible. Because the life span of the red blood cell is typically 120 days, the test reflects glycemic control for a 2-3 month period. Normal values for this test range from 4-7%. IHS standards of care consider a value of 7 or greater to be an indicator of poor glycemic control. This test is commonly used and preferred among practitioners managing people with diabetes (South Dakota Diabetes Control Program).

#### *Other Glucose Measurements*

Two different blood glucose measures were used. The fasting glucose is a simple blood test done after fasting for 8 hr. The postload glucose tolerance test is a measurement of blood glucose taken 2 hr after the participant has been given a drink

containing 75g of anhydrous glucose dissolved in water. The cutoff for fasting glucose is a lab value of less than 126 mg/dl. Scores above this suggest diabetes. If the value is between 110 and 126 mg/dl, the subject is considered to have impaired fasting glucose and is at-risk for developing diabetes mellitus (South Dakota Diabetes Control Program, 1999). Values that are greater than 200 mg/dl on the 2 hr glucose tolerance test are considered to be in the diabetic range, while values between 140-200 mg/dl are in the impaired glucose range (South Dakota Diabetes Control Program).

### Data Analysis

#### *Hypothesis 1*

Only those individuals that were either normal glucose tolerant or had impaired glucose tolerance at Time 1 were included in the analysis. A "new diabetic" variable was created by identifying those participants who had a fasting glucose value of 126 mg/dl or above during the third examination (Time 2) or who were taking medication for diabetes. Logistic regression analysis was used to analyze the predictions based on the independent variables from the second exam (Time 1; depression, hostility, anger, and demographic variables) and the "new diabetic" (Time 2) variable. All calculations were done using SPSS for Windows 10.1 (SPSS, Chicago) and  $p < 0.05$  values were considered statistically significant.

#### *Hypothesis 2*

Two separate analyses were conducted in order to determine what the impact of psychosocial variables was on glucose control. Subtracting the fasting glucose value of exam 2 from the fasting glucose at exam 3 created a "glucose change" variable. In the first analysis, only those individuals who were in the normal glucose tolerance range at

exam 2 (Time 1) were included. Linear regression analysis was used to correlate data between the independent variables from exam 2 (depression, hostility and anger, and demographic variables) and the "glucose change" variable. The second analysis was the same as the first, but included only those participants who fell in the abnormal glucose range at exam 2. All calculations were done using *SPSS for Windows 10.1* (SPSS, Chicago). The critical probability used to determine significance was  $p < 0.05$ .

### *Hypothesis 3*

Two separate analyses were conducted in order to determine the impact of psychological distress as measured by the SF-36 composite score, on glucose change. A glucose change variable was created for each of the dependent variables by subtracting the 2<sup>nd</sup> exam value from the 3<sup>rd</sup> exam value. Linear regression analysis was used to correlate data between the SF-36 mental health composite scores, demographic data, and the glucose change variable. All calculations were done using *SPSS for Windows 10.1* (SPSS, Chicago),  $p < 0.05$  was considered significant.

In order to determine the relationship between the psychosocial variables and other variables that may impact diabetic outcome, univariate analysis was run on the psychosocial variables and certain demographic variables to assess multicollinearity.

## CHAPTER IV

## RESULTS

## Descriptive Statistics

The sample consisted of 1,384 Native American males and 2,254 Native American females, with an average age of 60 years. Of the sample, 57% completed high school, 9% completed a bachelor's level education, 2% master's level, and less than 1% had completed a doctoral level education. Of the cohort, 14.5 % reported making less than \$5,000 annually, with 69% reporting to have less than a \$35,000 a year household income. Less than 2% of the sample reported having a household income of over \$50,000 a year. The psychosocial sample demographics were similar to those of the main cohort. It consisted of 201 Native American males and 352 Native American females, with an average age of 61 years. Of the sample, 63% completed high school, 8% completed a bachelor's level education, 3% master's level, and less than 1% had completed a doctoral level education. In the psychosocial sample, 15% reported making less than \$5,000 annually, with 77% reporting to have less than a \$35,000 a year household income. Less than 2% of the sample reported having a household income of over \$50,000 a year.

Descriptive statistics for each of the three glucose control measures were computed for all participants (see Table 2). Overall, the mean glucose measures for all participants in the study reflected a high prevalence of glucose intolerance. According to the guidelines set by the ADA (2003a), the criterion for diagnosis of diabetes for the fasting glucose test was  $\geq 126$  mg/dl and  $> 200$  mg/dl for the 2-hr glucose test. The overall mean score of the participants at the beginning of the study was 153.57 mg/dl. The 2-hr glucose mean score was 168.9 mg/dl. While the fasting glucose and 2-hr

Table 2

*Descriptive Statistics for Health-Related Variables for Entire Cohort*

Health variables	N	Min	Max	Mean	SD
BMI exam 2	3639	14.06	73.02	31.19	6.47
BMI exam 3	2889	15.98	65.44	31.22	6.50
BMI change	2877	-28.55	10.10	-.21	2.62
HgA <sub>1c</sub> exam 2	3509	1.8	19.7	6.91	2.39
HgA <sub>1c</sub> exam 3	1592	4.4	14.7	7.75	1.99
HgA <sub>1c</sub> change	1532	-12.3	9.30	-.08	2.25
2-hr glucose tol exam 2	2065	31.0	659.0	168.89	94.55
2-hr glucose tol exam 3	1212	5.0	468.00	146.71	66.20
2-hr glucose tol change	1200	-154.00	357.00	40.94	61.62
Fasting exam 2	3593	52.0	736.00	153.57	78.81
Fasting exam 3	2878	26.0	497.00	142.06	65.39
Fasting change	2841	-625.00	307.0	-10.35	68.98

*Note.* Change variables could only be calculated for those individuals who participated in both exam 2 and 3. It should be noted that this data set contained notable outliers, which are accurate results for this group. BMI ranges: < 18.5 underweight, 18.5-24.9 normal, 25.0-29.9 overweight, > 30.0 obese. HgA<sub>1c</sub> cutoff:  $\geq 7$ , 2-hr glucose cutoff; > 2000, Fasting glucose cutoff:  $\geq 110$  impaired glucose tolerance,  $\geq 126$  diabetes.

glucose tests are direct measures of a person's glucose levels, HgA<sub>1c</sub> measures the average glucose levels over a 3-month period. The HgA<sub>1c</sub> mean of 6.91 falls below the recommended cutoff  $\geq 7$ , but corresponds to 120 mg/dl average over a 3-month period (South Dakota Diabetes Control Program, 1999). Overall, 60.6% of the 3,660 participants fell either in the impaired glucose tolerance category (110-125 mg/dl) or actually met criteria for a diagnosis of diabetes mellitus.

At exam 3 the mean glucose measures for all participants in the study continued to reflect a high prevalence of glucose intolerance. The mean score of the participants at exam 3 was 142.06 mg/dl, a significant decrease. The 2-hr glucose mean score was 146.71 mg/dl at exam 3. Both of the state glucose measures showed a significant

decrease, while the HgA<sub>1c</sub> mean of 7.75, demonstrated a significant increase and was above the recommended cutoff of  $\geq 7$ . The HgA<sub>1c</sub> change variable does not reflect this increase, due to only those individuals who had HgA<sub>1c</sub> labs drawn at both exam 2 and 3 were used to calculate this variable, and actually showed a decrease of -.08.

Descriptive statistics were also computed for those individuals who participated in the psychosocial pilot (see Table 3), the sample that this study is largely comprised of. Overall, the psychosocial pilot sample has slightly better controlled glucose levels than the entire cohort. As with the cohort, the mean glucose measures reflected a high prevalence of glucose intolerance. The fasting glucose mean score of the participants at the beginning of the study was 139.60 mg/dl. The 2-hr glucose mean score was 139.60 mg/dl. The HgA<sub>1c</sub> mean of 6.46 also falls below the recommended cutoff  $\geq 7$ . 55.3% of the 568 psychosocial participants fell either in the impaired glucose tolerance category (110-125 mg/dl) or actually met criteria for a diagnosis of diabetes mellitus.

At exam 3 the mean glucose measures for the psychosocial group continued to reflect a high prevalence of glucose intolerance. The mean score of the individuals in the psychosocial group at exam 3 was 133.88 mg/dl. The 2-hr glucose mean score was 137.5 mg/dl at exam 3. Both of the state glucose measures showed a mild decrease, while the HgA<sub>1c</sub> mean of 7.75, demonstrated a significant increase and was above the recommended cutoff of  $\geq 7$ . Overall, the groups appear to look closer at exam 3 than at exam 2.

#### Difference Between Psychosocial Pilot Study and the Strong Heart Cohort

Independent group *t* tests were performed comparing the study variables

Table 3

*Descriptive Statistics for Health-Related Variables for Psychosocial Pilot Sample*

Health variables	N	Min	Max	Mean	SD
BMI exam 2	567	14.06	54.88	30.86	5.78
BMI exam 3	475	16.36	49.92	30.44	5.44
BMI change	474	-18.83	9.15	-.4527	2.61
HgA <sub>1c</sub> exam 2	554	3.7	14.50	6.46	2.06
HgA <sub>1c</sub> exam 3	224	5.0	13.40	7.65	1.93
HgA <sub>1c</sub> change	218	-7.5	6.50	.1459	1.95
2-hr glucose tol exam 2	391	40.0	628.0	139.60	76.38
2-hr glucose tol exam 3	240	46.0	380.0	137.5	54.87
2-hr glucose tol change	236	-152.00	271.0	32.31	51.70
Fasting exam 2	557	61.0	498.0	139.60	65.13
Fasting exam 3	480	66.0	465.0	133.88	61.59
Fasting change	470	-272.00	293.0	-3.18	58.94

between the individuals who were administered the psychosocial instruments and the rest of the study cohort (see Table 4). Participants in the psychosocial pilot study showed lower HgA<sub>1c</sub> results,  $t(3507) = -4.91$ ,  $p < .0001$ , than the rest of the cohort. Not only was this difference statistically significant, but clinically significant as well. While the abnormal glucose group appeared to have slightly higher HgA<sub>1c</sub> values, the mean of the psychosocial HgA<sub>1c</sub> results was 6.4, while the average for the rest of the cohort was 7.00, right at the clinical cutoff score. There were similar differences in the fasting glucose lab values as well,  $t(3591) = -4.56$ ,  $p < .0001$ . The two groups did not differ significantly on the 2-hr glucose values or by BMI.

Several of the psychosocial instruments were modified after initial field trials, mainly because some recruiters and participants believed that some items were either culturally inappropriate or not easily understood. As a result, many of the scores are not comparable to the established norms of the instruments. While the same battery of



Table 4

*Descriptive Statistics for the Psychosocial Pilot Study and the Strong Heart Cohort—H<sub>g</sub>A<sub>1c</sub>, 2-hr Glucose, Fasting Glucose, Body Mass Index, and Age*

Study variable	N	Mean	SD
Pilot H <sub>g</sub> A <sub>1c</sub>	554	6.4	2.06
Cohort H <sub>g</sub> A <sub>1c</sub>	2955	7.0	2.44
Pilot 2-hr glucose	391	162.6	76.4
Cohort 2-hr glucose	1674	170.4	98.3
Pilot fasting glucose	557	139.6	65.1
Cohort fasting glucose	3036	156.1	80.8
Pilot BMI	567	30.9	5.8
Cohort BMI	3072	31.3	6.6
Pilot age	568	61.1	8.1
Cohort age	3092	59.8	7.9

psychosocial instruments were administered to each participant in the psychosocial group, individual tests were only scored if they were complete, which causes variations in the individual *N* for each psychosocial instrument (see Table 5).

The mean score for all participants on the CES-D was 10.62 (*SD* = 8.01), with 21% scoring above the established clinical cutoff score of 16 for the CES-D. The mean score for all participants for the Cook and Medley Ho scale was 4.16 (*SD* = 4.9). Not all items of Cook and Medley Ho scale were administered during the phase II exam, so comparisons to national norms were not feasible. The Spielberger AX yielded three scores for all participants: total (*m* = 45.03, *SD* = 5.74), angerin (*m* = 25.43, *SD* = 4.18), and angerout (*m* = 12.94, *SD* = 3.46). Overall, the participants' scores for the Spielberger AX were lower than that of the national norms (46.30 for males, 48.05 for females), indicating a somewhat lower rate of anger expression (Spielberger et al., 1976). The overall average of the Rand SF-36 MCS was in the normal range (see Table 5).

Table 5

*Descriptive Statistics for Psychosocial Measures: Center for Epidemiological Studies—Depression Scale, Cook and Medley, Spielberger's AX, and RAND SF-36 Mental Health Composite Score*

Measure	<i>N</i>	Min	Max	Mean	<i>SD</i>
CES-D	568	0	46	10.62	8.01
Ho	529	0	72	4.16	4.90
AXTOT	527	21	61	45.03	5.74
ANGERIN	529	9	36	25.43	4.18
ANGEROUT	534	5.0	29	12.94	3.46
MCS	2890	16	74.28	53.11	9.11

*Note.* 21% of sample exceeded CES-D cutoff score, 10.5% showed mild psychological distress on the MCS, while 2% showed moderate psychological distress on MCS.

Difference Between Normal Glucose Group  
and Abnormal Glucose Group

Independent group *t* tests were performed comparing the study variables between the normal glucose tolerance group and the abnormal glucose tolerance group for exam 2 (Time 1). Participants in both groups were not statistically different for any psychosocial variables. Participants in the abnormal glucose tolerance group did show higher 2-hr glucose scores,  $t(2032) = 4.606, p < .000$ , than the normal glucose tolerance group. While the abnormal glucose group had slightly higher HgA<sub>1c</sub> scores  $t(3453) = 1.913, p < .056$ , and fasting glucose scores  $t(3536) = 2.749, p < .06$ , the differences were not statistically significant. The groups also did not differ on the diabetic risk factors of BMI,  $t(3581) = -1.46, p < .144$  or age,  $t(3602) = .193, p < .847$ .

### Difference Between Exam 2 and Exam 3 Groups

Independent group *t* tests were performed comparing the study variables between the those that completed both exam 2 and exam 3 and those individuals who dropped out of the study for some unforeseen reason. Only those individuals who were in the psychosocial pilot group were examined. Participants in both groups were not statistically different for any psychosocial variables. Individuals who dropped out of the study did show higher fasting glucose levels,  $t(389) = .429, p < .0001$ . They were also had slightly lower BMI,  $t(565) = -.443, p < .003$ . They did not differ on the other risk factors or glucose measures.

### Correlations Among Psychosocial Variables

The correlation matrix of psychosocial variables used in the analysis is presented in Table 6, in order to inspect for multicollinearity, which may have impacted the findings. An inspection of this matrix reveals noteworthy colinearity between the psychosocial variables. There was also a significant correlation between BMI and the 2-hr glucose change variable. The colinearity between the psychosocial variables and among the BMI and diabetic variables was not unexpected, and it did pose a potential problem when interpreting the regression analysis. This relation between the independent variables decreased the amount of unique variance accounted for by each when assessing relation to each of the dependent variables within a regression equation. (See Appendix B.)

#### *Question 1*

What is the relationship of depressive symptoms, cynical hostility or anger, and

Table 6

*Pearson Product Moment Correlation Matrix for Variables Used in Analyses*

Variables	MSC3	CESTOT	CMTOT	AXTOT	ANGERIN	ANGEROUT
MSC3	1.0	-.185**	-.047*	-.031	.110*	-.154**
CESTOT		1.0	.202**	.001	-.182**	.206**
CMTOT			1.0	-.081	-.130**	.014
AXTOT				1.0	.718**	.575**
ANGERIN					1.0	-.100*
ANGEROUT						1.0
	GLURCH	FASCH	HGA <sub>1c</sub> CH	NEWDIA	SHS2BMI	SHS2AGE
MSC3	.024	.063**	.076**	-.32	.000	.062**
CESTOT	-.134*	-.019	.064	-.009	.067	-.052
CMTOT	-.019	.019	.004	.009	.022	.088*
AXTOT	-.002	-.062	.158*	.047	.005	-.161**
ANGERIN	.028	-.030	.101	.043	.019	-.017
ANGEROUT	-.081	-.013	.107	.017	-.008	-.203**
GLURCH	1.0	.508**	.300**	.472**	.243**	.030
FASCH		1.0	.637**	-.115**	.014	.001
HGA <sub>1c</sub> CH			1.0	-.080*	.031	-.024
NEWDIA				1.0	.254**	-.024
SHS2BMI					1.0	-.153**
SHS2AGE						1.0

*Note.* MSC3 = mental health composite score; CESTOT = Center for Epidemiological Studies-Depression Scale total score; CM = Cook and Medley total score; AXTOT = Spielberger's AX total score; ANGERIN = Spielberger's AX anger in subscale; ANGEROUT = Spielberger's AX anger out subscale; GLURCH = 2-hr glucose change variable; FASCH = fasting glucose change variable; HGA<sub>1c</sub>CH = HGA<sub>1c</sub> change variable; NEWDIA = new diabetic variable; SHS2BMI = BMI at exam 2; SHS2AGE = Age at exam 2.

the development of diabetes mellitus over a 5-year period of time? It was hypothesized that those individuals with scores on psychosocial instruments that indicated depressive symptoms, cynical hostility, or anger, would be more likely to develop diabetes mellitus over a 5-year period of time as opposed to those individuals with lower scores on the same instruments. Only those individuals that were either normal glucose tolerant or had impaired glucose tolerance were included in the analysis. Any individual with a diagnosis of diabetes or those with a fasting glucose level of 126 mg/dl or greater at time 1 were excluded. See Tables 7 and 8.

Logistic regression analyses were conducted in order to determine if psychosocial variables contributed to the development of type 2 diabetes. Overall, none of the psychosocial variables were statistically significant in any of the models conducted, CES-D ( $p < .822$ ), Cook and Medley ( $p < .976$ ), Anger total ( $p < .262$ ), Anger-in ( $p < .450$ ), and Anger-out ( $p < .464$ ). In all models, BMI predicted the onset of type 2 diabetes ( $p < .0001$ ). Most striking was how powerful the odds of getting diabetes was to BMI. The Exp(B) for BMI ranged from 11 - 14%, depending on which psychosocial variable was included in the model. For example, when depression, BMI and age were used as predictors, every increase of 1 on the participant's BMI, increased their risk for becoming diabetic by 11%. Refer to Appendix D for detailed tables.

### *Question 2*

What impact did the psychosocial factors of depression, anger, and hostility have on glucose-control change between exam 2 and exam 3? It was hypothesized that worse outcomes on the psychosocial instruments would correlate with the HgA<sub>1c</sub>, the 2-hr glucose loading and the fasting glucose change measures, indicating that poor glycemic control was associated with worse psychological functioning. It was also

Table 7

*Comparison of Depression at Time 1 and Diabetic Status at Time 1*

Variables	DM/IGT No	DM/IGT Yes
Depression No	189	245
Depression Yes	58	76

*Note.* CES-D Score for Time 1, DM Status for Time 1 includes diabetes mellitus and impaired glucose tolerance.

Table 8

*Comparison of Depression at Time 1 and Diabetic Status at Time 2*

Variables	DM/IGT No	DM/IGT Yes
Depression No	108	326
Depression Yes	31	103

*Note.* CES-D Score for Time 1, DM Status for Time 1 includes diabetes mellitus and impaired glucose tolerance.

speculated that there was a relationship between psychosocial variables and glucose control among those individuals with normal glucose tolerance. Separate stepwise multiple regression analyses were conducted on those with normal glucose tolerance and on those who had abnormal glucose tolerance. Additionally, only one psychosocial variable was included in the analysis at a time because of multicollinearity.

*Normal glucose tolerance group.* Separate stepwise multiple regression analyses were conducted to examine the relationship between the psychosocial variables (mental health composite score, DES-D total score, Cook and Medley total score, Spielberger's AX total score, anger in and anger out subscales), diabetes risk factors (BMI and age) and glucose control change measures (H<sub>gA</sub><sub>1c</sub> change, 2-hr glucose change and fasting glucose change). For H<sub>gA</sub><sub>1c</sub>, the regression was statistically significant,  $F(1, 99) = 6.208$ ,

$p < .014$ . The CES-D (see Table 9) was the only psychosocial measure to load,  $R = .243$ ,  $p < .014$ . No other models loaded with either risk factors or psychosocial variables (refer to Appendix D).

BMI was a predictor in several models that used the 2-hr glucose change variable as the dependent variable were statistically significant. When CES-D was the psychosocial independent variable,  $F(1, 92) = 9.800$ ,  $p < .002$ , BMI loaded into the model,  $R = .310$ ,  $p < .043$ . BMI also loaded when the Spielberger's AX,  $F(1, 94) = 6.480$ ,  $p < .013$ , was used as the predictor variable,  $R = .254$ ,  $p < .013$ . BMI again loaded when the Cook Medley,  $F(1, 92) = 9.800$ ,  $p < .037$ , was used as the predictor variable,  $R = .215$ ,  $p < .037$ .

None of the models involving the fasting glucose change variable were statistically significant for the normal glucose tolerance group (refer to Appendix D).

*Abnormal glucose tolerance group.* The same models were run with the abnormal glucose tolerance group as were run with the normal glucose tolerance group. All models with the HgA<sub>1c</sub> change as the dependent variable were not statistically

Table 9

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Hemoglobin A<sub>1c</sub> Change as a Dependent Measure for the Normal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
Hemoglobin A <sub>1c</sub> and CES-D					
CES-D	Regression	22.698	1	22.698	6.208
	Residual	1769.793	440	4.022	
	Total	1785.641	441		

*Note.* Hemoglobin A<sub>1c</sub> and CES-D Beta = .243.

Change as the dependent variable were not statistically significant (refer to Appendix D). When the model was run with 2-hr glucose change as the dependent variable (see Tables 10 and 11), the diabetes risk factors entered the model, but the psychosocial variables did not statistically impact the outcome variable. When CES-D was the psychosocial independent variable,  $F(1, 134) = 6.246, p < .002$ , BMI and age both loaded into the model,  $R = .293, p < .043$ . BMI and age also loaded for all of the Spielberger's AX subscales (see Table 10). When the anger-in subscale,  $F(2, 132) = 4.570, p < .012$ , was used as the predictor variable,  $R = .254, p < .012$ . When the anger-out variable was the psychosocial predictor variable, BMI and age,  $F(2, 132) = 4.682, p < .011$ , again loaded in the model,  $R = .256, p < .011$ . With the Spielberger's AX total score,  $F(2, 132) = 4.570, p < .012$ , BMI and age were also statistically significant,  $R = .254, p < .012$ . Age was statistically significant,  $F(1, 133) = 4.024, p < .047$ , when hostility was the psychosocial predictor variable,  $R = .172, p < .047$ .

The fasting glucose change variable (see Table 12) had only one significant finding when used as the dependent variable in the abnormal group. With the Spielberger's AX anger-out subscale,  $F(1, 264) = 4.661, p < .032$ , BMI was a predictor of poorer glucose control,  $R = .132, p < .032$ . No other models loaded with either risk factors or psychosocial variables (refer to Appendix D).

### *Question 3*

What impact does the psychological distress as measured by the SF-36 MCS have on glucose-control change between exam 2 and exam 3? It was hypothesized that the levels of psychological distress would correlate with the HgA<sub>1c</sub>, the 2-hr glucose loading, and the fasting glucose change measures, indicating that poor glycemic control was associated with worse psychological functioning. The only model to show any



Table 10

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Glucose Tolerance Change as a Dependent Measure for Normal Glucose*

*Tolerance Group*

Model	Source	SS	df	MS	F
Glucose tolerance and CES-D					
SHS2BMI	Regression	24152.017	1	24152.017	9.800
	Residual	226742.59	92	2464.593	
	Total	250894.61	93		
Glucose tolerance and ANGERIN					
SHSAGE	Regression	15554.209	1	15554.209	6.480
	Residual	225619.75	94	2400.210	
	Total	241173.96	95		
Glucose tolerance and ANGEROUT					
SHS2BMI	Regression	15554.209	1	15554.209	6.480
	Residual	225619.75	94	2400.210	
	Total	241173.96	95		
Glucose tolerance and AXTOT					
SHS2BMI	Regression	15554.209	1	15554.209	6.480
	Residual	225619.75	94	2400.210	
	Total	241173.96	95		
Glucose tolerance and CMTOT					
SHS2BMI	Regression	10691.331	1	10691.331	4.492
	Residual	221353.26	93	2380.143	
	Total	232044.59	94		

*Note.* Glucose tolerance and CES-D Beta = .232; Glucose tolerance and ANGERIN Beta = .184; Glucose tolerance and ANGEROUT Beta = .181; Glucose tolerance and AXTOT Beta = .185; Glucose tolerance and CMTOT Beta = .172.

Table 11

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and 2-hr Glucose Tolerance Change as a Dependent Measure for Abnormal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
2-hr Glucose tolerance and CES-D					
SHS2BMI	Regression	22106.755	1	22106.755	7.637
	Residual	387886.65	134	2894.676	
	Total	409993.40	135		
SHS2BMI and SHS2AGE	Regression	35202.388	2	17601.194	6.246
	Residual	374791.02	133	2817.978	
	Total	409993.40	135		
2-hr Glucose tolerance and ANGERIN					
SHSAGE	Regression	12869.364	1	12869.364	4.717
	Residual	362833.57	133	2728.072	
	Total	134			
SHS2AE and SHS2BMI	Regression	24331.884	2	12165.942	4.570
	Residual	351371.05	132	2661.902	
	Total	375702.93	134		
2-hr Glucose tolerance and ANGEROUT					
SHS2BMI	Regression	12379.932	1	12379.932	4.538
	Residual	365576.30	134	2728.181	
	Total	135			
SHS2BMI and SHS2AGE	Regression	24858.278	2	12429.139	4.682
	Residual	353097.96	133	2654.872	
	Total	377956.24			
2-hr Glucose Tolerance and AXTOT					
SHSAGE	Regression	12869.364	1	12869.364	4.717
	Residual	362833.57	133	2728.072	
	Total	134			
SHS2AGE and SHS2BMI	Regression	24331.884	2	12165.942	4.570
	Residual	351371.05	132	2661.902	
	Total	3757002.93	134		
2-hr Glucose Tolerance and CMTOT					
SHS2AGE	Regression	10826.820	1	10826.820	4.024
	Residual	355125.27	132	2690.343	
	Total	365952.09	133		

significant findings were with the 2-hr glucose change variable as the dependent variable,  $F(1, 565) = 44.95, p < .000$ , with BMI being a predictor of poorer glucose control,  $R = .271, p < .000$  (see Table 13). No other models loaded with either risk factors or psychosocial variables (refer to Appendix D).

Table 12

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Fasting Glucose Change as a Dependent Measure for Abnormal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
Fasting glucose and ANGEROUT					
SHS2BMI	Regression	13324.008	1	13324.008	4.661
	Residual	754641.85	264	2858.492	
	Total	767965.85	265		

Table 13

*Regression Models Using Psychological Distress as a Predictor Variable and 2-hr Glucose Change as a Dependent Measure for Abnormal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
SHS2BMI	Regression	155045.99	1	155045.991	44.958
	Residual	1948526.9	565	3448.720	
	Total	2103572.9	566		

## CHAPTER V

### DISCUSSION

Native American tribes have a large number of risk factors that are associated with an increased incidence for developing diabetes and predicting poor diabetic outcome. These risk factors include but are not limited to, low SES, barriers influencing access to care, nutritional factors, specific cultural variables, substance abuse, inconsistency across tribes for access to quality diabetic education and treatment programs, alarming rates of obesity, higher prevalence rates of depression, and a likely genetic predisposition. Diabetes within this population is a relatively new phenomenon, with little or no incidence being reported before 1940 (West, 1974), and steadily increasing to the epidemic proportions seen today. There have been several theories proposed to try and explain this phenomenon, these range from emphasis on nutrition (Lang, 1985), to genetics (Brosseau et al., 1979; Ghodes & Acton, 2000, Goetsch & Wiebe, 1998; Lee et al., 1995; Neel, 1962), to specific lifestyles (Ravussin et al., 1994), to environment. One theory of particular interest to this study is the relationship between psychosocial variables and diabetes. Psychosocial variables among the general population have been shown to increase the incidence of the disease and contribute to increased detrimental effects of diabetes.

This study examined the way certain, specific psychosocial variables impact or influence the incidence of new cases of diabetes; if depression, anger, or hostility predict poor glycemic control; and whether a global measure of psychological distress influences glycemic control after a 4-year follow-up.

There are several striking findings based on the examination of the study group's basic demographics variables. One was the very high prevalence rate of those

participants who fell into the abnormal glucose tolerance group. This group consisted of participants who were either diabetic or had impaired glucose tolerance. In this study, 60.6% of the participants fell into this group, which is far higher than the national average of any group other than certain other Native American tribes. The averages of the individual glucose measures were all clinically high. The average for fasting glucose at exam 2 was 153.57 mg/dl, well above the diagnostic cut-off score of 126 mg/dl. The average for the 2-hr oral glucose tolerance test was 168.89 mg/dl, also well above the test's cut-off score of 155 mg/dl at 2 hours. The HgA<sub>1c</sub> average of 6.91 was below the clinical cut-off score of 7; but this would correspond to a glucose level of 120 mg/dl over a 3-month period in the impaired glucose tolerance range (South Dakota Diabetes Control Program, 1999). Overall, the average glucose control of this group was very poor, indicating both a high prevalence of diabetes and poorly controlled glucose levels. It should be noted that these rates only reflect the prevalence of the older community members who participated in the Strong Heart Study, because only those individuals who were 45-74 years old at the time of the first examination were eligible to be included in the cohort. The current data do not reflect the rates for younger members who were not included in the original cohort. These data should be available when the Strong Heart Study exam 4 data are completed, when younger family members of the original cohort are included.

A large part of this may be explained by the high prevalence of several diabetic risk factors. The first risk factor being the fact that 100% of the participants are Native American. Ethnicity has been shown to be a risk factor for developing diabetes, especially among certain Native American tribes (ADA, 2004; Lee et al., 1995; Rewers & Hamman, 1995). Another risk factor is the relatively advanced age of the participants. The average age of the participants is 60 years. Nevertheless when compared to the

national average of older adults, only 15% of adults nationally over the age of 65 years meet the criteria for diabetes (Rewers & Hamman). The review also showed relatively low income, which has been suggested to be a predictor of both higher rates of diabetes and poorer outcome (ADA, 2004; Nelson & Manson, 2000; Rewers & Hamman). Of the sample, 69% reported an average household income of lower than \$35,000, and 14.5% reported an income lower than \$5,000 a year. This is far below the national average for the recorded time period. Education has also been shown to be a predictor of diabetic outcome (Nelson & Manson). In this sample, 57% had completed high school and 9% had completed a 4-year college education. One of the strongest predictors of both developing diabetes and the severity of its complications is an individual's BMI (Rewers & Hamman). This risk factor appears to be quite prevalent in this sample with the average BMI being 31.19 kg/m<sup>2</sup>, well within the obese range.

The overall mean score ( $M = 10.62$ ) of the CES-D scale was in the average range. A total of 21% of the sample scored over the clinical cut-off score of 16 on this measure. This is alarming because the national prevalence rate of depression is around 5% (APA, 2000). While a score above the clinical cut-off score does not necessarily indicate a diagnosis of a mood disorder, it is considered a valuable screening tool, and such a score warrants further investigation. The overall reported anger scores ( $M = 45.03$ ) were lower than the standardization sample average, which could have some beneficial implications for cardiovascular disease. The mental health composite score ( $M = 53.11$ ), which in this study was used as a predictor of "psychological distress," also fell within the normal range.

When comparing the normal group to the abnormal glucose tolerance group, an expected difference among the glucose variables was observed, with those in the abnormal glucose tolerance producing higher scores on the 2-hr glucose and fasting

glucose measures. Surprisingly, there was not a statistically significant difference on the HgA<sub>1c</sub> measure. There was also no statistically significant difference between the groups on the risk factors or psychosocial variables. When the psychosocial pilot group was compared to the rest of the cohort, there was a difference, with the psychosocial group showing lower HgA<sub>1c</sub> and fasting glucose lab values. There were no significant differences on the 2-hr glucose lab values or the other risk factors. It is unclear what would account for these differences or lack thereof between the groups. There were no unexpected findings when multicollinearity between the variables was examined.

## Hypotheses

### *Hypothesis 1*

The first hypothesis of this study suggested that those individuals with scores on psychosocial instruments that indicated depressive symptoms, cynical hostility, or anger would be more likely to develop diabetes mellitus over a 5-year period of time than those individuals with lower scores on the same instruments. Only those individuals who were not diabetic or had impaired glucose tolerance at the time were included in the analysis, while those who had been diagnosed with diabetes were excluded.

This author's analysis found no evidence of an association between either depression, anger, or cynical hostility, and the incidence of diabetes at a 4-year follow-up. What was found in all models was that BMI was a predictor in developing type 2 diabetes in a 4-year period. This finding is consistent with the ADA (2004) recommendations for treatment and other outcome studies and reviews (The Diabetes Prevention Research Group, 2000, 2002; The Diabetes Prevention Program, 1999; Gohdes & Acton, 2000; Rewers & Hamman, 1995).

Previous studies have suggested a relationship between psychosocial variables and the incidence of diabetes (Eaton et al., 1996; Egede et al., 2002; Kawakami et al., 1999). Egede and associates concluded that individuals with diabetes were more likely to develop depression than nondiabetic individuals after examining outcomes of 825 adults with diabetes and 20,688 adults without diabetes using the 1996 Medical Expenditure Panel Survey. They also concluded that individuals with diabetes who were depressed had health care expenditures that were 4.5 times higher than individuals with diabetes who were not depressed. Further, this study also demonstrated the association between depressive symptoms and the occurrence of type 2 diabetes. Eaton and colleagues concluded that the presence of a major mood disorder substantially increased the risk of diabetes. In their 13-year prospective follow-up reports from a community-based study, major depression was associated with a 2.2 fold increase in the development of diabetes, after they adjusted for other risk factors. No similar increase was found associated with milder forms of depressive symptomatology. Kawakami and associates found that depressive symptoms may be associated with the onset of type 2 diabetes. Their study concluded that moderate or severe levels of depressive symptoms were associated with later occurrence of type 2 diabetes.

There are several possible reasons for the lack of significant findings. It is possible that due to the nature of the analysis, including very strong predictors such as BMI and age, any variance accounted for by the psychosocial barriers went unnoticed. Another possibility, is that differences due to genetic, cultural or lifestyle among the Native American groups examined in the current study, psychosocial variable do not predict the incidence of developing type 2 diabetes.



### *Hypothesis 2*

The second hypothesis predicted that those individuals who have higher scores on psychosocial instruments would show greater increases in glucose levels over a 5-year-period of time than those individuals who have lower scores on the same instruments. In the present study, depression was the only psychosocial variable to predict a change in glucose control. Separate analyses were conducted on both the normal glucose tolerant and abnormal glucose tolerant groups. In the normal glucose tolerance group, the CES-D was the only psychosocial variable to load predicting HgA<sub>1c</sub> change among the normal groups. BMI as a risk factor predicted 2-hr glucose change for all models. None of the variables showed a relationship with the fasting glucose change variable. In the abnormal glucose tolerance group, none of the psychosocial variables demonstrated a statistically significant relationship with glucose change over a 4-year period. Rather, BMI and age were both significant in certain models.

Multiple studies have suggested that psychosocial variables are related to poorer glucose control (Anderson et al., 2001; Booth-Kewley & Friedman, 1987; Ciechanowski et al., 2000; DiMatteo et al., 2000; Egede & Zheng, 2003; Lustman et al., 2000; Surwit et al., 2002; Talbot & Nouwen, 2000). There have been several different theories to explain this relationship. Several researchers have suggested that psychosocial variables impact glucose by means of lowering the probability that an individual will adhere to the complex diabetic regimen or make the lifestyle changes needed to prevent complications (Ciechanowski et al.; DiMatteo et al.; Peyrot et al., 1999; Talbot & Nouwen). Other studies found direct relationships between poorer glucose control and psychosocial measures. Talbot and Nouwen conducted an extensive literature review in order to determine the relationship between depression and diabetes. They concluded that depression is a predictor of type 2 diabetes, but a major depressive disorder is not a

predictor of type 1 diabetes, rather having type 1 diabetes is a risk factor for developing a major depressive episode. Poorer glycemic control among individuals with increased negative psychosocial variables were noted as well. Rubin and Peyrot (1994) demonstrated that depression and hyperglycemia may exacerbate each other at the neuroendocrine level.

Lustman and associates (2000) found a relationship between depression and glucose control, but concluded that relationship was bidirectional. Nichols and Brown (2003) determined depression was more common in individuals with type 2 diabetes than among matched control subjects, but also suggested that the relationship was bidirectional. Anderson and associates (2001) also determined that there was a bidirectional relationship between depression and diabetes, concluding that the presence of diabetes doubles the odds of comorbid depression. There was limited literature examining the relationship between anger or hostility with diabetes. Both of these variables have been used in examining the outcome of other diseases (Booth-Kewley & Friedman, 1987). Surwit and associates (2002) examined the relationship between hostility and glucose control. They found increased hostility appeared to have a negative impact on fasting glucose and insulin sensitivity.

### *Hypothesis 3*

Finally, the third hypothesis was that individuals who score lower on the mental health composite variable, thus self-reporting more psychological distress would have poorer diabetic outcomes. The findings for this question are similar to previous findings in this study. This analysis did not show any significant relationship between "psychological distress" and glucose control change. BMI was a predictor in the 2-hr glucose model, which is consistent with the findings in other models when other more

specific psychosocial variables with the two demographic risk factors. This finding is not consistent with findings in the literature suggesting that psychological distress may be a predictor (Talbot & Nouwen, 2000).

### Implications

One of the most profound findings of this study is that over 60% of the participants were either diagnosed with diabetes or met the criteria for impaired glucose tolerance. These individuals are at increased risk of cardiovascular disease, stroke, renal failure, blindness, and lower limb amputation. While the high prevalence rates of diabetes among Native Americans is well documented in the literature, these findings are consistent with diabetes being at epidemic levels among the Native American tribes in the Strong Heart Study. Another significant finding was the high report of depressive symptoms among the Strong Heart Study Cohort. Of those individuals who were administered the CED-D, 21% scored in the clinical range, which is four times the predicted rate when compared to the national base rates for depression.

Another primary finding of this study is that psychosocial variables as a whole do not appear to predict incidence of diabetes among individuals of the Native American tribes represented among the Strong Heart Study. There are several potential reasons for these findings. The fact that there are so many significant risk factors present among these particular groups, such as significant obesity, low SES levels, and potential genetic factors from having Native American heritage, it is possible that any variance accounted for by the psychosocial factors is relatively small in comparison. Another possibility is that psychosocial variables, indeed, do not represent the same risk as they have with the mainstream population.

The second significant finding is that psychosocial variables do not appear to predict glucose control in diabetes among Native Americans represented in the Strong Heart Study. The only significant finding was between depression and HgA<sub>1c</sub> among individuals with normal glucose tolerance. This finding is consistent with other studies examining depression and glucose control (O'Leary, 2001) among Native Americans. As with this study, the finding was only significant among individuals with normal glucose tolerance. The other psychosocial variables, anger, hostility, and perceived psychological distress, did not demonstrate a relationship with glucose control over a 4-year period. BMI demonstrated itself to be a significant predictor of both the incidence of new diabetic cases and poorer glycemic control over time.

These findings have implications for treatment among those tribes that participated in the study. Health care providers should be made aware of the high reported rate of depression among the participants and consider either a medical intervention or a referral to the appropriate mental health agency. Because of the reluctance of many individuals to seek mental health treatment, providers may wish to consider screening for depression as part of the normal primary care protocol, and then take time to educate individuals about the cause and different treatments available for depression.

Another treatment implication is the fact that BMI demonstrated itself to be the primary risk factor in this study for both incidence of developing new cases of diabetes and as a predictor of poorer glucose control over the course of the disease. Based on these findings, significant resources should be put into educating individuals that not only is this a significant risk factor for the development of diabetes, but that it is a changeable risk factor. Multiple studies have shown that life style modification can prevent or slow both the incidence of new diabetes cases and lower the chances of

developing significant complications (ADA, 2003a; Diabetes Control and Complications Trial Research Team, 1993a, 1993b; Gohdes & Acton, 2000; Fabricatore & Wadden, 2003; Tuomilehto et al., 2001). Tribes and the health care providers may wish to consider developing a multidisciplinary team approach, attempting to change this at the individual, family, and community levels. While such a program would prove to be costly in the short term, the long-term monetary and human cost savings could be profound.

### Limitations

There are several limitations of the current study that should be noted. While the Strong Heart Study Cohort was studied as a whole, it should be noted that it is comprised of 13 separate tribes, located in three distinct regions that are very different in culture, acculturation, SES, and have different levels of medical care and barriers to receiving care. This factor was not controlled for in this study and could have had significant implications to the results. Caution should be used in generalizing the results of this study to not only Native Americans, but also to the Strong Heart Study Cohort. The first is that many of the psychosocial instruments are designed to measure their construct at a 2- to 3-week period. In addition, they are screeners, designed to identify individuals who may need more follow-up. Thus these instruments may produce more false positives. To further confound this, sometimes the questionnaires were translated into Lakota, which may impact their validity. Another limitation is that of mortality among the study participants. While the Strong Heart Study has very good retention rates, 88% of surviving participants at exam 3 (Center for American Indian Health Research, 2006), those individuals who died or were unable to participate in exam 3 may have impacted the results of the study in some unforeseen way, especially in light of the fact that those who dropped out showed higher fasting glucose levels than

those who participated in exam 3. Of the original 568 individuals involved in the original psychosocial pilot, 481 (84.7%) participated in exam 3. Ideally, ongoing psychosocial assessment between the exams would have provided a more robust measure of the relationship between diabetes and psychosocial variables rather than the psychosocial “snapshot” provided in the current study. This would also allow the ability to differentiate between those variables that are trait versus state. This may also be true of the outcome variables as well. While glucose levels were drawn at two different points, several years apart, it may be again providing us with a “snap shot.” Impaired glucose levels can take years to decades before complications or poor health outcomes exhibit themselves. Body mass index was used as a risk factor, which is common in epidemiological studies, but it should be noted that it is not an actual measure of body composition. Individuals who are highly athletic or have a higher than average ratio of muscle mass will appear to be overweight or obese. Another factor that was not controlled for in the current study was diabetic medications, which may have resulted in a loss in our ability to measure the true impact of psychosocial variables because of stable glucose control.

During the conduct of this research our government decided it would be good for me to “vacation” in Afghanistan. That turned out to be a limiting factor in my ability to analyze and discuss the findings, as I would lose my train of thought while dodging bullets and rockets. Then my major professor moved to New York (something I still cannot understand), and although this really did not influence our ability to work together (Utah, New York, or Italy), what does it matter from Afghanistan.

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APPENDICES

## Appendix A:

## Data Request

**STRONG HEART STUDY****REQUEST FOR DATA**

**Title of project:** Psychosocial factors and their Relationship to Diabetic Outcome Among the Strong Heart Study Cohort

**Investigators:** Brian O'Leary, M.S. Utah State University  
Barbara Howard, Ph.D. MedStar Research Institute  
Kevin Masters, Ph.D. Utah State University

**Purpose:** Other, for completion of Dissertation  
(Will consult the Strong Heart Study for approval before any submission for journal publication)

**Date Needed:** 09 / 01 / 02 (please allow 1-2 weeks form data request received)  
mm dd yy

**Data for Study Period:** \_\_\_\_\_  X  \_\_\_\_\_  X   
Phase - I Phase - II Phase III

**Center:** \_\_\_\_\_ \_\_\_\_\_ \_\_\_\_\_  X   
Arizona Oklahoma South/North Dakota All 3 Centers

**Variables Needed:** (List all the variables)

I am requesting the data on the following individuals who participated in the psychosocial pilot study in the Oklahoma and South Dakota sites.

I request all of the variables raw and derived on the following protocols

CES-D	Participants in the Psychosocial Pilot
COOK Medley (HO)	Participants in the Psychosocial Pilot
Spielberger AX	Participants in the Psychosocial Pilot
SF-36	All participants Exam II and III
Personal Interview Form II	All participants Exam III
Medical History Form Items 7-11	All participants Exam III

Lab results	
HgA1c	All participants Exam II and III
Fasting Glucose	All participants Exam II and III
GTT	All participants Exam II and III
Diabetic Foot Screen	All participants Exam II and III
BMI	All participants Exam II and III

\*\*\*\*\*

**COORDINATING CENTER USE ONLY:**

**Date Received:** \_\_\_\_\_

**Date Data Delivered:** \_\_\_\_\_

Strong Heart Study

Request for Data

## Appendix B:

## Variable Explanation

**Variable Explanations**

Variables were requested for all participants

CESTOT	CES-D Total score
CMTOT	COOK Medley (HO) total score
AXTOT	Spielberger AX total score
ANGERIN	Spielberger AX internalized anger score
ANGEROUT	Spielberger AX externalized anger score
GLURCH	Glucose Tolerance Change Score
FASCH	Fasting Glucose Change Score
HGA1cCH	HGA1c Change Score
NEWDIA	New incidence between exam 2 & 3
SHS2BMI	BMI at exam 2
SHS2AGE	Age at exam 2

## Appendix C:

## Data Set Management Records

## DISSERTATION DATA SET MANAGEMENT RECORDS

**SF-36 Health Survey****Exam II**

The SF-36 to subscales are scored so that a higher score indicates a better health state.

The items for the specific subscales are as follows:

Physical Functioning: 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Role-Physical: 13, 14, 15, 16

Bodily Pain: 21, 22

General Health: 1, 33, 34, 35, 36

Vitality: 23, 27, 29, 31

Social Functioning: 20, 32

Role-Emotional: 17, 18, 19

Mental Health: 24, 25, 26, 28, 30

Reported Health Transition: 2

There are several items that need to be recoded as per the manual:

The first variable is the sum of the weighted scores. The second is the scaled score, which is:

$$\text{Transformed score} = \left\{ \frac{\text{actual score} - \text{lowest possible score}}{\text{Possible raw score range}} \right\} \times 100$$

**Physical Functioning:** 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Computed as: qua3 + qua4 + qua5 + qua6 + qua7 + qua8 + qua9 + qua10 + qua11 + qua12 = pf

Scaled score: ((pf - 10) / 20) \* 100 = pfs

This scale is directly scored,

The non-scaled variable is pf

Lowest possible: 10

Possible raw score: 20

The scaled variable is pfs

**Role-Physical:** 13, 14, 15, 16

Computed as: qua13 + qua14 + qua15 + qua16 = rp

Scaled Score:  $((rp - 4) / 4) * 100 = rps$

This scale is directly scored

The non-scaled variable is rp

Lowest possible: 4

Possible raw score: 4

The scaled variable is rps:

### **Bodily Pain: 21, 22**

Computed as:  $qua21r + qua22r = bp$

Scaled scale:  $((bp - 2) / 10) * 100 = bps$

Item 21: 1=6, 2=5.4, 3=4.2, 4=3.1, 5=2.2, 6=1

Item 22: If 22 is 1 and 21 is 1 = 6      If 22 is 1 and 21 is 2 through 6 = 5  
2 = 4, 3=3, 4=2, 5=1

This was done in syntax as follows:

DO IF (qua22 = 1 AND qua21 > 1) .

  COMPUTE qua22r = 5 .

ELSE IF (qua22 = 1 AND qua21 = 1) .

  COMPUTE qua22r = 6 .

ELSE IF (qua22 = 2 AND qua21 >= 1) .

  COMPUTE qua22r = 4 .

ELSE IF (qua22 = 3 AND qua21 >= 1) .

  COMPUTE qua22r = 3 .

ELSE IF (qua22 = 4 AND qua21 >= 1) .

  COMPUTE qua22r = 2 .

ELSE IF (qua22 = 5 AND qua21 >= 1) .

  COMPUTE qua22r = 1 .

END IF .

The non-scaled variable is: bp

Lowest possible: 2

Possible raw score: 10

The scaled variable is: bfs

### **General Health: 1, 33, 34, 35, 36**

Computed as:  $qua33 + qua36 + qua1r + qua34r + qua35r = gh$

Scaled score:  $((gh - 5) / 20) * 100 = ghs$

Item 1: 1=5, 2=4.4, 3=3.4, 4=2, 5=1

Items 33 & 36, Directly scored

Items 34 & 35: 1=5, 2=4, 3=3, 4=2, 5=1

The non-scaled variable is gh  
 Lowest possible: 5  
 Possible raw score: 20  
 The scaled score is: ghs

**Vitality:** 23, 27, 29, 31

Computed as:  $qua29 + qua31 + qua23r + qua27r = v$   
 Scaled score:  $((v - 4) / 20) * 100 = vs$

Items 29 & 31 are directly scored  
 Items 23 & 27 are 1=6, 2=5, 3=3, 4=4, 5=2, 6=1

The non-scaled variable is v  
 Lowest possible: 4  
 Possible raw score: 20  
 The scaled score is: vs

**Social Functioning:** 20, 32

Computed as:  $qua32 + qua20r = sf$   
 Scaled score:  $((sf - 2) / 8) * 100 = sfs$

Item 32 is directly scored  
 Item 20 is: 1=5, 2=4, 3=3, 4=2, 5=1

The non-scaled variable is sf  
 Lowest possible: 2  
 Possible raw score: 8  
 The scaled score is: sfs

**Role-Emotional:** 17, 18, 19

Computed as:  $qua17 + qua18 + qua19 = re$   
 Scaled score:  $((re - 3) / 3) * 100 = res$

All items are directly scored

The non-scaled variable is re  
 Lowest possible: 3  
 Possible raw score: 3  
 The scaled score is: res



**Mental Health: 24, 25, 26, 28, 30**

Computed as:  $qua24 + qua25 + qua28 + qua26r + qua30r = mh$   
 Scaled score:  $((mh - 5) / 25) * 100$

Items 24, 25, & 28 are directly scored  
 Items 26 & 30: 1=6, 2=5, 3=4, 4=3, 5=2, 6=1

The non-scaled variable is mh  
 Lowest possible: 5  
 Possible raw score: 25  
 The scaled score is mhs

**Reported Health Transition: 2**

Not scored as a subscale, but used are an independent variable to assess changes in health

**SF-36 Composite scores**

PF\_Z = (pfs - 84.52404) / 22.89490  
 RP\_Z = (rps - 81.19907) / 33.79729  
 BP\_Z = (bps - 75.49196) / 23.55879  
 GH\_Z = (ghs - 72.21316) / 20.16964  
 VT\_Z = (vs - 61.05453) / 20.86942  
 SF\_Z = (sfs - 83.59753) / 22.37642  
 RE\_Z = (res - 81.29467) / 33.02717  
 MH\_Z = (mhs - 74.84212) / 18.01189

Agg\_phys = (pf\_z \* .42402) + (rp\_z \* .35119) + (bp\_z \* .31754) + (gh\_z \* .24954) + (vt\_z \* .02877) + (sf\_z \* -.00753) + (re\_z \* -.19206) + (mh\_z \* -.22069)

Agg\_ment = (pf\_z \* -.22999) + (rp\_z \* -.12329) + (bp\_z \* -.09731) + (gh\_z \* -.01571) + (vt\_z \* .23534) + (sf\_z \* .26876) + (re\_z \* .43407) + (mh\_z \* .48581)

PCS = 50 + (agg\_phys \* 10)

MCS = 50 + (agg\_ment \* 10)

**Exam III**

The SF-36 to subscales are scored so that a higher score indicates a better health state.

The items for the specific subscales are as follows:

Physical Functioning: 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Role-Physical: 13, 14, 15, 16

Bodily Pain: 21, 22

General Health: 1, 33, 34, 35, 36

Vitality: 23, 27, 29, 31

Social Functioning: 20, 32

Role-Emotional: 17, 18, 19

Mental Health: 24, 25, 26, 28, 30

Reported Health Transition: 2

There are several items that need to be recoded as per the manual:

The first variable is the sum of the weighted scores. The second is the scaled score, which is:

$$\text{Transformed score} = \left\{ \frac{\text{actual score} - \text{lowest possible score}}{\text{Possible raw score range}} \right\} \times 100$$

**Physical Functioning:** 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Computed as: qua3\_3 + qua3\_4 + qua3\_5 + qua3\_6 + qua3\_7 + qua3\_8 + qua3\_9 + qua3\_10 + qua3\_11 + qua3\_12 = pf3

Scaled score: ((pf3 - 10) / 20) \* 100 = pf3s

This scale is directly scored,

The non-scaled variable is pf3

Lowest possible: 10

Possible raw score: 20

The scaled variable is pf3s

**Role-Physical:** 13, 14, 15, 16

Computed as: qua3\_13 + qua3\_14 + qua3\_15 + qua3\_16 = rp3

Scaled Score: ((rp3 - 4) / 4) \* 100 = rp3s

This scale is directly scored

The non-scaled variable is rp3

Lowest possible: 4

Possible raw score: 4

The scaled variable is rp3s:

**Bodily Pain: 21, 22**

Computed as:  $qua3\_21r + qua3\_22r = bp3$

Scaled scale:  $((bp3 - 2) / 10) * 100 = bp3s$

Item 21: 1=6, 2=5.4, 3=4.2, 4=3.1, 5=2.2, 6=1

Item 22: If 22 is 1 and 21 is 1 = 6      If 22 is 1 and 21 is 2 through 6 = 5  
2 = 4, 3=3, 4=2, 5=1

This was done in syntax as follows:

DO IF (qua3\_22 = 1 AND qua3\_21 > 1) .

  COMPUTE qua3\_22r = 5 .

ELSE IF (qua3\_22 = 1 AND qua3\_21 = 1) .

  COMPUTE qua3\_22r = 6 .

ELSE IF (qua3\_22 = 2 AND qua3\_21 >= 1) .

  COMPUTE qua3\_22r = 4 .

ELSE IF (qua3\_22 = 3 AND qua3\_21 >= 1) .

  COMPUTE qua3\_22r = 3 .

ELSE IF (qua3\_22 = 4 AND qua3\_21 >= 1) .

  COMPUTE qua3\_22r = 2 .

ELSE IF (qua3\_22 = 5 AND qua3\_21 >= 1) .

  COMPUTE qua3\_22r = 1 .

END IF .

The non-scaled variable is: bp3

Lowest possible: 2

Possible raw score: 10

The scaled variable is: bp3s

**General Health: 1, 33, 34, 35, 36**

Computed as:  $qua3\_33 + qua3\_36 + qua3\_1r + qua3\_34r + qua3\_35r = gh3$

Scaled score:  $((gh3 - 5) / 20) * 100 = gh3s$

Item 1: 1=5, 2=4.4, 3=3.4, 4=2, 5=1

Items 33 & 36, Directly scored

Items 34 & 35: 1=5, 2=4, 3=3, 4=2, 5=1

The non-scaled variable is gh3

Lowest possible: 5

Possible raw score: 20

The scaled score is: gh3s

**Vitality: 23, 27, 29, 31**

Computed as:  $qua3\_29 + qua3\_31 + qua3\_23r + qua3\_27r = v3$

Scaled score:  $((v3 - 4) / 20) * 100 = v3s$

Items 29 & 31 are directly scored  
 Items 23 & 27 are 1=6, 2=5, 3=3, 4=4, 5=2, 6=1

The non-scaled variable is v3  
 Lowest possible: 4  
 Possible raw score: 20  
 The scaled score is: v3s

**Social Functioning:** 20, 32

Computed as:  $qua3\_32 + qua3\_20r = sf3$   
 Scaled score:  $((sf3 - 2) / 8) * 100 = sf3s$

Item 32 is directly scored  
 Item 20 is: 1=5, 2=4, 3=3, 4=2, 5=1

The non-scaled variable is sf3  
 Lowest possible: 2  
 Possible raw score: 8  
 The scaled score is: sf3s

**Role-Emotional:** 17, 18, 19

Computed as:  $qua3\_17 + qua3\_18 + qua3\_19 = re3$   
 Scaled score:  $((re3 - 3) / 3) * 100 = re3s$

All items are directly scored

The non-scaled variable is re3  
 Lowest possible: 3  
 Possible raw score: 3  
 The scaled score is: res3

**Mental Health:** 24, 25, 26, 28, 30

Computed as:  $qua3\_24 + qua3\_25 + qua3\_28 + qua3\_26r + qua3\_30r = mh3$   
 Scaled score:  $((mh3 - 5) / 25) * 100 = mh3s$

Items 24, 25, & 28 are directly scored  
 Items 26 & 30: 1=6, 2=5, 3=4, 4=3, 5=2, 6=1

The non-scaled variable is mh3  
 Lowest possible: 5  
 Possible raw score: 25  
 The scaled score is mh3s

## Reported Health Transition: 2

Not scored as a subscale, but used are an independent variable to assess changes in health

### SF-36 Composite scores

$$\begin{aligned} \text{PF3\_Z} &= (\text{pf3s} - 84.52404) / 22.89490 \\ \text{RP3\_Z} &= (\text{rp3s} - 81.19907) / 33.79729 \\ \text{BP3\_Z} &= (\text{bp3s} - 75.49196) / 23.55879 \\ \text{GH3\_Z} &= (\text{gh3s} - 72.21316) / 20.16964 \\ \text{VT3\_Z} &= (\text{v3s} - 61.05453) / 20.86942 \\ \text{SF3\_Z} &= (\text{sf3s} - 83.59753) / 22.37642 \\ \text{RE3\_Z} &= (\text{re3s} - 81.29467) / 33.02717 \\ \text{MH3\_Z} &= (\text{mh3s} - 74.84212) / 18.01189 \end{aligned}$$

$$\text{Agg\_phy3} = (\text{pf3\_z} * .42402) + (\text{rp3\_z} * .35119) + (\text{bp3\_z} * .31754) + (\text{gh3\_z} * .24954) + (\text{vt3\_z} * .02877) + (\text{sf3\_z} * -.00753) + (\text{re3\_z} * -.19206) + (\text{mh3\_z} * -.22069)$$

$$\text{Agg\_men3} = (\text{pf3\_z} * -.22999) + (\text{rp3\_z} * -.12329) + (\text{bp3\_z} * -.09731) + (\text{gh3\_z} * -.01571) + (\text{vt3\_z} * .23534) + (\text{sf3\_z} * .26876) + (\text{re3\_z} * .43407) + (\text{mh3\_z} * .48581)$$

$$\text{PCS3} = 50 + (\text{agg\_phy3} * 10)$$

$$\text{MCS3} = 50 + (\text{agg\_men3} * 10)$$

### Spielberger AX Scales

- Spielberger AX Scale, Reverse score items 2, 4, 6, 7, 9, 11,13, 15,16, 17, 19, New variable names are same as old except with a R after (Spiel2R). Reversed scored by Menu Transform: Recode: In different Variable: Old and new values : 1=4, 2=3, 3=2, 4=1

Computed scores Total: Transform: Compute:  $\text{spiel3} + \text{spiel5} + \text{spiel8} + \text{spiel10} + \text{spiel12} + \text{spiel14} + \text{spiel18} + \text{spiel20} + \text{spiel21} + \text{spiel2r} + \text{spiel4r} + \text{spiel6r} + \text{spiel7r} + \text{spiel9r} + \text{spiel11r} + \text{spiel13r} + \text{spiel15r} + \text{spiel16r} + \text{spiel17r} + \text{spiel19r} = \text{Axtot}$

Compute scores Anger-In: Transform: Compute:

$\text{spiel4r} + \text{spiel6r} + \text{spiel7r} + \text{spiel9r} + \text{spiel11r} + \text{spiel13r} + \text{spiel15r} + \text{spiel16r} + \text{spiel17r} = \text{Angerin}$

Compute scores Anger-Out: Transform: Compute

$\text{spiel3} + \text{spiel8} + \text{spiel10} + \text{spiel12} + \text{spiel14} + \text{spiel18} + \text{spiel20} + \text{spiel21} = \text{Angerout}$

The possible ranges for these scales are as follows :

Total 20 - 80

Angerin 8-32

Angerout 8-32

**Cook Medley**

Compute scores Cmtot: Transform: Compute:  $\text{cook2} + \text{cook3} + \text{cook4} + \text{cook5} + \text{cook6} + \text{cook7} + \text{cook8} + \text{cook9} = \text{Cmtot}$

There were no reversed scores on this, possible range 0-8

**Center for Epidemiological Studies Depression Scale (CES-D)**

Items 5, 9, 13, & 17 were reversed scored: 1=4, 2=3, 3=2, 4=1, , and were labeled with a r to indicate reversal of item.

Compute scores Cesdtot: Transform: Compute:

$\text{ces2} + \text{ces3} + \text{ces4} + \text{ces6} + \text{ces7} + \text{ces8} + \text{ces10} + \text{ces11} + \text{ces12} + \text{ces14} + \text{ces15} + \text{ces16} + \text{ces18} + \text{ces19} + \text{ces20} + \text{ces22} + \text{ces5r} + \text{ces9r} + \text{ces13r} + \text{ces17r} = \text{cesdtot}$

Appendix D:  
Results of Analysis

**Question 1**

Table D-1

*Logistic Regression Models Using Psychosocial, BMI, and Age as Predictor Variables of New Cases of Diabetes between Exam 2 and Exam 3*

Variables	B	S.E.	Wald	df	Sig.	Exp(B)
<i>N</i> = 281						
1.0 = 157 (NGT)						
2.0 = 124 (DM)						
S2BMI	.105	.025	18.110	1	.000	1.111
S2Age	.025	.016	2.554	1	.110	1.025
CESTOT	.004	.016	.051	1	.822	1.004
Constant	-5.020	1.323	14.395	1	.000	.007
<i>N</i> = 263						
1.0 = 153						
2.0 = 110						
S2BMI	.114	.026	18.706	1	.000	1.121
S2Age	.019	.017	1.352	1	.245	1.020
CMTOT	-.001	.025	.001	1	.976	.999
Constant	-5.036	1.381	13.301	1	.000	.006
<i>N</i> = 269						
1.0 = 152						
2.0 = 117						
S2BMI	.129	.017	22.504	1	.000	1.138
S2Age	.025	.017	2.298	1	.130	1.026
AXTOT	.026	.023	1.258	1	.262	1.026
Constant	-6.955	1.887	13.590	1	.000	.001
<i>N</i> = 270						
1.0 = 153						
2.0 = 117						
S2BMI	.112	.026	18.466	1	.000	1.118
S2Age	.020	.016	1.568	1	.210	1.021
ANGERIN	.022	.029	.571	1	.450	1.022
Constant	-5.534	1.568	12.460	1	.000	.004
<i>N</i> = 271						
1.0 = 153						
2.0 = 118						
S2BMI	.132	.027	23.896	1	.000	1.141
S2Age	.025	.017	2.181	1	.140	1.025
ANGEROUT	.030	.041	.536	1	.464	1.030
Constant	-6.230	1.604	15.096	1	.000	.002

## Question 2

Table D-2

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Hemoglobin A<sub>1c</sub> Change as a Dependent Measure for the Normal Glucose Tolerance Group*

Model	Source	SS	df	MS	F	
Hemoglobin A <sub>1c</sub> and CES-D						
CES-D	Regression	22.698	1	22.698	6.208	
	Residual	1769.793	440	4.022		
	Total	1785.641	441			
Variables entered	N	R	R <sup>2</sup>	AdjR <sup>2</sup>	R <sup>2</sup> Change	Sig.
CESTOT	101	.243	.059	.050	.059	.014
Hemoglobin A <sub>1c</sub> and ANGERIN						
No variable entered model						
Hemoglobin A <sub>1c</sub> and ANGEROUT						
No variable entered model						
Hemoglobin A <sub>1c</sub> and AXTOT						
No variable entered model						
Hemoglobin A <sub>1c</sub> and CMTOT						
No variable entered model						



Table D-3

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Fasting Glucose Change as a Dependent Measure for Normal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
Fasting gluc <sub>os</sub> e and CES-D					
No variable entered model					
Fasting gluc <sub>os</sub> e and ANGERIN					
No variable entered model					
Fasting glucose and ANGEROUT					
No variable entered model					
Fasting gluc <sub>os</sub> e and AXTOT					
No variable entered model					
Fasting glucose and CMTOT					
No variable entered model					

Table D-4

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Glucose Tolerance Change as a Dependent Measure for Normal Glucose*

*Tolerance Group*

Model	Source	SS	df	MS	F
Glucose tolerance and CES-D					
SHS2BMI	Regression	24152.017	1	24152.017	9.800
	Residual	226742.59	92	2464.593	
	Total	250894.61	93		
Glucose tolerance and ANGERIN					
SHSAGE	Regression	15554.209	1	15554.209	6.480
	Residual	225619.75	94	2400.210	
	Total	241173.96	95		
Glucose tolerance and ANGEROUT					
SHS2BMI	Regression	15554.209	1	15554.209	6.480
	Residual	225619.75	94	2400.210	
	Total	241173.96	95		
Glucose tolerance and AXTOT					
SHS2BMI	Regression	15554.209	1	15554.209	6.480
	Residual	225619.75	94	2400.210	
	Total	241173.96	95		
Glucose tolerance and CMTOT					
SHS2BMI	Regression	10691.331	1	10691.331	4.492
	Residual	221353.26	93	2380.143	
	Total	232044.59	94		

Table D-5

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Hemoglobin A<sub>1c</sub> Change as a Dependent Measure for the Abnormal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
Hemoglobin A <sub>1c</sub> and CES-D					
No variable entered model					
Hemoglobin A <sub>1c</sub> and ANGERIN					
No variable entered model					
Hemoglobin A <sub>1c</sub> and ANGEROUT					
No variable entered model					
Hemoglobin A <sub>1c</sub> and AXTOT					
No variable entered model					
Hemoglobin A <sub>1c</sub> and CMTOT					
No variable entered model					

Table D-6

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and  
2-hr Glucose Change as a Dependent Measure for Abnormal Glucose Tolerance*

*Group*

Model	Source	SS	df	MS	F
Fasting glucose and CES-D					
No variable entered model					
Fasting glucose and ANGERIN					
No variable entered model					
Fasting glucose and ANGEROUT					
SHS2BMI	Regression	13324.008	1	13324.008	4.661
	Residual	754641.85	264	2858.492	
	Total	767965.85	265		
Fasting glucose and AXTOT					
No variable entered model					
Fasting glucose and CMTOT					
No variable entered model					

Table D-7

*Regression Models Using Psychosocial, BMI, and Age as Predictor Variables and Glucose Tolerance Change as a Dependent Measure for Abnormal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
Glucose tolerance and CES-D					
SHS2BMI	Regression	22106.755	1	22106.755	7.637
	Residual	387886.65	134	2894.676	
	Total	409993.40	135		
SHS2BMI and SHS2AGE	Regression	35202.388	2	17601.194	6.246
	Residual	374791.02	133	2817.978	
	Total	409993.40	135		
Glucose tolerance and ANGERIN					
SHSAGE	Regression	35202.388	2	17601.194	4.717
	Residual	374791.02	133	2817.978	
	Total	409993.40	135		
SHS2AGE and SHS2BMI	Regression	24331.884	2	12165.942	4.570
	Residual	351371.05	132	2661.902	
	Total	375702.93	134		
Glucose tolerance and ANGEROUT					
SHS2BMI	Regression	12379.932	1	12379.932	4.538
	Residual	1365576.30	134	2728.181	
	Total	1377956.23	135		
SHS2BMI and SHS2AGE	Regression	24858.278	2	12429.139	4.682
	Residual	353097.96	133	2654.872	
	Total	365952.09	135		
Glucose tolerance and AXTOT					
SHSAGE	Regression	12869.364	1	12869.364	4.717
	Residual	362833.57	133	2728.072	
	Total	375702.93	134		
SHS2AGE and SHS2BMI	Regression	24331.884	2	12165.942	4.570
	Residual	351371.05	132	2661.902	
	Total	375702.93	134		
Glucose tolerance and CMTOT					
SHS2AGE	Regression	10826.820	1	10826.820	4.024
	Residual	355125.27	132	2690.343	
	Total	365952.09	133		

Table D-8

*Regression Models Using Psychological Distress as Predictor Variables and 2-hr  
Glucose Change as a Dependent Measure for Abnormal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
SHS2BMI	Regression	155045.99	1	155045.991	44.958
	Residual	1948526.9	565	3448.720	
	Total	2103572.9	566		

Table D-9

*Regression Models Using Psychological Distress as Predictor Variables and  
Hemoglobin A<sub>1c</sub> Change as a Dependent Measure for Abnormal Glucose  
Tolerance Group*

Model	Source	SS	df	MS	F
No variable entered model					

Table D-10

*Regression Models Using Psychological Distress as Predictor Variables and Fasting  
Glucose Change as a Dependent Measure for Abnormal Glucose Tolerance Group*

Model	Source	SS	df	MS	F
No variable entered model					

Table D-11

*Pearson Product Moment Correlation Matrix for Variables Used in Analyses*

Variables	MSC3	CESTOT	CMTOT	AXTOT	ANGERIN	ANGEROUT
MSC3	1.0	-.185**	-.047*	-.031	.110*	-.154**
CESTOT		1.0	.202**	.001	-.182**	.206**
CMTOT			1.0	-.081	-.130**	.014
AXTOT				1.0	.718**	.575**
ANGERIN					1.0	-.100*
ANGEROUT						1.0
	GLURCH	FASCH	HGA <sub>1c</sub> CH	NEWDIA	SHS2BMI	SHS2AGE
MSC3	.024	.063**	.076**	-.32	.000	.062**
CESTOT	-.134*	-.019	.064	-.009	.067	-.052
CMTOT	-.019	.019	.004	.009	.022	.088*
AXTOT	-.002	-.062	.158*	.047	.005	-.161**
ANGERIN	.028	-.030	.101	.043	.019	-.017
ANGEROUT	-.081	-.013	.107	.017	-.008	-.203**
GLURCH	1.0	.508**	.300**	.472**	.243**	.030
FASCH		1.0	.637**	-.115**	.014	.001
HGA <sub>1c</sub> CH			1.0	-.080*	.031	-.024
NEWDIA				1.0	.254**	-.024
SHS2BMI					1.0	-.153**
SHS2AGE						1.0

*Note.* MSC3 = mental health composite score; CESTOT = Center for Epidemiological Studies-Depression Scale total score; CM = Cook and Medley total score; AXTOT = Spielberger's AX total score; ANGERIN = Spielberger's AX anger in subscale; ANGEROUT = Spielberger's AX anger out subscale; GLURCH = 2-hr glucose change variable; FASCH = fasting glucose change variable; HGA<sub>1c</sub>CH = HGA<sub>1c</sub> change variable; NEWDIA = new diabetic variable; SHS2BMI = BMI at exam 2; SHS2AGE = Age at exam 2.

\* $p < .05$ , \*\* $p < .01$ .

## Appendix E:

## Strong Heart Study Protocols

## THE STRONG HEART STUDY II

## CES-D SCALE

1. How was the questionnaire administered? \_\_\_\_\_  
 1=By interviewer, 2=By self, 3=Refused

*Here are some questions (Q2-Q21) about your feelings during the past week. For each of the following statements, please respond as to whether you felt that way: Rarely or Not At All, Some of the time, Often, or Most of the Time. This is a measure of your feelings so there are no right or wrong answers. If you do not understand a question, answer it how you best understand the question.*

1	2	3	4	9
Rarely or Not at All	Some (1-2 days)	Often (3-4 days)	Most of the Time (5-7 days)	Not Applicable

During the **past week**.

2. I was bothered by things that don't usually bother me. \_\_\_\_\_
3. I did not feel like eating, my appetite was poor. \_\_\_\_\_
4. I felt that I could not shake the blues even with help from my family or friends. \_\_\_\_\_
5. I felt that I was just as good as other people. \_\_\_\_\_
6. I had trouble keeping my mind on what I was doing. \_\_\_\_\_
7. I feel depressed. \_\_\_\_\_
8. I felt that everything I did was an effort. \_\_\_\_\_
9. I felt hopeful about the future. \_\_\_\_\_
10. I thought my life had been a failure. \_\_\_\_\_
11. I felt fearful. \_\_\_\_\_



- 12. My sleep was restless. \_\_\_\_\_
- 13. I was happy. \_\_\_\_\_
- 14. I talked less than usual. \_\_\_\_\_
- 15. I felt lonely. \_\_\_\_\_
- 16. People were unfriendly. \_\_\_\_\_
- 17. I enjoy life. \_\_\_\_\_
- 18. I had crying spells. \_\_\_\_\_
- 19. I felt sad. \_\_\_\_\_
- 20. I felt that people dislike me. \_\_\_\_\_
- 21. I felt that people disliked me. \_\_\_\_\_

For Question 22, please use the following scale

1	2	3	4	9
Rarely or Not at All	Some	Often	Most of the Time	Not Applicable

- 22. I have felt depressed or sad in the **past year**. \_\_\_\_\_
- 23. Interviewer's code: \_\_\_\_\_
- 24. Date completed (mo/day/yr) \_\_\_\_\_/\_\_\_\_\_/\_\_\_\_\_

## THE STRONG HEART STUDY II

### SPIELBERGER - AX

1. How was the questionnaire administered? \_\_\_\_\_  
 1=By interviewer, 2=By self, 3=Refused

*A number of statements which people have used to describe themselves when they feel angry or furious are given below (Q2-Q21). Please read each statement and then indicate how often you feel or act in the manner describe when you are angry. This is a measure of your feelings; so there are no right or wrong answers.*

0	1	2	3
Rarely	Sometimes	Often	Almost
Or Never		or Always	Always

#### When I feel angry....

- |     |   |       |
|-----|---|-------|
| 2.  | I control my temper.  | _____ |
| 3.  | I express my anger.   | _____ |
| 4.  | I keep my feelings to myself.   | _____ |
| 5.  | I make threats I don't really mean to carry out.                                | _____ |
| 6.  | I withdraw from people when I'm angry.  | _____ |
| 7.  | I give people "the silent treatment" when I'm angry.                            | _____ |
| 8.  | I make hurtful remarks to others.   | _____ |
| 9.  | I keep my cool.   | _____ |
| 10. | I do things like slam doors when I'm angry.                                     | _____ |
| 11. | I boil inside, but don't show it.   | _____ |
| 12. | I argue with others.  | _____ |
| 13. | I hold grudges that I don't tell anyone about.                                  | _____ |
| 14. | I strike out (emotionally or physically) at whatever makes my angry.            | _____ |
| 15. | I am more critical of (judge or find fault with) others than I let people know. | _____ |
| 16. | I get angrier than I usually admit.   | _____ |

17. I calm down faster than most people. \_\_\_\_\_
18. I say mean things. \_\_\_\_\_
19. I am irritated (frustrated, annoyed) much more than people are aware of. \_\_\_\_\_
20. I lose my temper. \_\_\_\_\_
21. If someone bothers (frustrates, irritates) me, I am likely to tell him/her. \_\_\_\_\_
22. Interviewer's code: \_\_\_\_\_
23. Date completed (mo/day/yr) \_\_\_\_\_ / \_\_\_\_\_ / \_\_\_\_\_

## THE STRONG HEART STUDY II

## COOK MEDLEY

1. How was the questionnaire administered? \_\_\_\_\_  
 1=By interviewer, 2=By self, 3=Refused

*These next questions (Q23- Q30) are about how you think about other people. Although we cannot really know what people would think or do unless they tell us, we would like to know your opinion as to whether you think each of the following statements is "True or False." Once again, this is your opinion, so there is no right or wrong answer.*

0  
True

1  
False

2. No one cares much about what happens to me. \_\_\_\_\_
3. It is safer to trust nobody. \_\_\_\_\_
4. \_\_\_\_\_ Most people would lie to get ahead. \_\_\_\_\_
5. Most people inwardly dislike putting themselves out to help other people. \_\_\_\_\_
6. Most people will use unfair means to gain an advantage rather than lose it. \_\_\_\_\_
7. Most people are honest mainly through fear of being caught. \_\_\_\_\_
8. I often wonder what hidden reason another person may have for doing something nice for me. \_\_\_\_\_
9. Most people make friends because friends are likely to be useful to them. \_\_\_\_\_
10. Interviewer's code \_\_\_\_\_
11. \_\_\_\_\_ Date completed (mo/day/yr) \_\_\_\_\_ / \_\_\_\_\_ / \_\_\_\_\_

## THE STRONG HEART STUDY II

### *Quality of Life*

How was the questionnaire administered?  
(1=By interviewer, 2=By self, 3=Refused)

1. In general, would you say your health is: **(Circle One Number)**
- |           |   |
|-----------|---|
| Excellent | 1 |
| Very Good | 2 |
| Good      | 3 |
| Fair      | 4 |
| Poor      | 5 |
2. **Compared to one year ago**, how would rate you health in general now?  
**(Circle One Number)**
- |                                       |   |
|---------------------------------------|---|
| Much better now than one year ago     | 1 |
| Somewhat better now than one year ago | 2 |
| About the same                        | 3 |
| Somewhat worse than one year ago      | 4 |
| Much worse than one year ago          | 5 |

The following items are about activities you might do doing a typical day. Does **your health now** limit you in these activities? If so, how much?

**(Circle One Number on Each Line)**

- |   |   |   |   |
|---|---|---|---|
| 3. <b>Vigorous activities</b> , such as running, lifting heavy objects, participating in strenuous sports.  | 1 | 2 | 3 |
| 4. <b>Moderate activities</b> , such as moving a table, pushing a vacuum cleaner, bowling, or playing golf. | 1 | 2 | 3 |
| 5. Lifting or carrying groceries.   | 1 | 2 | 3 |
| 6. Climbing <b>several</b> flights of stairs.   | 1 | 2 | 3 |
| 7. Climbing <b>one</b> flight of stairs.  | 1 | 2 | 3 |
| 8. Bending, kneeling, or stooping.  | 1 | 2 | 3 |
| 9. Walking <b>more than a mile</b> .  | 1 | 2 | 3 |
| 10. Walking <b>several blocks</b> .   | 1 | 2 | 3 |
| 11. Walking <b>one block</b> .  | 1 | 2 | 3 |
| 12. Bathing or dressing yourself.   | 1 | 2 | 3 |
-

Questions adopted from the RAND 36-Item Health Survey 1.0.  
Strong Heart Study II 10/20/93

Quality of Life

During the **past 4 weeks**, have you had any of the following problems with you work or other regular daily activities **as a result of you physical health?**

(Circle One Number on Each Line)

	Yes	No
13. Cut down the <b>amount of time</b> you spent on work or other activities.	1	2
14. <b>Accomplished less</b> than you would like.	1	2
15. Were limited in the kind of work or other activities.	1	2
16. Had <b>difficulty</b> performing the work or other activities (for example, it took extra effort)	1	2

During the **past 4 weeks**, have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)?

(Circle One Number on Each Line)

	Yes	No
17. Cut down the <b>amount of time</b> you spent on work or other activities.	1	2
18. <b>Accomplished less</b> than you would like.	1	2
19. Didn't do work or other activities as <b>carefully</b> as usual.	1	2
20. During the <b>past 4 weeks</b> , to what extent has you physical health or emotional problems interfered with your normal social activities with family, friends, neighbors, or groups?	(Circle One Number)	
	Not at all	1
	Slightly	2
	Moderately	3
	Quite a bit	4
	Extremely	5
21. How much <b>bodily</b> pain have you had during the <b>past 4 weeks?</b>	(Circle One Number)	
	None	1
	Very mild	2
	Mild	3
	Moderate	4
	Severe	5
	Very severe	6

22. During **the past 4 weeks**, how much did pain interfere with your normal work (including both work outside the home and housework).

**(Circle One Number)**

- |              |   |
|--------------|---|
| Not at all   | 1 |
| A little bit | 2 |
| Moderately   | 3 |
| Quite a bit  | 4 |
| Extremely    | 5 |

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Quality of Life

These questions are about how you feel and how things have been with you during the **past 4 weeks**. For each question, please give the one answer that comes closest to the way you have been feeling.

How much of the time during the **past 4 weeks**. **(Circle One Number on Each Line)**

	All of the Time	Most of the Time	A Good Bit of the Time	Some of the Time	A Little of the Time	None of the Time
23. Did you feel full of pep?	1	2	3	4	5	6
24. Have you been a very nervous person?	1	2	3	4	5	6
25. Have you felt so down in the dumps that nothing could cheer you up?	1	2	3	4	5	6
26. Have you felt calm and peaceful?	1	2	3	4	5	6
27. Did you have a lot of energy?	1	2	3	4	5	6
28. Have you felt downhearted and blue?	1	2	3	4	5	6
29. Did you feel worn out?	1	2	3	4	5	6
30. Have you been a happy person?	1	2	3	4	5	6
31. Did you feel tired?	1	2	3	4	5	6

32. During the **past 4 weeks**, how much of the time has your **physical health** or **emotional problems** interfered with your social activities (like visiting friends, relatives, etc.)?

**(Circle one Number)**

- |                          |   |
|--------------------------|---|
| All of the time          | 1 |
| Most of the time         | 2 |
| Some of the time         | 3 |
| A little bit of the time | 4 |
| None of the time         | 5 |

How TRUE or FALSE is each of the following statements for you?

**(Circle One Number on Each Line)**

Definitely True	Mostly True	Don't Know	Mostly False	Definitely False
-----------------	-------------	------------	--------------	------------------

- |   |   |   |   |                             |   |
|---|---|---|---|-----------------------------|---|
| 33. I seem to get sick a little easier than other people. | 1 | 2 | 3 | 4                           | 5 |
| 34. I am as healthy as anybody I know.                    | 1 | 2 | 3 | 4                           | 5 |
| 35. I expect my health to get worse.                      | 1 | 2 | 3 | 4                           | 5 |
| 36. My health is excellent.                               | 1 | 2 | 3 | 4                           | 5 |
| 37. Interviewer's code                                    |   |   |   | ___ / ___ / ___             |   |
| 38. Date (mo/day/yr)                                      |   |   |   | ___ ___ / ___ ___ / ___ ___ |   |

Strong Heart Study II 10/20/93



## VITA

**Brian David O'Leary**

Address: CMR 427 Box 244  
 APO AE 09630  
 Phone: 39-0444-89-3333  
 Email: [brian.d.oleary@us.army.mil](mailto:brian.d.oleary@us.army.mil)  
 Date of Birth: 25 December 1973

**Education**

Utah State University Ph.D. in Professional-Scientific Psychology	Projected completion: 2007
Utah State University M.S. in Psychology	Completed: 2001
South Dakota School of Mines and Technology B.S. in Interdisciplinary Science, emphasis in Psychology	Completed: 1996
Fort Sill Officer Advanced Course	Completed: 2001
Fort Sill Officer Basic Course	Complete: 1997

**Clinical Experience**

173<sup>rd</sup> (ABN) Brigade Combat Team, Vicenza, Italy June 2004 – Current  
 BDE Psychologist, responsible the overall mental health of 10,000 soldiers and their families. Responsible for the planning and execution of combat stress control missions and supervises the brigade mental health section. Examines, diagnoses and treat individuals suffering from emotional or mental illness, situational maladjustment, combat stress reactions, and misconduct stress behaviors. Serves as a consultant to commanders on issues regarding human behavior and mental health. Deployed to Afghanistan for one year in support of Operation Enduring Freedom. Currently preparing Brigade Combat Team for second deployment to Afghanistan.

Walter Reed Army Medical Center, Washington D.C. June 2003 – June 2004  
 Psychology Resident, successfully completed all requirements of a nationally recognized professionally accredited Clinical Psychology Residency Program. Conducted psychological assessments, individual and group psychotherapy, crisis intervention while providing services at the Behavioral Health Clinics at WRAMC, Ft. Meade, Psychiatry Consultation Liaison Service, Inpatient Psychiatry and Neuropsychology. Primarily provided services to wounded soldiers returning from Operation Enduring Freedom and Operation Iraqi Freedom. Supervisor: COL Larry James, Ph.D.

- Bear River Mental Health Sept 2001 – May 2003  
Community Mental Health Center, Adult outpatient therapist, Provided individual and group therapy in a outpatient, hospital, nursing home, prison and crisis setting, conducted psychological evaluations, assisted with competency evaluations, gained experience in working with multiple federal and state agencies. Supervisor: Russ Seigenberg, Ph.D.
- Brigham City Community Hospital, Cardiac Rehabilitation May 2002 – Dec 2003  
Performed health psychology interventions, taught classes in the role of psychosocial variables and health outcome, stress reduction, performed individual therapy on an as needed basis. Supervisor: Kevin Masters, Ph.D.
- Utah State University Counseling Center Sept 1 1999 – May 1 2000  
Counseling Center rotation for doctoral training program, Provided individual therapy to student population at Utah State University. Supervisor: Gwena Couillard, Ph.D. and Jeff Dolin, M.C.
- Clinical Services, Center for Persons with Disabilities July 1 1998 – July 1 1999  
Conducted psychological and educational evaluations for children and adults. Provided both family and individual therapy to clients. Gained skills in achievement, cognitive, socialemotional, personality, language and attention assessment instruments. Also gained experience in child and family based therapy. Supervisors: Pat Truhn, Ph.D. and Phyllis Cole, Ph.D.
- Ogden School District Sept 1 1998 – May 1999  
School Psychology rotation for doctoral training program, Conducted educational evaluation, consulted with parents and teachers regarding identified problems with children. Provided special education assessments and limited therapy to emotionally disturbed children. Supervisor: Cher King, Ph.D.
- Utah State Psychology Community Clinic Jan 1997 – May 2002  
Department's Training Clinic, Two training rotations as well as continued participation in the clinic. Provided therapy to individuals in the Cache Valley Area. Supervisor: Kevin Masters, Ph.D. Also participated as a co-therapist with Carolyn Barcus Ed.D., serving a long-term sexual abuse victims group.
- Black Hills Children's Society Sept 1995 – May 1996  
Counselor working with sexually abused children in an inpatient treatment facility.

### Teaching Experience

- Psy 3660, Educational Psychology Fall 2000 – Spring 2001  
T.A. Lectured, graded assignments
- Psy 1010 General Psychology Fall 1999 – Spring 2000  
T.A. Lectured, supervised 20 labs and 13 lab instructors.
- Psy 4210, Theories of Personality Winter 1997, Winter, Spring 1998  
T.A. Lectured, graded assignments
- Psy 520 Intro to Counseling Fall 1997, Fall 1999  
T.A. Limited lecture experience, graded papers

### Research Experience

Investigator with MedStar Research Institute, Washington D.C., assist with the planning of psychosocial instruments, analysis of psychologically related variables as they apply to epidemiological studies. Supervisor: Dr. Barbara Howard

Received junior investigator training grant from the National Institute of Health to implement the psychosocial aspect of a large cardiovascular and genetic study in a Native American population.

Worked with Missouri Breaks Research, on the Strong Heart Study, field work collecting data on the Cheyenne River, Standing Rock, Rosebud, and Pine Ridge Sioux Tribes.

### Publications

- O'Leary, B. & Barcus, C. (2003). *Suicide: Assessment and Intervention among Native Americans.* Professional presentation given to members of various Lakota tribal agencies from across South Dakota. Deadwood, SD.
- O'Leary, B., Red Willow, F., & O'Leary, C. (2003) *Leadership: an introduction to basic leadership skills, methods and ethics*, Profession presentation given to community members from various tribal agencies from across South Dakota, Rapid City, SD.
- O'Leary, B., Masters, K., & Howard, B. (2002). The role of Psychosocial Factors and Their Relationship to Type-2 Diabetes Mellitus Outcome among the Strong Heart Study Cohort. Symposium conducted at the Tenth Annual National Heart, Lung and Blood Institute Cardiovascular Minority Research Supplement Awardees Session held in conjunction with the 75<sup>th</sup> Scientific Sessions of the American Heart Association, Chicago, IL.
- O'Leary, B., Masters, K., & Howard, B. (2001). Psychosocial Factors and Their Relationship to Type-2 Diabetes Mellitus Outcome among the participants of the Strong Heart Study. Symposium conducted at annual national convention and Society of Indian Psychologists, Logan, UT.
- O'Leary, B. (2001). *Psychosocial Variables and their Relationship to Diabetic Outcome Among the Strong Heart Study Cohort.* Unpublished masters thesis, Utah State University, Logan.

### Other Relevant Experiences

- United States Army Sept 2002 – May, 2003  
 Armed Forces Health Professions Scholarship Program recipient S-6, 1/145<sup>th</sup> FA
- Nov 2001 – May 2002
- Staff Officer for the 1/145<sup>th</sup> BN FA, Responsible for planning communications related issues for a battalion of 560 men, assisted S-3 in planning and executing combat missions. Activated for homeland security during the 2002 Olympics in Salt Lake City, UT. Highest achieved rank: O-3 Captain

- XO, B Battery, 1/148<sup>th</sup> FA Nov 2000 – Nov 2001  
Executive Officer for B Battery, 1/148<sup>th</sup> FA, Responsible for the training and welfare of 150 soldiers and \$23 million equipment. Gained experience in administration, planning, public speaking and leadership.
- CO, Det 3 HHB 1/148<sup>th</sup> FA Dec 1998 – Nov 2000  
Commander of Det 3 HHB 1/148<sup>th</sup> FA, Responsible for the training and welfare of 60 soldiers and \$15 million equipment. Gained experience in administration, planning, public speaking and leadership.
- FSO, 1/116<sup>th</sup> Cav Brigade May 1996 – Nov 1998  
Fire Support Officer of the 1/116<sup>th</sup> Brigade, served as the Field Artillery liaison to both armor and infantry company-sized elements
- 91B May 1993 – May 1996  
Army Combat Medic, highest achieved rank E-5 sergeant

#### **Professional Organizations / Honors**

American Psychological Association  
APA Division 19  
WICHE Scholar  
Emergency Medical Technician, National Registry  
Saint Barbara's Medal  
Bronze Star Medal  
Army Commendation Medal  
Army Achievement Medal (2)  
Army Reserve Component Achievement Medal (2)  
National Defense Service Medal (2)  
Afghanistan Campaign Ribbon  
Global War on Terrorism Service Ribbon  
Army Service Ribbon  
Overseas Service Ribbon (3)

References available upon request