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The comparison of physical fitness between Type Ii diabetics and the apparently healthy population

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The Comparison of Physical Fitness between Type II Diabetics and the Apparently Healthy Population

by

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of the requirements for the degree of
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The thesis of Joan K. MacDonald for the degree of

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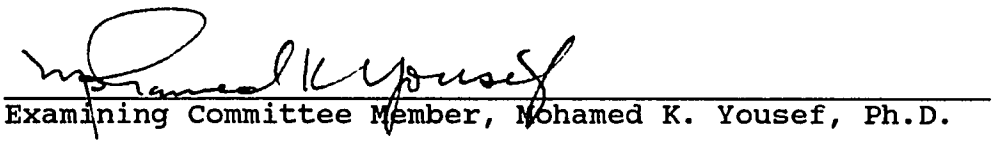

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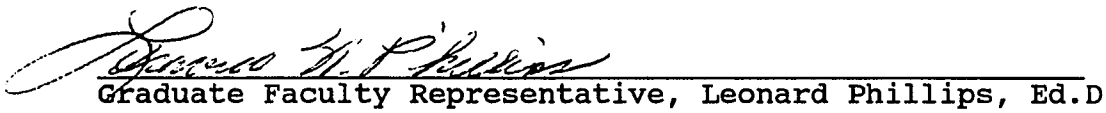

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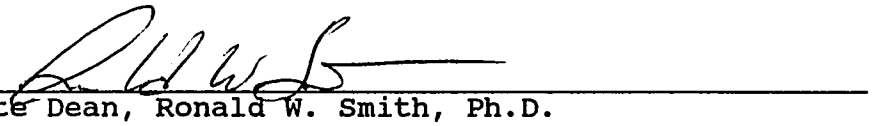

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Abstract

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COMPARISON OF PHYSICAL FITNESS BETWEEN TYPE II DIABETICS AND THE APPARENTLY HEALTHY POPULATION

Director of Thesis: Lawrence A. Golding, Ph.D.

Physical fitness was measured in a Type II diabetic population and compared to apparently healthy men and women. Eighty-eight female Type II diabetics, aged over 40 years were compared to 2,008 normal females of the same age, and 138 male Type II diabetics were similarly compared to 2,271 normal males. The National YMCA physical fitness test battery was used to measure fitness which consisted of flexibility, muscular strength and endurance, cardiorespiratory fitness, body composition, and resting values of heart rate and blood pressure. Differences in the mean values of each parameter were tested for significance ($p \leq 0.05$) using the $Z\ \bar{x}$ test. Diabetic females were significantly heavier, stronger in the upper body and had higher resting heart rates and blood pressures. Their recovery heart rates and abdominal strength were poorer than normal females. Male diabetics also were significantly heavier and had higher heart rates and blood pressures than normal males. Their recovery heart rates and abdominal strength were poorer than normal males. There was no

significant difference between normals and diabetics in percent body fat or flexibility. Although significant differences were found in $\dot{V}O_2$ max and PWC max in both males and females, the results were conflicting. This was believed due to measurement errors, and consequently, it is too premature to make a statement concerning this measurement of cardiorespiratory fitness. Since significant differences in most fitness variables existed, it must be assumed that Type II diabetics do not belong to the apparently healthy population fitness wise, therefore, norm tables should be developed for Type II diabetics.

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CHAPTER 1

Introduction

Diabetes mellitus, a syndrome of insulin deficiency manifested by abnormalities in the metabolism of carbohydrates, fats, and proteins, is the third leading cause of death and the primary cause of blindness in the United States (Mazzaferri, 1986; Guthrie and Guthrie, 1982). There are currently more than 10 million diabetics in this country and it is predicted that by 1990, 20 million people will have the disease (National Commission Reports, 1975). The nearly incomprehensible mortality rate of several hundred thousand deaths per year associated with diabetes and its complications (Guthrie and Guthrie, 1982; Krolewski and Warram, 1985) and the impact on related economic costs, underscore a desperate need to either find a cure or learn how to properly control the disease.

Although physical exercise is not a panacea, its role in preventing certain diseases and as a therapeutic agent in others has long been recognized. Although still controversial, the value of exercise in the treatment of diabetics has been emphasized for more than one thousand years (Kemmer and Berger, 1986). However, it has only been within the last

decade that research in this area has accelerated, with the vast majority of it being centered around the metabolic adaptations or modifications to exercise training. Little research has been completed establishing guidelines for exercise in this population, and as such, the current ones are often inadequate.

Nationally acceptable guidelines are available for the apparently healthy individuals who wish to begin an exercise program, as extensive baseline data have been accumulated for this population (Golding et al., 1982; Golding et al., in press). After receiving proper medical approval, the apparently healthy individuals may complete a battery of standardized tests designed to measure their exercise tolerance and cardiorespiratroy reserve capacity. The results can then be compared to norm tables and an individual's fitness level, relative to a specific sex and age group, can be determined. Exercise programs can subsequently be recommended, giving the appropriate kind of exercises at the proper intensity. However, it is not known at this time if safe and effective exercise guidelines can be established for the diabetic based on these nationally established norms for the apparently healthy population.

STATEMENT OF THE PROBLEM

Diabetes is often associated with additional medical complications such as atherosclerosis, nephrosclerosis, neuropathy and other cardiovascular changes (Legg and Harawi, 1985) and as such, the physical capacity of the diabetic may be lower than that of the normal population. If this is true, prescribing exercise programs based on the healthy population may not be entirely appropriate. The purpose of this study was to determine whether Type II diabetics have a fitness level different from that of the apparently healthy population. If found to be different, norm tables specific to the diabetic population should then be developed.

STATEMENT OF NEED AND SIGNIFICANCE

The value of exercise has long been recognized in the treatment and control of the diabetic, especially when combined with proper diet and insulin control (Ruderman and Schneider, 1986). Universally acceptable exercise programs, however, do not currently exist for this population. In order to diminish the mortality and morbidity rate associated with this disease, there is an obvious need for the development of safe, proper and effective exercise

programs. The first step, however, in designing any program should be to determine the fitness level.

LIMITATIONS AND/OR ASSUMPTIONS

In all studies, there are unavoidable limitations that may restrict the inferences resulting from the collected data. Additionally, certain assumptions must be made about the subjects and the procedures that may or may not be true. The following are the most apparent limitations and assumptions encountered in this study.

1. This study was limited to determining the fitness level of Type II (NIDDM) diabetics and as such, no inferences should be made about the Type I (IDDM) diabetics.

2. There were no time restraints imposed on the actual testing. The subjects were instructed to eat, drink, sleep as usual, and to take their medication as prescribed. They were asked, however, to refrain from smoking for at least two hours prior to testing.

3. Circadian rhythms and their effect on performance were not be considered during the study.

4. Since the majority of type II diabetics are over the age of 40, there will be an insufficient number of younger subjects to supply data for new norm

tables if developed. This will also limit the inferences that can be made to the entire population.

5. It was assumed that the subjects obtained were a true representation of the type II diabetic. This, however, may not be the case, as all subjects were volunteers, and in so being, possibly represent a more highly motivated group than the average type II diabetic.

DEFINITIONS

1. DIABETES--defined as a variety of disorders characterized by polyuria (excessive urination). It is derived from the Greek work meaning "to siphon or to pass through".

2. MELLITUS--a general term used to identify a group a syndromes stemming from abnormal glucose oxidation and utilization. It is derived from the Latin word for "honey" and refers to the urine being sweetened by sugar.

3. TYPE I DIABETIC--also called insulin-dependent diabetes mellitus (IDDM), or formerly known as ketosis-prone, juvenile, unstable, or brittle diabetes. The insulin-producing cells of the pancreas fail to produce enough insulin to maintain normal levels of blood sugar. Lifelong

exogenous insulin injections are, therefore, required to maintain normal metabolic function.

4. TYPE II DIABETIC--also called non-insulin-dependent diabetes (NIDDM), or formerly known as adult-onset, maturity-onset, ketosis-resistant, or stable diabetes. Insulin is generally produced in sufficient quantities, but because of a malfunction that is usually associated with obesity, the system cannot effectively utilize this insulin to control the blood sugar. The majority of type II diabetics can be controlled by diet and/or oral medications. Some, however, may temporarily require exogenous insulin to achieve control, especially while they are obese.

5. GLUCOSE--a simple sugar providing the chief source of energy for the body cells. The metabolic rate of glucose is controlled by insulin.

6. GLYCOGEN--a polysaccharide considered to be the chief carbohydrate storage material in animals. It is formed and stored in the liver and muscles.

7. GLYCOGENOLYSIS--the conversion of glycogen to glucose in the liver.

8. GLUCONEOGENESIS--the synthesis of glucose from noncarbohydrate sources such as amino acids and glycerol usually stimulated by cortisol and other

glucocorticoids and tetraiodothyronine (T4). This synthesis generally occurs in the liver and kidneys.

9. KETOGENESIS--the formation of ketone bodies that are the normal metabolic products of lipid and pyruvate within the liver. These are normally oxidized by the muscles.

10. INSULIN--the major fuel-regulating hormone produced in the beta cells of the pancreas that is secreted into the blood in direct response to blood glucose or amino acid concentrations. It facilitates the uptake, utilization, and storage of glucose, fat, and amino acids; it increases the synthesis of glycogen and fatty acids in the liver, of glycerol and fatty acids in adipose tissue, and of protein and glycogen in muscle tissue; it inhibits glycogenolysis, gluconeogenesis, and ketogenesis in the liver, lipolysis in adipose tissue; and decreases protein catabolism and amino acid output in muscle tissue (Sherwin et al., 1978).

CHAPTER 2

Review of the Related Literature

Historical Aspects of Diabetes Mellitus

The earliest knowledge of diabetes can be traced to 1500 B. C. when an illness characterized by the passage of much urine was described by the German archaeologist, Papyrus Ebers (Cahill, 1985). The disease was named diabetes, meaning siphon, in the 2nd Century by Aretaeus who also provided the first clinical description as cited below:

> Diabetes is a wonderful affection...being a melting down of the flesh and limbs into urine...the patients never stop making water, but the flow is incessant as if from the opening of aqueducts...the patient is short lived...for the melting is rapid, the death speedy.
>
> (Aretaeus, 2nd century)

Many centuries passed with little progress made in the understanding of the disorder. Finally in 1869, the German physician Paul Langerhans discovered "islands" within the pancreas, although he was unsure of their physiological function. This, however, eventually lead to the next major discoveries by the German scientists von Mering and Minkowski and the American scientist Opie early in the 19th Century. The results of their works implicated the beta cells in the pancreas as the origin of the disease. The greatest scientific breakthrough occurred in 1921 when

the Canadian physician Fredrick Banting and his graduate assistant, Charles Best were able to lower the blood glucose levels in diabetic dogs by administering prepared extract of pancreas. This resulted in a Nobel Prize and to a reversal in the morbid outcome of the disease by treating diabetics with exogenous insulin (Poulsen, 1967).

Classification of Diabetes Mellitus

Cahill (1985) stated that diabetes mellitus "is a grouping of anatomic and chemical problems resulting from a number of factors in which an absolute or relative deficiency of insulin or its function usually is present" (page 3). The disease can be classified through an entire spectrum of clinical symptoms ranging from a total lack of insulin to a slightly diminished functional ability of the insulin. It has, however, historically been classified into the two main groups of "juvenile-onset" and "maturity-onset" diabetes.

In the juvenile-onset diabetic, insulin is almost always totally lacking and those primarily affected are children or adolescents. Without insulin, death is probable within a few days from the ketoacidosis resulting from the impaired carbohydrate metabolism. Consequently, this is also referred to as

"ketosis-prone" or "insulin-dependent" diabetes mellitus (IDDM). It is currently more commonly referred to as Type I diabetes (Mazzaferri, 1986).

The maturity-onset, or Type II, diabetic, primarily has a deficiency in the functional ability of the insulin, not necessarily a reduction in quantity. In many cases there is an increase in the insulin level which is believed to be due to the obesity and/or lack of exercise that often accompanies this particular form of diabetes (Cahill, 1985). Ketoacidosis and death are unlikely to occur even in the absence of exogenous insulin. Consequently, this is called "non-insulin dependent diabetes mellitus" (NIDDM) although insulin may be required for control and regulation. Also as implied by the name, maturity-onset diabetes is usually manifested during the later years of life (50s or 60s).

Interrelationships of Fuels and Hormones in the Apparently Healthy Population

In order to understand the defective metabolism resulting from diabetes, the normal relationship between insulin and other hormones to fuel metabolism is reviewed from several sources.

Insulin-Glucose Mechanism

Glucose. Since the nervous system is virtually incapable of oxidizing any nutrients other than

glucose for energy, a constant supply of this sugar is necessary for continued survival. Fortunately, the composition of glucose can be altered following absorption so that the glucose not immediately used for energy may either be stored as glycogen in the liver and skeletal muscle or transformed into fat and stored in the liver and adipose tissue for future use in the postabsorptive state (Aoki, 1985). Through the actions of various hormonal and neuronal factors, the levels of this indispensible fuel are precisely regulated and controlled.

The molecular weight of glucose, although relatively small at 180, is actually too large to allow this sugar to cross the cellular membrane and enter the cytoplasm through the rapid process of simple diffusion. It, nevertheless, can be transported across the membrane through the mechanism of facilitated diffusion. This process can be increased by more than a factor of 10 when stimulated by insulin (Guyton, 1986). In fact, without insulin, only trace amounts of glucose could be transported into most cells of the body and death would be inevitable. The brain, being the major exception, will take up glucose in the absence of insulin.

Insulin. Insulin is secreted by the beta cells of the islets of Langerhans within the pancreas

primarily in response to an elevation in blood glucose. Specific amino acids (arginine and leucine) and beta adrenergic activity may also stimulate insulin secretion (Mazzaferri, 1986). The majority of this protein binds to specific receptors in the cell membrane of the target cells that subsequently become activated to initiate the insulin effects. These receptors are believed to be more than simple carriers of insulin from the extracellular to the intracellular space. Several investigators (Craig et al., 1981; LeBlanc et al., 1979) have shown that the insulin effect can be significantly modified by changing the numbers of receptors per cell and/or by altering the affinity of the receptors for insulin. These structures may, therefore, be the first site at which insulin exerts its effects.

The most important effect of insulin is to increase the uptake, storage, and utilization of glucose in most of the cells in the body, particularily in liver, muscle and adipose tissues. The insulin not used is expeditiously removed from the circulatory system and decomposed in the liver and to a lesser degree in the kidneys (Guyton, 1986).

The Liver and Insulin

The major effects of insulin on the liver are to stimulate glycogen synthesis and to inhibit glucose

output. This is accomplished through the following steps which are performed almost simultaneously upon the secretion of insulin: the enzyme, phosphorylase, that causes glycogen to be split into glucose is inhibited thereby preventing the breakdown of glycogen already stored in the liver; the activity of the enzyme, glucokinase, which causes the initial phosphorlation of glucose within the liver cells is increased causing the enhancement of glucose uptake; and the activity of the enzymes responsible for promoting glycogen synthesis, phosphofructokinase and glycogen synthetase, are increased thus increasing the glycogen concentration (Guyton, 1986).

Insulin also acts on the liver cells to help convert any excess glucose into fatty acids for storage as fat in adipose tissue. It additionally functions to inhibit gluconeogensis (hepatic synthesis of glucose from amino acids, glycerol, pyruvate and/or lactate) by decreasing the amount and the activity of the enzymes responsible for the creation of glucose from these noncarbohydrate sources (Guyton, 1986).

Once the blood glucose falls to low levels, as occurs within a few hours following a meal, all the above mentioned effects of insulin are reversed: insulin secretion is decreased; uptake of glucose from the blood is no longer enhanced thereby preventing

glycogen synthesis; the enzyme, phosphorylase, is activated causing glycogen to split into glucose phosphate; and the phosphate radical is removed from the glucose by the enzyme glucose phosphatase, allowing free glucose to diffuse back into the blood. The process of glycogenolysis, or the converting of glycogen into glucose is thus enhanced when blood glucose levels, and corresponding insulin levels, are low.

Muscle Tissue and Insulin

When insulin secretion is minimal, as in the postabsorptive state, the resting muscle does not generally use glucose as an energy source. The cellular membrane, in this situation, is nearly impermeable to glucose. Fatty acids, instead, provide the energy. This is evidenced from the fact that the resting respiratory quotient (RQ) for muscle is approximately 0.7, indicating that fat is almost the sole substrate used for oxidative metabolism in the postabsorptive state (Bjorkman, 1986). Resting muscle, however, will utilize glucose before fats during the first few hours following a meal as the insulin concentration is high at this time (Guyton, 1986).

The majority of the glucose taken into the resting muscle following a meal is stored in the form of muscle glycogen and used most frequently to provide

immediate energy during short spurts of anerobic activity through the mechanism of glycolysis (anaerobic conversion of glucose to lactate or pyruvic acid primarily to provide energy in the form of ATP to working muscles). Those carbohydrates stored as muscle glycogen are not readily available to the plasma glucose pool as the enzyme, glucose phosphatase, as mentioned above, is not present in muscle tissue as it is in liver tissue (Wahren, 1979).

Fat Metabolism and Insulin

The ultimate effect of insulin on fat metabolism is to enable fat to be properly stored. This is essentially accomplished through the promotion of fatty acid synthesis in the liver and to a lesser degree in the adipocytes themselves.

Glycogen synthesis within the liver becomes inhibited once the glycogen concentration reaches approximately 5 to 6 percent. Consequently any additional glucose in the liver will be transformed into fatty acids as acetyl-CoA, the substrate responsible for the synthesis of fatty acids, is produced from the glucose entering the glycolytic pathway (Guyton, 1986). Some of these fatty acids are retained in the liver to synthesize triglycerides, the majority of which are subsequently released into the blood in the lipoproteins. Once the triglycerides

reach the adipocytes, if insulin is present, they will be converted back into fatty acids by the action of the enzyme, lipoprotein lipase, so that they may be absorbed into the fat cell. Once inside the cell, they will be reconverted back into triglycerides for storage. Those fatty acids produced in the liver and not synthesized into triglycerides are simply transported to the adipose cells for storage.

Insulin also stimulates the synthesis of fatty acids within the adipose cells in a similar manner as observed in the liver cells. However, since the adipocytes only receive about one-tenth as much glucose as do the liver cells, the synthesis of fatty acids is drastically reduced here (Guyton, 1986). However, not all of the glucose entering the fat cell in response to insulin is used to synthesize fatty acids. Some of it is used to produce alpha-glycerophosphate from which the essential substance, glycerol, is formed. Glycerol is what binds with fatty acids in the adipose cells to produce the storage form of fats, the triglycerides.

In addition to the important function of synthesizing fatty acids, insulin inhibits the enzyme responsible for the hydrolysis of the triglycerides and the subsequent release of fatty acids into the

blood. This helps to maintain acceptable circulating blood levels of free fatty acids and glycerol.

Protein Metabolism and Insulin

Although the mechanisms are not completely understood, insulin serves two vital functions in the metabolism of proteins, namely the promotion of protein formation and the prevention of protein degradation. These are, in part, accomplished by the following actions of increased insulin: the uptake of certain amino acids into cells is increased; the ribosomes are stimulated to produce new proteins by increasing the translation of messenger RNA (ribonucleic acid); the catabolism of proteins is inhibited resulting in a decreased release of amino acids from the cells; and the rate of gluconeogenesis is decreased by decreasing the activity of certain enzymes (Guyton, 1986).

The Control of Insulin Secretion

Glucose. According to Zawalich (1985), the most important physiologic regulator of insulin secretion is glucose. This insulin-glucose mechanism provides a positive feedback control over the plasma levels of both insulin and glucose. A rise in blood glucose stimulates insulin secretion which in turn enhances the entry of glucose into most cells. This decreases

the blood glucose level which subsequently causes the beta cells to reduce their secretion of insulin.

Amino Acids. The amino acids, particularily arginine and leucine (Mazzaferri, 1986) have a similar effect on insulin secretion as does glucose. However, it has been shown that when amino acids are present without an increase in the glucose level, insulin secretion will be minimal. If, however, the amino acids are administered at the same time the blood glucose level is elevated, the insulin secretion may even double that normally seen with the glucose elevation alone. This helps to promote the active transport of amino acids into the cells and eventual new protein formation as discussed above.

Other hormones. Almost immediately after a meal is eaten, certain hormones are released from the gastrointestinal tract to effect a rise in the insulin level. These include gastrin, secretin, cholecystokinin, and gastric inhibitory peptide. They, in essence, act as early messengers, causing an initial rise in insulin in order to be ready for the increased glucose and amino acids to be forthcoming after absorption.

The insulin stimulating hormones such as glucagon, growth hormone, cortisol, epinephrine, and

others will be discussed in greater detail during the section on fuel metabolism and exercise.

Interrelationships of Fuels and Hormones in the Diabetic Individual

It should be evident from the previous discussion that the effects of insulin are far reaching, and that a deficiency of this hormone will be manifested by an impairment of carbohydrate, fat and protein metabolism. The spectrum of insulin's deficiencies, again, range anywhere from an absolute lack to a slight reduction in its functional ability, causing a wide range of complications from death to feeling tired.

Type I Diabetes

The demise of the beta cells have been implicated as being the sole cause for either a reduction in or a complete absence of insulin secretion leading to Type I diabetes. The actual cause of this destruction currently remains unknown. It has been hypothesized, however, that the damage may result from any one or from a combination of the following factors: genetic inheritance of a cell-mediated autoimmunity producing blood antibodies against pancreatic tissue; viral infection possibly producing lesions in the islet cells; and/or a simple deterioration of the cells due to age and/or obesity (Kahn, 1985).

Type II Diabetes

The reduction in the functional ability of insulin rather than its absence characterizes this type of diabetic. Currently, the most commonly accepted belief is that the reduced biologic effect is due to a reduction in the number of the target-tissue insulin receptors or to a reduction in the insulin sensitivity of the receptors (Guyton, 1986). The main underlying cause of both of these possible explanations is obesity, as approximately 85 percent of the adults with the disease are obese (Guthrie and Guthrie, 1982). Obesity, as explained by Guyton (1986), may cause the beta cells themselves to become less responsive to insulin and it may also decease the number and the sensitivity of the target receptors.

It has also been suggested that in some Type II diabetics, the deficiency in insulin action may be the result of the beta cells secreting a less active insulin molecule or that the normal insulin molecule is somehow rendered inactive during its transportation to the target cells (Mazzaferri, 1986).

Additionally, there seems to be a strong hereditary trait for Type II diabetes. This was supported by a study in which normoglycemic individuals with familial aggregation of Type II diabetes were compared to normal control subjects

(Berntorp and Lindgarde, 1984). All subjects were presented with oral glucose tolerance tests and simultaneously measured for insulin, C-peptide levels and submaximal exercise performance ability. The results showed that those with familial history had significantly lower insulin levels after 40 minutes of exercise and significantly lower maximal oxygen uptake values. These data suggest that the hereditary trait was manifested as a decreased sensitivity of the beta cells to glucose stimulation and as a lower fitness level.

Regardless of the type of diabetes, an absolute or relative lack of insulin will produce the following effects in varying degrees: (1) abnormal carbohydrate metabolism producing an abnormal rise in blood glucose (from 300--1200 mg/dl); (2) abnormal fat metabolism causing increased mobilization of fat and consequent increased deposit of lipids in the circulatory system; and (3) abnormal protein metabolism resulting in protein depletion of the tissues (Guyton, 1986). The diabetic essentially presents as a paradox in that their cells are starving even in the presence of abnormally high blood glucose levels.

Abnormal Carbohydrate Metabolism of Diabetes

Just as insulin increases the uptake, storage and utilization of glucose in nearly all the cells in the

body, a lack of this protein will produce the opposite effect. Glucose entry into most of the cells is diminished causing an elevation in the blood glucose concentration. Glycogenolysis is enhanced as the cells signal for food thus releasing more glucose into the blood. And without excess glucose in the cells, muscle glycogen will not be available to furnish immediate energy through glycolysis (Guthrie and Guthrie, 1982).

If the fasting glucose levels rise above 180 mg/dl of blood, the renal threshold is exceeded and glucose "spills" (Cahill, 1985) into the urine (glucosuria). This fasting hyperglycemia is indicative of an insulin deficiency; the higher the blood glucose concentration, the more severe the deficiency (Cahill, 1985). This in turn leads to an increase in thirst (polydipsia) and hunger (polyphagia), and to excessive urine output (polyuria) (Guyton, 1986).

Abnormal Fat Metabolism of Diabetes

In the absence of insulin, the fat cells attempt to provide the main energy substrate for almost all the cells except those in the brain. The fat cells become activated to hydrolyze the stored triglycerides and thus release large amounts of fatty acids and glycerol. Many of the free fatty acids are used for

energy but the majority of them diffuse into the liver cells where they are reconverted and stored as triglycerides.

Phospholipids and cholesterol are produced in the liver in response to the excessive fatty acids. They then may join some of the triglycerides, leave the liver and enter the blood stream in the lipoproteins. This elevation of the blood lipid concentration is responsible for the atherosclerosis present in those having a serious diabetic disorder (Guyton, 1986).

Also, in the absence of insulin, the increased fatty acids in the liver produce large amounts of acetyl-CoA which can be used, to some extent, to provide energy. The unused portion is condensed to form acetoacetic acid and released into the blood. Under normal circumstances, this is reconverted in the periphery to form acetyl-CoA for use as energy. However, this process is diminished when insulin is lacking, leading to a large accumulation of acetoacetic acid. Two additional acids are also formed in the liver from acetoacetic acid. These are beta-hydroxybutyric acid and acetone. Collectively, these three acids are referred to as the ketone bodies. When present in large quantities a state of "ketosis" results. These acids, especially acetoacetic acid and beta-hydroxybutyric acid,

generate large amounts of hydrogen ions leading to acidosis, coma and death if left untreated (Vignati, Asmal et al., 1985).

Abnormal Protein Metabolism of Diabetes

Without insulin, the following events occur: the active transport of many of the amino acids into the cells is inhibited; new protein formation is diminished; the amino acids are released from the cells at an increased rate, especially from the muscle cells; and gluconeogenesis is enhanced as the plasma amino acids are the primary substrate used for the production of new glucose, and these have drastically increased (Guyton, 1986). A lack of insulin, therefore, promotes the degradation of proteins and practically causes protein synthesis to cease. Protein degradation ultimately leads to a wasting away of tissue, or to the "melting down of the flesh and limbs into urine" (Cahill, 1985).

Exercise and Diabetes Mellitus

Fuel Regulation during Exercise in General

At the onset of exercise, metabolic fuels must be rapidly mobilized and redistributed throughout the body in order to provide energy for the working muscles. Since glycogen is stored in the muscles and does not have to be mobilized and recirculated before

use, it is the initial source of energy during exercise. It, however, is in limited quantity and other fuel substrates must be made available for continued work. The primary circulating fuels used by contracting muscles are glucose and free fatty acids with amino acids and ketone bodies being used to lesser degree (Richter and Galbo, 1986).

Free fatty acids provide the majority of energy to the muscles in the resting state. However, with the initiation of exercise, glycogen is utilized first. Then, depending on the intensity and duration of exercise, glycogen, plasma glucose and free fatty acids are used in varying combinations. Finally, if exercise is prolonged, the free fatty acids become the major energy-providing fuel (Zinman and Vranic, 1985). Plasma glucose concentration is, however, the limiting factor during prolonged exercise, as the body cannot exist soley on free fatty acids (Guyton, 1986). If the hepatic production of glucose through glycogenolysis and/or gluconeogenesis is so low that a hypoglycemic condition exists and if the muscle glycogen stores become depleted, working capacity decreases (Bergstrom and Hultman, 1967).

The normal physiologic responses to exercise of varying intensity and duration depend upon proper metabolic, hormonal, and neuronal regulation.

Diabetes represents a multifaceted disease in which regulatory control mechanisms have been altered, and as such, the physiologic responses to exercise may not be the same in these individuals as in healthy subjects. Although there are differences between the two main diabetic types, both have abnormal glucose homeostasis mechanisms potentially causing abnormal responses to exercise. The responses are primarily dependent upon the diabetic's current state of metabolic control and are discussed below.

Metabolic Responses of the Diabetic to Exercise

Muscle Glycogen. Three studies cited by Bjorkman (1986) have revealed that exercising diabetics utilize glycogen at the same rate as the apparently healthy population. They may also have the same muscle glycogen content, depending upon their state of metabolic control, a state directly related to the level of insulin (Wahren, 1979). Those who are in a state of chronic insulin deficiency, however, are likely to show a decreased glycogen content with abnormal metabolism and an impaired aerobic capacity. It has, in fact, been demonstrated that after 40 minutes of exercise, the metabolic profile of moderately insulin deficient diabetics was similar to that of non-diabetics who had exercised up to 4 hours (Wahren, Sato et al., 1984), indicating considerably

accelerated responses in this diabetic state. Insulin therapy has been shown to return decreased glycogen levels and the aerobic exercise capacity to that of healthy individuals (Yki-Jarvinen and Koivisto, 1983).

The main difference between muscle glycogen in the diabetic and in the normal individual is that the contribution of glycogen to the total exercising energy expenditure is less in the diabetic. This was indirectly evidenced from a study on insulin dependent diabetics whose muscle oxygen and substrate uptakes were measured during exercise (Wahren, Hagenfeldt et al., 1975). The measurement of arteriovenous glucose concentration differences across the leg in combination with the local RQ provided an indirect estimation of muscle glycogen utilization to total muscle energy turnover (Bjorkman, 1986). This study showed similar estimated glucose contributions to total oxidative metabolism (28% versus 25% in controls). However, the contribution from free fatty acids (FFA) was measured to be 60% higher in the diabetic group. Supporting this claim was the observation of a lower RQ in the diabetics. Greater plasma substrate levels noted in the diabetics (70% versus 52%) also support the claim of decreased muscle glycogen availability in diabetic subjects (Wahren, Hagenfeldt et al., 1975).

Hepatic Glucose Production. In normal subjects, total glucose utilization has been shown to increase up to threefold during exercise whereas the blood glucose concentration remains virtually the same in normal subjects (Wasserman and Vranic, 1986). This is because the liver has the ability to synthesize glucose from stored glycogen and other fuel substrates, providing a source of glucose replenishment at almost the same rate as muscle glucose utilization.

In normal subjects, the primary source of glucose replacement during exercise is hepatic glycogenolysis. However, when the glycogen stores become depleted as occurs during prolonged exercise, hepatic gluconeogenesis is stimulated to increase its role in glucose replacement, but it fails to completely compenstate for the decreased glycogen breakdown. The diminished splanchnic production of glucose then leads to lower plasma glucose concentrations and insufficient peripheral glucose uptake (Bjorkman, 1986).

Wahren, Hagenfeldt and Felig (1975) have observed that the hepatic glucose production in the resting state was similar in both insulin deprived diabetics and healthy subjects. A considerable increase in the contribution of gluconeogenesis to glucose production

was, however, demonstrated in exercising diabetics (from 25% in normal subjects to 40% in diabetic subjects). This was evidenced from a two-to-fivefold increase in gluconeogenic precursors (lactate, amino acids, and pyruvate) along with an increased uptake of oxygen and FFA during submaximal exercise in diabetics. This finding also helps to support the theory that the insulin deficient diabetic may show an accelerated response to exercise, possibly lowering aerobic endurance.

Glucose utilization. One study (Wahren, Felig, Ahlborg, and Jorfeldt, 1971) reported a 7 to 10-fold increase in leg muscle glucose uptake during mild to moderate bicycle riding (65 and 135 watts) and a 20-fold rise during strenuous work intensities (200 watts) in normal subjects. This was evidenced from a gradual widening in the femoral arteriovenous glucose concentration curve with exercise. Additionally, two of these same investigators, Jorfeldt and Wahren, demonstrated a similar rise in glucose uptake in the small muscles of the forearms during exercise (Wahren, 1979). The conclusion was that with brief periods of work (from 120 to 180 minutes depending upon whether the work is low intensity or strenuous, respectively) the contribution of glucose to the total oxidative metabolism gradually increased, suggesting a greater

importance of this substrate with duration of exercise. The muscle glucose, however, did fall in conjunction with the plasma glucose level with prolonged exercise (after approximately 2--3 hours) (Wahren, Felig, Ahlborg, and Jorfeldt, 1971).

Wahren, Hagenfeldt and Felig (1975) also demonstrated that the total glucose uptake by leg muscle during exercise in hyperglycemic, insulin-deprived diabetics was at least as great as in healthy controls. This could, however, be due to the fact that the initial blood glucose concentration was considerably higher in the diabetics thereby accounting for the relatively normal glucose uptake in the leg tissues. Lactate levels were significantly higher in these diabetics, suggesting incomplete oxidation of glucose through the glycolytic pathway. The amount of blood glucose contributing to total oxidative metabolism by the leg muscles was not found to differ measurably between the controls, mildly ketotic or nonketotic diabetic subjects. Oxygen uptake, however, was significantly higher in the ketotic group, again suggesting an increased use of additional energy substrates while in this diabetic state (Wahren, Hagenfeldt and Felig, 1975).

Hormonal Control of Responses to Exercise

Apparently Healthy Subjects. To provide for the increased need of fuel during exercise, certain hormonal responses have been characterized. The plasma insulin concentration decreases almost instantaneously with exercise through a suspected alpha-adrenergic stimulation of the beta cells and an indirect local release of catecholamines in the liver (sympathetic nervous system stimulation) that causes an instantaneous release of hepatic glucose (Galbo, 1986; Kemmer and Berger, 1986). This hypoinsulinemia is believed to play an important physiologic role in directly increasing hepatic glucose output and in facilitating the mobilization of FFA from adipose tissue. It may also inhibit the glucose uptake by non-exercising tissues (Richter and Galbo, 1986). The decrease in insulin apparently has little effect on glucose uptake by working muscles. Richter and Galbo (1986) refer to a study by Ploug et al. in which it was demonstrated that muscle contractions and insulin have additive effects on glucose transport and, consequently, only minute concentrations of insulin are needed to increase glucose uptake.

The question as to how the counter-regulatory hormones (catecholamines, glucagon, growth hormone and

cortisol) respond to exercise and the hypoinsulinemia remains somewhat unresolved. It is currently believed that the plasma glucagon levels do not change but that the circulating levels of catecholamines and growth hormones do increase with exercise (Bjorkman, 1986; Richter and Galbo, 1986). Conflicting data have shown that an exercise-induced rise in glucagon levels was immediately observed in sheep, rats, and dogs, whereas in humans, these levels did not increase until late in prolonged exercise when plasma glucose concentrations decreased (Richter and Galbo, 1986). It has been concluded that these differences are species related and that glucagon does not play the same important role in increasing hepatic glucose production in humans as it does in other species.

The circulating levels of catecholamines, norepinephrine and epinephrine, have been seen to vary exponentially with intensity of exercise, with norepinephrine levels significantly increasing at lower workloads than those required to cause a significant rise in epinephrine (Galbo, 1980). Richter and Galbo (1986) mention a study by Christensen and Galbo in which the increase in norepinephrine was correlated to the proportional increase in pulmonary arterial oxygen saturation commonly observed with increasing workloads. The

secretion of epinephrine was determined to be dependent upon the levels of norepinephrine as well as on the plasma glucose concentration. Specifically, if the glucose level was kept constant through continuous infusion, the epinephrine levels decreased. Other hormones, such as cortisol, growth hormone and adrenocorticotropic hormone (ACTH), have all been observed to increase with exercise. It is, however, currently believed that they do not play a major role in maintaining hepatic glucose production except during prolonged exercise (Kemmer and Berger, 1986).

Diabetic Individuals. In the type I diabetic, the plasma insulin levels are dependent upon the time and site of subcutaneous administration and the rate of absorption (Zinman, 1984). The levels can fluctuate from excessive to deficient. If the pre-existing insulin levels are high, less hepatic glucose will be produced and greater glucose clearance will occur leading to a lower plasma glucose concentration or to a hypoglycemic state. Conversely, in the insulin- deficient state, the plasma glucose levels will be elevated leading to a hyperglycemic and/or ketotic state. This occurs because the liver, due to an exaggerated exercise-induced increase in the counter-regulatory hormones, increases glucose production whereas the muscle tissue shows a

diminished uptake of glucose (Zinman and Vranic, 1985).

The majority of type II diabetics experience hyperglycemia due to an insulin resistance. This occurs even in the presence of elevated insulin levels that are present in most diabetics (due to obesity). The glucose level, however, is still greater than the insulin level thereby creating a hyperglycemic environment (Ruderman and Schneider, 1986). In a study by Galbo (1986), obese diabetics who participated in bicycle exercises showed a fall in plasma glucose while the insulin level remained the same. Obese control subjects, on the other hand, remained euglycemic and had a normal decrease in insulin concentration (Galbo, 1986).

Bjorkman (1986) referred to a study by Simonson et al. in which beta-adrenergic blockade in insulin-dependent diabetics inhibited the exercise-induced rise in glucose production, suggesting that beta-adrenergic stimulation is critical during exercise in these subjects. Galbo (1986) concluded that the catecholamine response to exercise is enhanced in insulin-deficient states such as in poorly controlled diabeates, and that this response will be depressed if the diabetic has autonomic neuropathy. He also found glucagon levels

to be higher in the insulin-deficient state compared to normal subjects.

Fitness Level and the Diabetic

Very few studies comparing the physical fitness level of diabetics to the normal population have been cited in the literature, and those that have been identified have compared only the Type I diabetics against the normal population. From those, it was concluded that this diabetic population, for not fully understood reasons, show a reduced ability to perform physical activities (Coram and Mangum, 1986).

The major reason for this decreased ability is suspected to be the cardiovascular changes that often accompany diabetes. Many additional complications that reduce the exercise capacity, such as microvascular diseases that induce retinopathy, nephropathy, and neuropathy are also often present (McMillan, 1979). A discussion follows on those factors believed to be responsible for decreasing exercise performance in diabetics, especially as related to the specific fitness categories of body composition, cardiovascular capacity, muscular strength and endurance, and flexibility.

Diabetes, Exercise, and Body Composition

The initial discovery of hyperinsulinemia in the type II diabetic by Berson and Yalow (Sherwin and Felig, 1978) was surprising until it was later discovered that obesity itself causes hyperinsulinemia with an accompanying reduction in insulin resistance (Sherwin and Felig, 1978; Stout, 1981). This resistance, which has been correlated to a reduction in insulin receptors within fat cells (Cooppan and Flood, 1985), increases the demand for insulin. In the diabetic, however, the defective beta cell function of the pancreas will prevent an adequate increase in insulin and, consequently, the deficient secretory mechanism will be recognized. Obesity has, therefore, been labeled as a "diabetogenic factor" (Cooppan and Flood, 1985).

Whether obesity itself can be called a major risk factor for developing coronary artery disease (CAD) remains questionable. Most of the epidemiologic data that has been assessed using multivariate analyses has lead to the conclusion that obesity is not a separate risk factor. However, Keen et al. (cited by Leland and Maki, 1985) noted a study showing an increased incidence of angina in an obese population that was otherwise matched to all other risk variables. Obesity has more frequently been associated with

inducing other major risk factors such as hypertension and increasing the levels of cholesterol and triglycerides (Cooppman and Flood, 1985), subsequently causing large vessel abnormalities (Stout, 1981). Therefore, any deleterious effect on exercise caused by obesity appears to be an indirect one.

The occurance of obesity in diabetes has been claimed to be approximately 75 to 85% in the type II diabetic (Mazzaferri, 1986; Kahn, 1985; Guthrie and Guthrie, 1982) whereas the Type I diabetic is normally associated with weight loss, especially during periods of poor control (Davidson, 1986).

Although it is generally accepted that the incidence of hypertension is increased in the diabetic (McMillan, 1979; Coram and Mangum, 1986; Christlieb, 1985), conflicting data has been reported. Ganda (1985) cited 5 studies in which diabetes was strongly related to hypertension and 2 which failed to show such a correlation.

Data from the Framingham study (Kannel and McGee, 1979) have provided evidence for the conculsion that obesity is associated with hyperlipidemia and low HDL-cholesterol (HDL-C) levels. HDL-C levels have, alternately, been shown to be inversely related to coronary disease (Nikkila, 1981). Numerous studies as mentioned by Coram and Mangum (1986) when comparing

diabetes and hyperlipidemia have, however, produced conflicting results. These will be mentioned in the discussion of cardiovascular capacity. The results, however, appear to be dependent upon the present state of metabolic control, type and severity of diabetes, sex and other factors.

It cannot presently be concluded that the reduced exercise capacity of diabetics is influenced by obesity itself, however, obesity is strongly related to other risk factors that have deleterious effects on exercise. It is well known that obese individuals will not perform as well during strenuous physical activity as their age and sex matched lean counterparts (Katch and McArdle, 1983). The same relationship is expected to occur between obese diabetics and lean normal subjects. However, if the obese diabetics have no additional risk factors and if the diabetes is well controlled, it is questionable as to whether their physical performance would differ significantly from their age, sex and weight matched normal counterpart. Given that the largest majority of type II diabetics are obese (Kahn, 1985; Mazzafferri, 1986; Davidson, 1986; Guthrie and Guthrie, 1982) and that many of them develop additional risk factors and complications, it seems reasonable to predict that their physical capacity

would be lower than their age and sex matched non-diabetic counterparts.

Cardiovascular Capacity and the Diabetic

Diabetics are known to have more coronary risk factors than the normal population, leading to a higher incidence of CAD, greater atherosclerotic involvement and more myocardial infarctions (MI) (Coram and Mangum, 1986). Brownlee (1985) has stated that because of the increased atherogenesis, diabetics have a 2-fold greater risk of developing CAD and stroke (CVA--cerebral vascular accident) than the normal population and a 3 to 4-fold greater chance of developing symptomatic peripheral arterial disease which generally manifests itself as nephropathy, retinopathy, and/or neuropathy. These conditions, either when present singly or in combination, cause a reduction in both maximum oxygen consumption and in oxygen delivery to the tissues, thereby decreasing the exercise performance capacity of the diabetic. Two main categories of diseases, macroangiopathy and microangiopathy, form the basis of classification for many of the cardiovascular complictions of diabetes.

Macroangiopathy. According to Ganda (1980), macrovascular disease (atherolsclerosis) that primarily manifests itself as a cardiovascular disorder accounts for approximately 75% of all

diabetic deaths, and it is not restricted to either type of diabetic. It is more prevalent in the Type II diabetic between the ages of 50 and 70, and it is observed with equal prevalence in both male and female diabetics. This finding is discordant with the normal populaton in which women have been shown to possess a degree of protection against arterial diseases (Schneider, Vitug et al., 1986), presumably due to an inhibitory effect of estrogens on atherosclerosis (Stout, 1981).

LDL-C Levels. There is strong evidence linking atherosclerosis to elevated plasma lipid levels, especially when considering total serum cholesterol concentrations and low density lipoproteins (LDL) (Ganda, 1980). In addition, independent plasma triglycerides have been labeled a risk factor for CHD by several investigators (Schneider, Vitug et al., 1986). Although never adequately shown, the increased CAD and other atherosclerotic vascular diseases associated with diabetes have frequently been attributed to the abnormal metabolism of lipoproteins (Nikkila, 1981). Triglycerides, very low-density lipoproteins (Nikkila, 1981), and plasma fatty acids (McMillan, 1981) are regularly elevated in diabetes, but there is no general agreement that these are responsible for the

increased incidence of CAD. As reported by Coram and Mangum (1986), there is conflicting data as to whether the LDL-C levels are increased, decreased, or constant when comparing diabetics to the normal population. One study reported continually high LDL-C levels; another reported high LDL-C levels only during periods of poor metabolic control in Type I diabetics; the levels were reported to be high in males but not in females in yet another study; and there were two reports showing no difference between diabetics and the apparently healthy population. These studies, again, indicate that this ratio may change depending upon the type and severity of the diabetes, glycemic control, metabolic profile, sex, and/or other factors.

HDL-C Levels. High density lipoprotein (HDL-C) levels, on the other hand, have been shown to have a strong inverse relationship to both the incidence and the severity of atherosclerotic disease (Nikkila, 1981). Researchers, however, do not agree on the HDL-C levels found in diabetics. Nikkila (1981) has proposed an explanation for the conflicting evidence in that HDL-C concentrations are based on the insulin levels and on the insulin sensitivity of the tissues. He found that Type I diabetics with moderate to good control, had HDL-C levels in the high normal range. The levels in the Type II diabetic were

similar to non-diabetic matched subjects but lower than the Type I diabetics. Those showing low HDL-C levels displayed advanced renal involvement, repeated periods of ketoacidosis, or excessively high triglyceride and uric acid levels. From this he suggested that perhaps all the excessive CAD noted in the diabetic population originates in this subgroup of diabetics. There appears to be a definite need for further investigation into the role of plasma lipids and their relationship to atherosclerotic disease in the diabetic population before concrete conclusions can be drawn.

Atherogenic Agents. Excessive insulin has recently been identified as a potential atherogenic source (Schneider et al., 1986). It has been linked to atherosclerosis through both clinical and epidemiologic evidence and through biological evidence. Three prospective studies, as referred to by Stout (1981), all showed that increased plasma insulin levels, independent of other major risk factors, were positively related to the development of CAD. He also noted the concentrations could even have a predictive value. Schneider et al. (1986) additionally referred to more than 20 studies showing a similar relationship.

Numerous animal studies showed that insulin concentrations correlated to lipid inflitration and deposition, glucose incorporation, and smooth muscle proliferation in arterial walls (Stout, 1981). These biologic factors are believed to play a major role in the development of atheromatous lesions. Ross and co-workers have established that the hallmark of atheromatous lesions is the deposition of lipids in the smooth muscle cell, and that the proliferation of the smooth muscle is the initiator and perpetuator of the fibrous plaque (Ganda, 1980).

Additional support for the positive association between insulin levels and atherosclerosis comes from Stout (1981). He found the following: lipid containing lesions, not associated with weight change or lipid level changes, were noted in the aortas of chickens following 19 weeks of insulin administration; experimental atherosclerosis in cholesterol-fed rabbits was reduced following insulin-secreting tissue ablation and it returned to the original atheromatous state upon treatment with insulin, and; progressive smooth muscle proliferation was noted in monkey cells exposed to increasing levels of insulin.

Other possible atherogenic agents have been purposed. Growth hormone (GH) levels are known to be higher in both the fasting and the post-glucose fed,

uncontrolled Type I diabetic as well as in the Type II diabetic. A study by Merimee (Ganda, 1985) showed that dwarfs who had deficiencies in GH were resistant to atherosclerosis, even though they were not resistant to developing diabetes. Catecholamines have also been incriminated as atherogenic agents, and increased concentrations of these are often seen in uncontrolled diabetes (Wasserman and Vranic, 1984).

Although diabetes is caused by either a relative or absolute insulin deficiency, this does not necessarily mean that low insulin concentrations are present. As previously stated, obesity itself is associated with high insulin levels, and, consequently, obese Type II diabetics have higher concentrations than the non-diabetics who are thinner. In the insulin-dependent diabetic, the administered daily dose of insulin is considerably greater than the normal pancreatic secretion, and, consequently, the concentration in the Type I diabetic is also often higher than in the non-diabetic. According to Stout (1981), only two studies comparing atherosclerosis to insulin levels in the diabetic population had been completed prior to 1981. These both, however, showed that insulin concentration had a direct effect on atherosclerosis.

Based on the above research, a critical area for further investigation is to determine whether reduced insulin levels will prevent or diminish the development of atherosclerosis. Professed methods of accomplishing the diminution of insulin include weight loss, regular physical exercise and a better method of controlling exogenous insulin administration so that it is more in keeping with the normal physiologic response (Stout, 1981).

Microangiopathy. The chances of becoming blind are 25 times greater in the diabetic population than in the normal one; chronic renal failure has a 17 times higher frequency in diabetics; and a higher incidence of motor, sensory and autonomic nerve impairment are seen in the diabetic (Brownlee, 1985). These three areas represent the main classification of microangiopathy and are related to the pathologic conditions of retinopathy, nephropathy and neuropathy, respectively. Miocroangiopathy is specific to the diabetic and is characterized by anatomical and functional changes in the basement membranes of capillaries and in the walls of arterioles and venules (Coram and Mangum, 1986), with the functional alterations preceeding the structural changes (McMillan, 1979). This distrubance in the microcirculation causes a relative tissue hypoxia, and

therefore, diabetics thus affected have a lower maximal exercise ability. The microvascular alterations are believed to be due to a variety of hematologic abnormalities.

Hematologic Abnormalities. Hematologic abnormalities are associated with a decrease in tissue oxygenation and diabetes. They include decreased erythrocyte (RBC) deformability, increased hemoglobin content, and increased blood viscosity (Brownlee, 1985). Erythrocytes often travel through capillaries much smaller than their own diameter so they must have the ability to rapidly deform and reform upon entering and exiting the capillary, respectively. By aspirating RBCs from diabetics and normal controls into micropipets of 4 micometers, it was shown that a regular and unique impairment of RBC deformability occurred in diabetics (McMillan, Utterback et al., 1978). The lack of deformability is suspected to create an impairment of maximal blood flow during exercise, and may also have a direct relationship on the increased blood pressure response often observed in exercising diabetics.

Approximately 5 to 10 % of the total hemoglobin content in normal adult RBCs is comprised of minor hemoglobin species (Brownlee, 1985). Patients with uncontrolled diabetes exhibit a 2 to 3-fold increase

in these minor hemoglobins, with the quantity reflecting the degree of hyperglycemia. These glycosylated hemoglobins, especially the Hb Alc species, have a high affinity for oxygen, thereby reducing the oxygen release and subsequent delivery to the tissues (Coram and Mangum, 1986).

Decreased serum albumin, increased globulins (McMillan, 1981) and increased fibrinogen (Brownlee, 1985) have been identified as the primary cause of the increase in blood viscosity noted in diabetics. The fibrinogen increase also enhances RBC aggregation. These factors, in addition to the lack of deformability cause an increase in the flow resistance of whole blood which ultimately leads to increased work for the heart and a reduction in oxygen delivery.

Although other hematologic abnormalities such as increased erythrocyte aggregation, leukocyte cell host defense response impairment, cell surface changes, and increased platelet adhesion are observed in diabetics, their relationship to microangiopathy are presently unclear, and further research in these areas is highly suggested (Brownlee, 1985).

Effects of Angiopathy on the Performance Capacity of Diabetics. Diabetics with angiopathy tend to exhibit higher blood pressure responses to exercise, reduced maximal heart rates, lower maximal

cardiac outputs (McMillan, 1979), and lower maximal oxygen uptakes (Murphy et al., 1981). The RBC lack of deformability, increased Hb Alc content, increased viscosity of the blood, and general thickening of the capillary basement membranes individually and collectively reduce oxygen delivery to the tissues. It has been suggested that this impaired delivery forces the muscle cells to undergo more anerobic metabolism, thus producing the higher lactate and pyruvate levels observed in diabetics.

A study in which the functional capacity of diabetic-induced rats without insulin replacement was compared to diabetic-induced rats on insulin and to normal control rats concluded that those diabetic rats without insulin had significantly lower maximal $\dot{V}O_2$ than either of the other groups (Murphy et al., 1981). Additionally, as the condition became more severe, the rats showed a lower submaximal and maximal HR response to the same intensity of exercise than they had shown prior to becoming diabetic. This finding contradicts the normally observed higher resting and exercise HR found in both untreated and treated diabetics. However, McMillan (1979) reported that Christensen et al. observed the same response in human subjects with long duration diabetes. This controversy appears to reflect the degree of diabetic control and the

duration of the disease. It is supported by the finding that insulin therapy in the rats appeared to reverse the abnormal responses, although not to quite to their same pre-diabetic level. An impaired sympathetic drive of the heart due to diabetic neuropathy is believed to be responsible for this response. Murphy et al. (1981) reported that Giachetti observed significantly decreased norepinephrine concentrations in those sympathetic nerves innervating the heart in one study. One extremely important finding in this experiment was that the normal linear relationship between $\dot{V}O_2$ and HR was not maintained in the diabetic rat without insulin (Murphy et al., 1981). Therefore, it may be possible that submaximal testing in which submaximal HR are used to predict max $\dot{V}O_2$ will result in erroneous information in this population. Further investigation in this area is indicated.

The exercise induced rise in systolic and diastolic blood pressure in the presence of microvascular changes, as reported by McMillan (1979), could severely stress the occular vessels, provoking hemorrhage. Consequently, diabetics with retinopathy should be cautioned about exercise, and it should perhaps be limited to isotonic low resistance exercise

using the larger muscle groups (Coram and Mangum, 1986).

Exercise causes a redistribution of blood flow in the normal individual. In order to provide for adequate flow to working muscles, a compensatory reduction in both flow and volume occurs in the splanchnic area as well as in the kidneys (Coram and Mangum, 1986). Total blood volume, however, was shown to increase as a result of prolonged physical training (Oscai et al., 1968). The redistribution and blood volume reduction are even more drastically altered in those diabetics with microangiopathy as this condition favors protein and water loss from the plasma thereby reducing cardiac output (McMillan, 1979). Less blood is shunted to the viscera possibly creating renal corticol ischemia in the diabetic with advanced nephropathy. This may result in acute renal insufficiency; therefore, exercise should also be indicated with caution in this situation.

Muscular Strength and Endurance and the Diabetic

Published data specifically comparing muscular strength and endurance between the diabetic and the apparently healthy individual are extremely sparce. As previously indicated the majority of the research performed on fitness levels in the diabetic population have been centered around physcial working capacities

in Type I diabetics. One study was identified, however, in which handgrip strength and local muscular endurance comparisons were made between young Type I diabetic boys and girls and normal age-matched boys and girls (Hebbelinck et at., 1974). The results indicated that there were no significant differences between the diabetic and non-diabetic groups on handgrip strength. Muscular endurance, however, as expressed by a thirty seconds sit-ups test, revealed significant differences in both male and female diabetic children.

Muscular strength, as determined by the size of muscle is dependent primarily upon heredity, testosterone levels, and the degree of physical training (Guyton, 1986; Katch and McArdle, 1983). Males with greater concentrations of testosterone who are physically trained (strength related training) will have larger muscle cross-sectional areas than those with lower concentrations of testosterone who are matched on the other variables, thus enabling greater contractile forces within the muscles (Guyton, 1986).

There is no evidence suggesting lower than normal levels of testosterone in the diabetic, although higher than normal levels have been encountered following exercise (Vignati and Cunningham, 1985) in

this population. The significance of these higher levels, however, is unknown. Additionally, there is no evidence suggesting that the muscular strength of the diabetic cannot be increased with strength training as it can be in the normal subject. The data are far too limited to speculate on the effect of diabetes on muscular strength. However, considering the results of the Hebbelinck et al. study and the fact that no associated deleterious physiologic responses have been identified, there appears to be no reason why strength itself would differ significantly in the diabetic population, provided all other variables were similar. Those diabetics, however, with proliferatiave retinopathy, hypertension and other cardiovascular disorders should avoid performing heavy resistance exercises that elicit excessive intra-abdominal pressures, as this could worsen their condition (Vignati and Cunningham, 1985; Coram and Mangum, 1986).

Muscular endurance, on the other hand, is directly related to the muscle's nutritive support, and primarily upon the amount of glycogen stored prior to exercise (Guyton, 1986). As previously noted, muscle glycogen, in the well controlled diabetic is within or just slightly below the normal range (Bjorkman, 1986). If this were the only factor

responsible for endurance then, it would be possible for the diabetic's performance to be similar to the normal individual's. This, however, is not the case.

Christensen, as reported by Gollnick and Hodgson (1986), found that endurance-trained normal subjects had higher endurance capacities than those who were untrained primarily because the trained muscles exhibited an increased ability to oxidize a higher percentage of fats. This allows for a reduction in the glycogen depletion rate, thus enabling muscular work to be performed for longer periods of time. The mechanism responsible for this is associated with an increase in both the quantity and the size of muscle mitochondria (Holloszy, Dalsky et al., 1986), which has, in turn, been associated with an increase in oxygen utilization. Endurance training in normal subjects also results in lower concentrations of lactate in the blood and muscles. Lactate has been strongly associated with muscle fatique as it is believed to have a negative effect on the contractile elements within the muscle (Astrand and Rodahl, 1977).

Diabetics also, typically, exhibit an increased oxidation of fatty acids during exercise, however, as indicated by Murphy et al. (1981), a study by Armstrong and co-workers showed decreases in the mitochondrial protein concentrations in diabetes

induced animals with consequent reductions in maximal $\dot{V}O_2$ and performance time. Additionally, diabetics have increased lactate levels both at rest and at exercise, potentiating early muscle fatique (Bjorkman, 1986). Poorly controlled diabetic rats showed lower maximal $\dot{V}O_2$ than either the well controlled diabetic rats or the normal rats (Murphy, 1981). Holloszy et. al. (1986) also found that capillary density did not increase with training in the Type II diabetic. Increased capillary density, being a normal adaptation to exercise training, provides for an increase in fuel and oxygen delivery to the working muscles. Based on these findings, it is speculated that the endurance capacity of the diabetic will be lower that of the normal individual, and that the diabetic will not be able to effect the same degree of increases in endurance with training as seen in the normal individual.

Flexibility and the Diabetic

Flexibility, being defined as the possible range of motion within a joint or in a series of joints (deVeries, 1980) is one of the four main components of physical fitness. Although it is generally used in the determination of the overall fitness rating, less emphasis seems to be placed on this area when compared to the other areas of body composition, cardiovascular

fitness, and muscular strength and endurance. A review of the literature indicates that there have been no studies specifically comparing flexibility between the diabetic and the apparently healthy individual.

Flexibility appears to be more related to the grace and finesse with which a sport or activity can be performed rather than to the performance of the activity itself. Of course, there are exceptions in that sports such as competetive diving, hurdling, and ballet will probably not be undertaken by individuals who exhibit inflexibility in those joints critical to the performance of the specific sport.

The range of motion limitations in any joint are set by such mechanical and physical factors as the: bony structure; muscles; connective tissue, including liagments and tendons; and the skin surrounding the joint. Diabetes does not appear to have an unequivocal effect on any of these structures. However, the complications so often encountered in diabetes, particularly neurological disorders and obesity may play a role in reducing joint flexibility. Diabetic neuropathies have most commonly been associated with atrophy of the interosseous musculature of the hands and feet and neuroarthropathy of the tarsometatarsal joints, causing decreased

mobility and flexibity in these areas (Podolsky and Marble, 1985). The excess quantity of fat in the obese diabetic may also cause a reduction in flexibility simply due to physical restriction. It is, therefore, speculated that the presence of diabetes with an absence of other complications should have no significant effect on flexibility of joints.

Benefits of Exercise for the Diabetic

The mechanisms responsible for the benefits produced by exercise are as of yet not completely understood in either the diabetic or the healthy individual. However, there is little doubt that through a conscientious program of regular exercise, the apparently healthy individual can demonstrate improvement in all areas of physical fitness providing the training is performed with adequate frequency, duration and intensity (Astrand and Rodahl, 1977). The area of special importance is in improving cardiovascular fitness, and reductions in several risk factors associated with macroangiopathy have been observed in the normal population. The primary physiologic changes observed include: reductions in blood lipids, blood pressures, and resting and exercise HR; increases in maximal oxygen consumption and peripheral vascular circulation; and improved

insulin sensitivity and glucose tolerance (Vignati and Cunningham, 1985).

It is reasonable to assume that some of the same benefits to the cardiovascular system exhibited in the general population will also occur in the diabetic population, and physical training has been shown to reduce some of the risk factors associated with artherosclerosis (Vignati and Cunninghan, 1985; Richter and Galbo, 1986; Devlin, 1986; DiNovis, et al., 1985; Coram and Mangum, 1986). The primary determing factor as to whether the diabetic can benefit from habitual exercise appears to be related to the state of metabolic control (Bergman and Auerhahn, 1985). Exercise may even be a "perturbation" to metabolism in the poorly regulated, Type I diabetic. One of two responses are likely to occur in this situation, either an increase in hyperglycemia or an exercise induced hypoglycemia.

As summarized by Richter and Galbo (1986), if the pre-exercise insulin levels are low, an exaggerated exercise induced increase in the counter-regulatory hormones occurs causing hepatic glucose output to increase. Additionally, the uptake of glucose in the muscles is impaired due to a lack of insulin-effect as well as to the inhibiting effect of the increased FFA, ketone bodies and catecholamines that normally

accompany the insulin deficient state. Increased hyperglycemia subsequently results. The dangerous condition of diabetic coma is also possible during exercise in the insulin deficient state as this enhances lipolysis and FFA metabolism which, in turn, enhances ketone production (Vignati and Cunningham, 1985). Exercise is, therefore, contraindicated when the diabetic is in a ketogenic state.

Hypoglycemia, on the other hand, is likely to occur in the insulin-dependent diabetic with increased concentrations of insulin either because of enhanced absorption from the injection site or to high plasma levels from a previous injection. These high levels create a situation whereby the hepatic glucose production is decreased and the muscle glucose uptake is increased, possibly potentiating neurological disorders associated with hypoglycemia such as confusion, irritability, and coma (Bergman and Auerhahn,1985; Richter and Galbo, 1986).

Physical training has been shown to produce the following benefits in the well controlled Type I and in the Type II diabetic: increased insulin sensitivity; decreased plasma glucose concentrations; improved lipid profile, including lower cholesterol and triglyceride levels and higher plasma HDL levels; decreased blood pressures; increased maximal $\dot{V}O_2$;

decreased obesity; and increased sense of well-being (Devlin, 1986; Vignati and Cunningham, 1985).

Although conflicting data exists on the ability of exercise to enhance glycemic control, Vignati and Cunningham (1985) refer to studies of Type II diabetics in which regular programs of moderate exercise (greater than 50% max $\dot{V}O2$) resulted in uniformily improved glucose tolerance, whereas acute short-term exercise resulted in a decrease in glucose tolerance. It is fairly well accepted that habitual exercise improves insulin resistance in both diabetic types providing good control is maintained, thus possibly allowing a reduction in the daily dose of insulin or oral hypoglycemic agents (Bergman and Auerhahn, 1985).

Summary

As was discussed, diabetes mellitus is an extremely complex syndrome causing alterations in the normal metabolism of carbohydrates, fats, and proteins both at rest and during exercise. The complications generally associated with this disease create additional impairments in the functional ability of the diabetic individual. The consequences of these abnormalities vary depending upon the severity and state of control, however, it is quite probable that

the physical performance of the diabetic individual will be less than that of the apparently healthy individual.

CHAPTER 3

Methodology

The benefits of exercise as a therapeutic agent for the type II diabetic have long been realized, however, current knowledge in establishing training programs is quite inadequate. The purpose of this study was to compare the fitness levels of type II diabetics with those of an apparently healthy population. If proven to be different, norm tables should be designed specifically for the diabetic population in order provide a solid foundation for the development of safe and effective exercise programs.

Although the data collected at the University of Nevada at Las Vegas (UNLV) was an independent venture, the same study was also performed at both the International Diabetes Center in Minneapolis, Minnesota and at the Alvarez Diabetic Center in San Diego, California. The data from all three locations will be compiled and used in the final analyses.

In addition, an independent study measuring pre and post exercise blood glucose levels was performed at the same time on the UNLV subjects. Although there may be reference to this study, especially in the

questionnaires and letter of explanation to the or subjects, the data accumulated will not be presented or discussed in this paper.

Experimental Design

Each volunteer, after receiving medical clearance agreed to be tested according to the procedures described in The Y's Way to Physical Fitness (Golding et al., 1982) in the following four fitness categories: body composition; cardiovascular endurance; flexibility; and muscular strength and endurance. All data was assembled and analyzed according to age and sex and then compared to the norm tables established by the YMCA for the apparently healthy population (Golding et al., in press).

Subjects

Only the subjects tested at UNLV will be described in this section. With the assistance of several local physicians, potential type II diabetic subjects were identified and a letter of explanation was sent to each (Appendix A). Those who expressed interest in participating were asked to be subjects providing they received medical approval from their primary care physician. The subjects were 12 adult males ranging in age from 53 to 69 and 14 adult

females between 41 to 67 years of age. The descriptive data of this diabetic population are summarized in Appendix E.

Additionally, there were 126 male and 74 female diabetic subjects from the other testing centers for a total sample size of 138 males and 88 females. The number of males and females included in the YMCA norm tables were 2271 and 2008, respectively. They represented the same age group as the diabetic sample.

Procedures

All subjects reported to the laboratory in exercise clothing at a time convenient to their schedules. They had been instructed earlier to follow their normal daily routine of eating, sleeping and taking medications but to refrain from smoking for at least two hours prior to the testing. It was also suggested that for their comfort they not eat a large meal immediately before exercising.

The purpose, procedures, risks and benefits were explained and each subject signed an informed consent form (Appendix B).

Upon the completion of a medical history and physical activity questionnaire (Appendix C), the actual testing was explained and performed in the following manner:

Sitting Heart Rate and Blood Pressure

The sitting heart rate and blood pressures were recorded while the subject remained seated with both feet flat on the floor using the following techniques and equipment:

1. heart rates were determined by counting the pulse for one full minute with the aid of a stethescope.

2. left arm systolic and diastolic blood pressures were measured in the standard manner using a sphygmomanometer and a stethescope.

Standing Height and Weight

The height in centimeters (cm) and the weight in kilograms (kg) were measured using the standard physician's scale allowing the subject to remain clothed but without shoes.

Pre-test Blood Glucose Determination

At this point, the pre-exercise blood glucose level was measured for the independent study using the blood glucose meter and the Chemstrip bG reagent strips.

Skinfold Measurements

Skinfold measurements were performed to determine percent subcutaneous fat distribution using the Harpenden skinfold calipers graduated to 0.2mm. The sites used included:

MEN	WOMEN
chest	thigh
abdomen	abdomen
ilium	ilium
tricep	tricep
scapula	scapula
thigh	

The estimation of percent body fat was calculated according to the new Jackson and Pollock equations (unpublished data) based on the sum of four skinfold measurements (abdomen, ilium, tricep, and thigh) for both men and women. The formulae are listed below:

Percent Body Fat--Sum of Four

MEN % Fat = -5.76377 + 0.29288 ($\Sigma 4$) - 0.0005 ($\Sigma 4^2$) + 0.15845 (Age)

WOMEN % Fat = 1.40724 + 0.29669 ($\Sigma 4$) - 0.00043 ($\Sigma 4^2$) + 0.0293 (Age)

Trunk Flexion Flexibility

The trunk flexibility measurement was then obtained in the following manner:

1. the subjects were allowed to perform slow, non-ballistic stretching exercises for a few minutes while the actual procedure was explained and demonstrated.

2. they then sat on a specially designed flexibility board with their hips flexed, knees extended and arms outstretched between their legs with one hand on top of the other.

3. they were then instructed to slowly reach forward as far as possible along the ruler on the board and to hold that position temporarily while the testor mentally recorded the distance. They were encouraged to keep their trunk straight, and to insure that their lower legs remained extended, the testor held their knees down.

4. the best of three trials was recorded to the nearest one-half inch (in).

Three-Minute Step Test

The three-minute step test was performed to measure cardiorespiratory fitness using the following equipment and procedures:

1. a timing clock was set for three minutes and a recovery clock for one minute.

2. a calibrated metronome was set at 96 beats per minute (bpm) which is equivalent to 24 steps per minute on a 12-inch bench.

3. the one-minute recovery heart rate was obtained using a stethescope.

4. the procedure was explained and demonstrated.

5. the clock was turned on as the subject began stepping and continued for three minutes.

6. at the end of stepping, the subjects sat on the stepping bench.

7. the one-minute recovery heart rate started within 5 seconds of the cessation of stepping.

8. the score was recorded as the total 1-minute recovery HR.

Muscular Strength and Endurance

Muscular strength and endurance was measured by both the bench press and the one-minute timed situps. The bench press was performed as follows with males using an 80-pound barbell and females using a 35-pound barbell:

1. the procedure was explained and demonstrated.

2. a metronome was set for 60 beats per minute and the subject reclined on a standard bench in the supine position with the feet firmly supported.

3. with elbows flexed, hands pronated and shoulder width apart, the barbell was given to the subject.

4. the barbell was pressed upward to full extension of the elbows on the first beat of the metronome and it was returned to the starting position on the second beat. This sequence was

repeated until the subject could either no longer maintain the rhythm or until full elbow extension could not be accomplished.

5. the maximum number of presses was recorded.

After explaining and demonstrating the one-minute timed situps, they were performed as follows:

1. the subject reclined in a supine position on an exercise mat with the knees bent and the hands clasped lightly behind the head.

2. the testor held the subject's feet down to provide stability.

3. when the clock was started, the subject began performing as many situps in one minute as possible. The situp was considered to be correct if the subject sat up, touched one elbow to the opposite knee (the opposite elbow and knee were used during the next situp), and then returned to the starting position.

4. the maximum number of situps was recorded.

Submaximal Bicycle Ergometer Test

A submaximal bicycle test, another indicator of cardiovascular fitness, was performed using the following equipment and procedures:

1. a Monark bicycle ergometer.

2. the YMCA"S "Guide to setting workloads" for males and females (Golding et al., 1982).

3. a Microcore portable ECG monitor and electrodes for recording the heart rate.

4. metronome set at 100 beats per minute to provide a speed of 50 rpm throughout each workload.

5. timing clock.

6. each subject was connected to a CM-5 ECG lead for the purpose of recording heart rates during each stage, rather than using the heart rate conversion chart listed in the Y's test battery.

7. the procedure was explained and demonstrated, and the subject mounted the bicycle.

8. the metronome was turned on and the subject was asked to begin pedalling with no resistance in order to synchronize the beat and the pedalling. This was assured if each foot was at the same position relative to the crankshaft with each beat of the metronome.

9. the test was initiated by applying a resistance of 150 kgm to the bicycle ergometer in accordance with the Y's "Guide to Setting Workloads" while simultaneously starting the

clock.

10. heart rates were recorded at the end of the second and third minutes of this workload, and if a steady-state heart rate was reached (no more than five beats per minute difference between each minute), the resistance was increased in accordance with the same guidelines.

11. this process continued until two workloads yielding steady-state heart rates with at least ten beats per minute difference were encountered, assuming that the first workload used resulted in a heart rate of at least 110 beats per minute.

12. the results were recorded on the "Physical Working Capacity Test" sheets (Appendix D).

13. the electrodes were disconnected from the subject.

Post-test Blood Glucose Determination

The post-exercise blood glucose level for the independent study was then determined and recorded using the same procedures as before.

Analyses of the Data

This study compared the fitness levels of Type II diabetics to the apparently healthy population. The four general fitness categories used for comparison were body composition, cardiovascular fitness, muscular strength and endurance, and flexibility. The subjects were grouped according to age and sex and the variability between both groups was determined for the following variables: weight; percent body fat; sitting heart rate; sitting blood pressure (systolic and diastolic); one-minute recovery heart rate (bench step test); predicted maximum workload and $\dot{V}O2$ (submaximal bicycle test); muscular strength and endurance (sit-ups and bench press); and trunk flexibility (Appendix E).

The null hypothesis is that the physical fitness level of Type II diabetics is no different from the apparently healthy population. The Z $\bar{x}$ test was used to determine whether any observed differences between the groups' means were statistically significant. An alpha level of $p < 0.05$ was chosen.

CHAPTER 4

Results and Discussion

This study compared the physical fitness levels of male and female Type II diabetics, ages 40 and above, to apparently healthy subjects of the same age group. Raw data on the diabetics are presented (Appendix E) and the data on the diabetics from UNLV are identified separately as well as being included with the entire group of diabetics. Raw data on the apparently healthy populations are not presented. Means and standard deviations were determined for all groups on the following variables: age; weight; % body fat; 7 skinfold site measurements; resting heart rate (RHR); resting systolic and diastolic blood pressures (RSBP and RDBP respectively); trunk flexion flexibility; 3-minute step test; $\dot{V}O2$ max and PWC max predicted from the submaxmimal bicycle ergometer test; and muscular strength and endurance measurements as measured by the bench press and situps tests.

The data were treated with a two-tailed Z test of significant differences using the 0.05 level of significance. A significant difference was determined to exist if the absolute value of the Z $\overline{x}$ (observed) was greater than or equal to the Z critical value of 1.96. Tables 1 and 2 present the number of subjects

(N), means, standard deviations and Z scores on male subjects, and tables 3 and 4 present the same information on female subjects. The results of each comparison will be presented and examined separately.

Age

Significant differences in age were encountered in both males and females with the diabetic subjects being older than the normal population (males 56 vs 53; females 55 vs 53). Although a decline in physical working capacity is associated with the aging process, it is doubtful that these small differences in age would be of any practical significance. Astrand and Rodahl (1977) noted only a 0.3 liter reduction in $\dot{V}O_2$ max over a ten year period in non-active subjects between the two age groups of 50 to 59 and 60 to 69. When expressed as milliliters of oxygen per kilogram of body weight, the difference over a two or three year period (differences noted in the diabetic groups over the normal subjects) would probably be insignificant. Other studies indicate the average rate of decline in $\dot{V}O_2$ max for non-active men is approximately 0.40 to 0.45 $ml \cdot kg^{-1} min^{-1}$ per year and 0.30 $ml \cdot kg^{-1} min^{-1}$ per year for sedentary women (Stamford, 1988). These represent reductions of less

Table 1. Comparison of means between male diabetics and apparently healthy males (all variables except skinfold measurements)

	DIABETICS			APPARENTLY HEALTHY			
	n	$\bar{X}$	s	N	μ	σ	Z score
age	138	56	8.4	2226	53	9.3	3.80*
weight	136	196	29.8	2098	186	27.7	4.20*
% Fat	132	25	6.3	1932	24	5.6	0.21
RHR	138	75	11.9	2151	71	11.2	4.41*
RSBP	138	140	14.0	2177	129	14.7	8.80*
RDSP	126	85	8.9	2178	81	9.4	4.76*
Flexi-bility	136	12	3.9	2101	12	5.2	0.00
3 min step	123	119	18.0	795	109	19.4	5.71*
bench press	136	10	6.4	1534	13	8.8	0.40
situps	138	17	9.3	1989	23	10.6	6.65*
$\dot{V}O_2$ max	122	32	11.4	1208	34	10.4	0.21
PWC max	121	1103	369.1	1220	1171	382.2	1.96*

*significant difference at 0.05 level

Table 2. Comparison of means between male diabetics and apparently healthy males (skinfold measurements)

	DIABETICS			APPARENTLY HEALTHY			
	n	$\bar{X}$	s	N	μ	σ	Z Score
chest	137	22	7.7	1918	20	8.7	2.70*
abdomen	132	32	13.5	1943	31	11.1	1.03
ilium	135	27	11.3	1944	24	10.3	3.39*
axilla	137	22	8.0	1130	21	7.8	1.49
scapula	136	23	8.4	1141	20	7.9	4.41*
triceps	133	12	5.6	1208	13	5.3	2.17*
thigh	136	16	6.8	1179	17	6.9	1.69

* significant difference at the 0.05 level

Table 3. Comparison of means between female diabetics and apparently healthy females (all variables except skinfold measurements)

	DIABETICS			APPARENTLY HEALTHY		
	n	$\bar{X}$	s	N	μ	σ	Z Score
age	88	55	8.1	2000	53	9.1	2.06*
weight	87	174	36.3	1934	147	25.9	9.71*
% fat	80	33	6.7	1740	31	6.9	0.26
RHR	88	78	13.5	1929	72	10.3	5.45*
RSBP	87	135	12.8	1917	121	14.8	8.81*
RDBP	85	84	7.9	1936	77	9.6	6.73*
Flexi-bility	87	15	4.3	1841	16	4.1	0.23
3 min test	73	123	18.7	627	117	18.8	2.73*
bench press	84	15	8.7	1286	13	9.3	1.98*
situps	86	10	10.0	1491	16	9.5	3.92*
$\dot{V}O_2$ max	83	26	6.5	1012	30	9.3	3.92*
PWC max	84	767	203.1	1021	767	264.1	0.00

* significant difference at 0.05 level

Table 4. Comparison of means between female diabetics and apparently healthy females (skinfold measurements)

	DIABETICS			APPARENTLY HEALTHY			
	n	$\bar{X}$	s	N	μ	σ	Z Score
chest	52	34	12.2	254	18	9.4	12.31*
abdomen	82	39	13.5	1764	31	11.8	6.15*
ilium	84	32	12.5	1761	23	10.8	7.63*
axilla	51	33	10.6	240	21	8.9	9.60*
scapula	87	28	9.2	1101	20	8.4	8.89*
triceps	88	27	9.0	1745	24	7.9	3.57*
thigh	86	34	10.0	1103	34	11.8	0.00

* significant difference at the 0.05 level

than 1 percent per year, and would, therefore, be of no biological significance.

Weight and Percent Body Fat

Table 1 shows significant differences in body weight between male diabetics and normals (196 vs 186) with similar results for females in Table 3 (174 vs 147). There were, however, no significant differences in percent body fat determined from skinfold measurements of 4 sites in either males or females (males 25 vs 24; females 33 vs 31). When comparing actual skinfold measurements (tables 2 and 4), numerous significant differences were noted between the groups. Male diabetics had significantly greater measurements at the chest, ilium, and scapula whereas the normals had a significantly higher tricep measurement. Normal males had a greater mean thigh measurement, but it was not significant. The female diabetics had significantly greater measurements at all sites except the thigh.

An increased body weight without a corresponding increase in percent body fat could be explained as either: (1) the diabetics had a higher percent of muscle mass; or (2) the diabetics were significantly taller; and/or (3) the diabetics had a higher percent of deep body fat. An increased muscle mass seems

unlikely in the diabetic samples for two reasons. The loss of lean tissue has been associated with the aging process (Stamford, 1988), and the diabetics were significantly older. Although this age associated muscle loss can be diminished through chronic exercise, it is doubtful that the daily activity levels were substantially different between the diabetics and the apparently healthy subjects. In addition, the very nature of diabetes with a lack of insulin promotes protein degradation and muscle atrophy.

If the diabetic subjects were significantly taller than the normal population, a greater weight would be expected without an associated increase in body fat. This variable was not analyzed, however, there appears to be no correlation between diabetes and height, thus no significant differences were expected to exist.

The third explanation of a greater percent of internal fat is the most reasonable since the altered lipid metabolism of diabetes often leads to fatty livers and hyperlipidemia. Skinfold calipers assume a constant relationship between subcutaneous fat and deep fat (Stamford, 1988). This relationship may not be applicable in the diabetic or in older subjects.

It is interesting to note that when comparing the mean percent body fat of the apparently healthy subjects from the new YMCA fitness norm tables (Golding et al., in press) to the mean ranking of the old YMCA fitness norms (Golding et al., 1982) for the age group 46 and older, significant increases have occurred during the past 6 years (t-test at p 0.05). The results showed that the percent body fat increased 4 % in males (from 21 to 25 %) and 6 % in females (from 25 to 31 %).

Cardiovascular Variables

Resting HR. Male and female diabetics showed significantly greater resting mean heart rates than the apparently healthy populations (males 75 vs 71; females 78 vs 72). Since the resting HR is affected by so many factors such as age, sex, size, posture, ingestion of food, emotions, body temperature, environmental factors and smoking (deVries, 1980), predicitng physical performance based on heart rates is meaningless. However, it is known that the slower the heart rate, the greater the efficiency of the heart, all things being equal. Also, Astrand and Rodahl (1970) showed that individuals with larger stroke volumes and lower heart rates have a greater capacity for oxygen transport. These facts allow for

a strong implication that the significantly higher heart rates observed in the diabetic samples lower the cardiac efficiency and oxygen consumption resulting in a decreased physical performance.

Resting systolic and diastolic blood pressures. The diabetic population showed significantly greater mean RSBP and RDBP than the normal subjects (males RSBP 140 vs 129, RDBP 85 vs 81; females RSBP 135 vs 121, RDBP 84 vs 77). This supports the generally agreed upon fact that hypertension is approximately twice as common in the diabetics as in the non-diabetic population (Christlieb, 1985). The diabetic males, with a mean RSBP of 140 mm Hg are considered to be borderline hypertensive, and the mean RSBP of the female diabetics is clearly at the upper limits of normal. High blood pressure causes an increased workload on the heart which is generally asociated with hypertrophy of the heart muscle. The accompanying coronary blood supply, however, does not increase as much as the actual increase in muscle mass. This ultimately leads to a relative ischemia (Guyton, 1986) and a reduction of cardiovascular capacity.

Cardiovascular Fitness

Three-minute step test. The three-minute step test, an indicator of cardiovascular fitness based on a one-minute recovery heart rate resulted in significantly higher recovery heart rates for both male and female diabetics (males 119 vs 109; females 123 vs 117). This indicates that the cardiovascular stress of bench stepping was significantly greater for the diabetics than for the normal population. Katch and McArdle (1983) additionally concluded that those subjects with higher recovery heart rates also tended to have lower maximal oxygen uptakes, again indicating a potentially decreased physical capacity in the diabetic subjects.

$\dot{V}O_2$ max and PWC max. The submaximal bicycle ergometer tests showed conflicting results both within the test itself and to the one-minute recovery heart rate indication of cardiorespiratory fitness. Male diabetics had a significantly lower maximal working capacity, or PWC max (1103 vs 1171) without a corresponding decrease in $\dot{V}O_2$ max (32 vs 34), whereas the female diabetics showed a significantly lower $\dot{V}O_2$ max (26 vs 30) without a corresponding decrease in PWC max (174 vs 174). The measures of $\dot{V}O_2$ max and PWC max should parallel each other since both depend upon the individual's ability to supply oxygen to the working

muscles. The discrepant findings in this study can best be explained as an error in measurement, for when prediction equations are used to predict one variable from another, any initial measurement error has the potential of becomming magnified during the derivation, thus invalidating the results. Without further investigation, a statement comparing the cardiorespiratory fitness of diabetics to normals based on the submaximal bicycle test is premature. The three-minute step test results, in this case, appear to be the most appropriate indicator of cardiorespiratory fitness as less measurement error is likely to exist.

Muscular Strength and Endurance

No significant difference in the number of bench presses was noted between the male diabetics and the normal males although the diabetics performed fewer repetitions (10 vs 13). The male diabetics performed significantly fewer situps (17 vs 23). The female diabetics performed a significantly greater number of bench presses than the normal females (15 vs 13). The results of the situps were the same as the males in that the female diabetics performed a significantly fewer number (10 vs 16).

Since cross sectional areas and muscle mass were not measured in this study, it is difficult to speculate as to why the female diabetics showed superior upper body strength and endurance to the apparently healthy females. It has been observed that a heavier person may have greater total strength over a lighter one when lifting a heavy weight, but the lighter person may have greater strength per unit of body weight (Morehouse and Miller, 1967). Therefore, the fact that the female diabetics were 18 percent (27 lb) heavier than the apparently healthy females may have had some impact on this finding. The male diabetics were also heavier, but only by 5 percent (10 lb). This weight may not have been excessive enough to cause greater strength as observed in the female diabetics.

Trunk Flexion Flexibility

No significant difference was shown to exist in either the males or the females in the area of flexibility (males 12 vs 12; females 15 vs 16). As previously mentioned, this area of fitness was not assumed to reveal significant differences unless the diabetic individual had neurological disorders leading to inflexible joints.

Percentile tables on all variables are located in Appendix E for comparisons between the diabetic population and the apparently healthy population.

CHAPTER 5

Summary and Conclusions

Summary

This study was designed to compare the physical fitness levels of Type II diabetics to the apparently healthy population. The particular fitness categories of body composition, cardiovascular capacity, flexibility, and muscular strength and endurance were examined using the Y's Way to Physical Fitness (Golding et al., 1982) battery of tests. Eighty-eight Type II diabetic females, ages 40 and above, (14 from the Las Vegas area and 74 from diabetic centers in Minneapolis and San Diego) were compared to 2,008 apparently healthy female subjects, and 138 Type II diabetic males (12 from Las Vegas and 126 from the other centers) were compared to 2,271 apparently healthy males of the same age group.

Means and standard deviations were obtained on all groups for the variables of age, weight, percent body fat, resting heart rate (RHR), resting systolic and diastolic blood pressures (RSBP and RDSP, respectively), one-minute recovery HR (3 minute step test result), bench press, situps, and $\dot{V}O2$ and PWC max (submaximal bicycle test results), and flexibility. The Z $\bar{x}$ test was then used to determine whether

variability between the groups' means was statistically significant at the $p < 0.05$ level.

Significant differences were determined on several measurements in the female diabetics when compared to the apparently healthy females. These include greater: age; weight; RHR; RSBP; RDBP; one-minute recovery HR; and number of bench presses performed as well as a significantly lower number of situps performed. No significant differences were observed in flexibility or percent body fat (although all skin fold measurements except the thigh were significantly higher in the diabetic females). Although significant differences were noted on the submaximal bicycle test no statement comparing cardiorespiratory fitness between the diabetic population and the normal population will be offered at this time as errors in measurements are believed to exist.

Significant differences were determined on several measurements in the male diabetic population. They, like the female diabetics, showed significantly greater age, weight, RHR, RSBP, RDPB, and one-minute recovery HR, and they performed a significantly fewer number of situps. No significant differences were noted in the number of bench presses, flexibility, or percent body fat. Conflicting data was also observed

in $\dot{V}O_2$ max and PWC max due to possible errors in measurements. Consequently, a statement on these variables seems premature.

Conclusion

1. Since no significant difference was observed in percent body fat in either diabetic group, it cannot be stated that Type II diabetics are fatter in this category. It should be noted that all groups are considered to have at least "marginal obesity" according to YMCA data (Golding et al., 1982). The mean percent body fat has increased significantly in both the male and female apparently healthy populations during the past 6 years. The males showed a 4 % increase from 21--25 %, and the females showed a 6 % increase from 25--31% (Golding et al., in press).

2. Based on the findings of significantly greater RHR, RSBP, and RDSP, both male and female Type II diabetics have a reduced circulatory efficiency and a lower physical performance ability over the apparently healthy populations. This is supported by the significantly greater one-minute recovery HR in both diabetic groups.

3. Both the male and female diabetic populations performed a significantly fewer number of situps, and can, therefore, be considered to have weaker abdominal

muscles. Although the female diabetics performed a significantly greater number of bench presses, it seems premature to classify them as the more fit group, as they had an additional 18 percent body weight to assist in the weight lifting. No significant difference in the number of bench presses occurred in the males.

4. No significant difference was determined for trunk flexion flexibility, and, consequently, no difference in the flexibility levels are assumed to exist between Type II diabetics and the normal population.

Recommendations

Based on the results and observations of this research, the following recommendations are made:

1. New norm tables should be developed for male and female Type II diabetics, ages 40 and above, as significant differences were determined between diabetics and the apparently healthy population in many of the fitness variables.

2. The same testing should be performed in large groups of younger male and female Type II diabetics to determine whether the same fitness differences noted in this study would prevail. If differences occur,

new fitness norms should also be prepared for these age groups.

3. Since Murphy et al. (1981) found that the normal linear relationship between $\dot{V}O_2$ and HR was not maintained in exercising diabetic rats without insulin, and considering the fact that this study used submaximal heart rates to predict $\dot{V}O_2$ max, further investigation in this areas is recommended in order to assure the validity of this prediction in Type II diabetics.

4. Since male and female diabetics both failed to show a significant increase in percent body fat in conjunction with the significantly higher body weight, a correlational study between hydrostatic weighing and skinfold measurements is recommended for Type II diabetics. The validity of the skinfold measurements, as performed in this study, may be questioned as the diabetic population is generally known to have a higher plasma lipid concentration as well as increased fat content of certain organs (namely the liver). The increase in deep fat would probably not be reflected in the currently used skinfold equations.

APPENDIX A

Letter of Explanation

April 29, 1988

Dear potential participant:

The department of exercise physiology at the University of Nevada, Las Vegas, is currently working with the Inter-national Diabetic Center in Minneapolis, Minnesota on a study designed to assess the exercise tolerance of type II diabetics. This is the first step necessary before exercise programs can be designed specifically for the diabetic population.

We are in need of approximately 100 type II diabetics between the ages of 20 and 65 who would be willing to donate no more than one hour of their time for this purpose. The procedure will be completed at the UNLV campus lab and will, in general, consist of a fitness test and a determination of percent body fat (please see the separate, enclosed sheet for a more detailed explanation of the actual testing procedure).

If you meet the above criteria and are interested in assisting us, please have your doctor read the enclosed explanation sheets and sign the medical release form. Then please make an appointment for testing by calling 739-0980 or if no answer, leave a message with Janice at 739-3766 and I will return your call.

Thank you for your time and consideration.

Very cordially,

Joan K. MacDonald
Research Associate

Type II Diabetic Exercise Tolerance Assessment
General Explanation of the Testing Procedure

Once you have been selected and medically cleared by your physician to be a participant in this study, you will be asked to come to the Exercise Physiology Lab on the UNLV campus to complete the following battery of tests at no cost to you (this is the Y's Way to Fitness test battery):

1. Standing height
2. Weight
3 Resting heart rate
4. Resting blood pressure
5. Skinfold measurements (consists of pinching the skin in several sites for the determination of percent body fat)
6. 3-minute step test (consists of fairly slow stepping up and down on a special bench for three minutes in order to evaluate cardiovascular fitness)
7. Flexibility test (consists of sitting on the floor with the legs fully extended and simply bending over at the hips and reaching as far as possible)
8. Bench Press (consists of lifting a set of barbells off your chest as many times as possible while lying on a weight bench; 35 lb for women and 80 lb for men)
9. Situps (consists of properly performing as many situps as possible in one-minute)
10. Submaximal bicycle test (consists of riding a stationary bicycle for approximately 10 minutes while your heart rate response is monitored)

Your confidentiality as a participant will be maintained by keeping all files in a locked cabinet to which only research personnel will have access. All statistial data in the computer files will be coded. Names, addresses and telephone numbers will not be released without your written permission, and all subject data will be destroyed after final summarization.

Additional information, such as a medical history and signed informed consent form will be obtained at a later date. We will also notifiy you as to the type of clothing to wear for the test, when and what to eat or drink, etc. prior to your actual testing on campus.

MEDICAL CLEARANCE FOR TYPE II DIABETIC STUDY
at UNLV

I, Dr. _____ am aware
(physician's name)
that my patient, _____ is
(patient's name)
interested in participating in a study designed to test the fitness level of Type II diabetics at UNLV's Department of Exercise Physiology. I understand that the Y's Way to Fitness battery of tests will be the standard testing tool, and that each patient will only undergo testing only one time. It is currently my professional decision to (please initial the appropriate space):

1. _____ grant medical clearance for my patient to participate.
2. _____ deny medical clearance at the present time.

_____
(Printed physician's name)

_____
(physician's signature)

_____
(date)

APPENDIX B

Informed Consent

Consent to Participate in a
Research Study
International Diabetes
Center/University of Nevada,
Las Vegas
and YMCA of the USA

Title of Study: The physical fitness level of Type II diabetics.

Purpose: You are being asked to participate in a research study. We hope to learn if there is a difference in the physical fitness levels of Type II Diabetics and a similar apparently healthy population. This is the first step in determining exercise programs for diabetics.

Subjects: Because you are an adult (between the ages of 20-65 years) and a Type II Diabetic you have been recruited as a subject.

Procedures: If you decide to volunteer, you will be asked to get medical clearance from your personal physician. If medically cleared you will have the following tests administered to you:

Height	3 minute step test
Weight	Flexibility test
Resting heart rate	Bench press (Men 80 lbs-
Resting blood pressure	women 35 lbs.)
Skinfold caliper measurements	One minute timed situps
(7 sites)	Submaximal bicycle test

This test battery is the "Y's Way to Physical Fitness" fitness evaluation tool and has been administered to thousands of adults and excellent norms have been developed. The test requires approximately 30 minutes to take.

Risks: Anytime adults exercise there is a potential risk. The medical clearance and physical fitness evaluation attempts to define limitations or contraindications for exercise. While testing there is always risk of tripping or falling. Muscle soreness and stiffness usually occurs in beginner exercisers. Overexertion can result in nausea and/or fainting. Every effort will be made to monitor exercise intensity and to provide safe supervison, and to safeguard your health, although you agree to look to your personal physician for medical care and treatment. The test battery is a sub maximal one.

Benefits: There are obvious benefits to this research. No physical fitness norms have been established for the diabetic population. Norms will enable diabetics to compare their fitness level with others, it will also give a starting point for an exercise program and will enable participants to measure improvement in fitness due to exercise.

Confidentiality: Subject's confidentiality will be maintained by keeping subjects files in a locked file. Only research per-

sonnel have access to the files. Statistical data in computer files are coded. Names, addresses and telephone numbers will not be released without subjects permission. At the conclusion of the study or when subjects leave the study, their files will be destroyed after data is summarized.

Right to refuse or withdraw: You may refuse to participate in any part of this study and you may change your mind about being in the study and withdraw after the study has started.

Questions: Any questions you had about the study, its purpose, design, methodology, procedures, or significance have been answered to your satisfaction. If you have additional questions later research personnel will be happy to answer them.

Your signature below will indicate that you have decided to volunteer as a research subject and that you have read the information provided above and understand the study.

Date:_____ _____
Signature of participnat

Date: _____ _____
Signature of witness

Print Participant name:_____

Print Witness name: _____

APPENDIX C

Medical History

and

Physical Activity Questionnaire

The Y's Way to Diabetes Control
International Diabetes Center/University of Nevada, Las Vegas
and the YMCA of the USA

Questionnaire (card 1)

Name _____ Date ___/___/___ Time _____

Address _____

Home Phone _____ Work Phone _____

Name, address and phone number of physician who manages your diabetes care: _____

_____

Note: All spaces must be filled in, if necessary with 0, unless no data, then leave blank.

___ ___ ___ ___	(1-4)	Identification number
___	(5)	Sex (1=male, 2=female)
___ ___	(6,7)	Age (years on day of test)
___ ___	(8,9)	Height in inches.
___ ___ ___	(10-12)	Weight in pounds.

Health History (1=yes; 2=no; 3=don't know)

___	(13)	Heart disease
___	(14)	High blood pressure
___	(15)	Diabetes
___	(16)	Lung disease
___	(17)	Kidney problems
___	(18)	Muscle disease
___	(19)	Back/joint problems
___	(20)	Arthritis
___	(21)	Eye Problems
___	(22)	Stroke, Blood vessel or circulatory problems
___	(23)	Are you currently taking medication (1=yes; 2-no)

If yes, give name of medication, reason for taking it, and number of times taken daily.

_____ _____

_____ _____

_____ _____

___ ___ (24,25) Year of onset of Diabetes

___ ___ (26,27) Age at onset of Diabetes

Nutrition

___ (28) Are you overweight (1=yes; 2=no)

___ (29) If yes, approximately how many pounds (1=under 10 lbs; 2=10-20 lbs; 3=over 20 lbs.)

___ (30) Are you <u>now</u> on a weight control program? (1=yes; 2=no)

___ ___ ___ ___ (31-34) If yes, how many calories?

___ ___ (35,36) Percent cholesterol (if known)

___ ___ (37,38) Percent fat (if known)

___ ___ (39,40) Percent protein (if known)

Exercise

___ (41) Do you <u>now</u> engage in sports or fitness activities on a regular basis (1=yes; 2=no)

___ (42) If yes, what sport or activity

1=Swimming	7=Basketball
2=Running	8=Cycling
3=Exercise class	9=Other____
4=Racquet Sports	_____
5=Walking	_____
6=Weight Training	

___ (43) If yes, how often (days per week)

___ ___ ___ (44-46) If yes, how long per session (mins)

___ (47) How physically fit do you feel now?
1=Unfit 4=Above average
2=below avg. 5=Very fit.
3=about avg.

___ (48) Have you ever been told that you should <u>not</u> exercise vigorously? (1=yes; 2=no)

___ (49) When exercising vigorously, do you usually eat more, change insulin?
1=decrease insulin
2=take extra feeding
3=both 1 & 2
4=neither 1 or 2

The Y's Way to Diabetes Control
International Center/University of Nevada, Las Vagas
and the YMCA of the USA

Test Results Tester (initials) ___

Name _____ Date ___/___/___ Time_____

Note: All spaces must be filled in, if necessary with 0, unless no data, then leave blank. (e.g. if individual tries and cannot do one bench press fill in 0-if the person has a sore arm and cannot do a bench press-leave blank).

Card 2

___ ___ ___ ___	(1-4)	Identification Number
___	(5)	Sex (1=male, 2=female)
___ ___	(6,7)	Age (years on day of test)
___ ___ ___	(8-10)	Sitting systolic pressure
___ ___ ___	(11-13)	Sitting diastolic pressure
___ ___ ___	(14-16)	Sitting heart rate
___ ___	(17,18)	Height (nearest inch)
___ ___ ___	(19-21)	Weight (nearest pound)
___ ___	(22,23)	Pectoral Skinfold
___ ___	(24,25)	Abdominal Skinfold
___ ___	(26,27)	Ilium Skinfold
___ ___	(28,29)	Axilla Skinfold
___ ___	(30,31)	Tricep Skinfold
___ ___	(32,33)	Scapula Skinfold
___ ___	(34,35)	Front Thigh Skinfold
___ ___	(36,37)	Percent fat (sum of 6 men, sum of 5 women)
___ ___	(38,39)	Percent fat (sum of 4 men, sum of 3 women)
___ ___ ___	(40-42)	3 minute step test (recovery HR-1 min)
___ ___	(43,44)	Flexibility (nearest inch)

___ ___ (45,46) Bench press (repetitions, 35 lbs women, 80 lbs men, 60 bpm, start down position)

___ ___ (47,48) Timed Situps

___ ___ ___ (49-51) 1st workload used to graph

___ ___ ___ (52-54) HR at above

___ ___ ___ ___ (55-58) 2nd workload used to graph

___ ___ ___ (59-61) HR at above

___ ___ ___ ___ (62-65) Predicted PWC MAX

___ (.) ___ (66-68) Predicted MAX V02-Liters (leave 2nd space alone with decimal point)

___ ___ (.) ___ (69-72) V02 Max (ml/kg/min) divided last number by weight (kg) - (leave 3rd space alone with decimal point)

___ (73) Location of testing. (1= Ridgedale, 2=UNLV, 3=San Diego)
4=_____, 5=_____

6=_____, 7=_____

8=_____, 9=_____

Glucose 1 _____

Glucose 2 _____

APPENDIX D

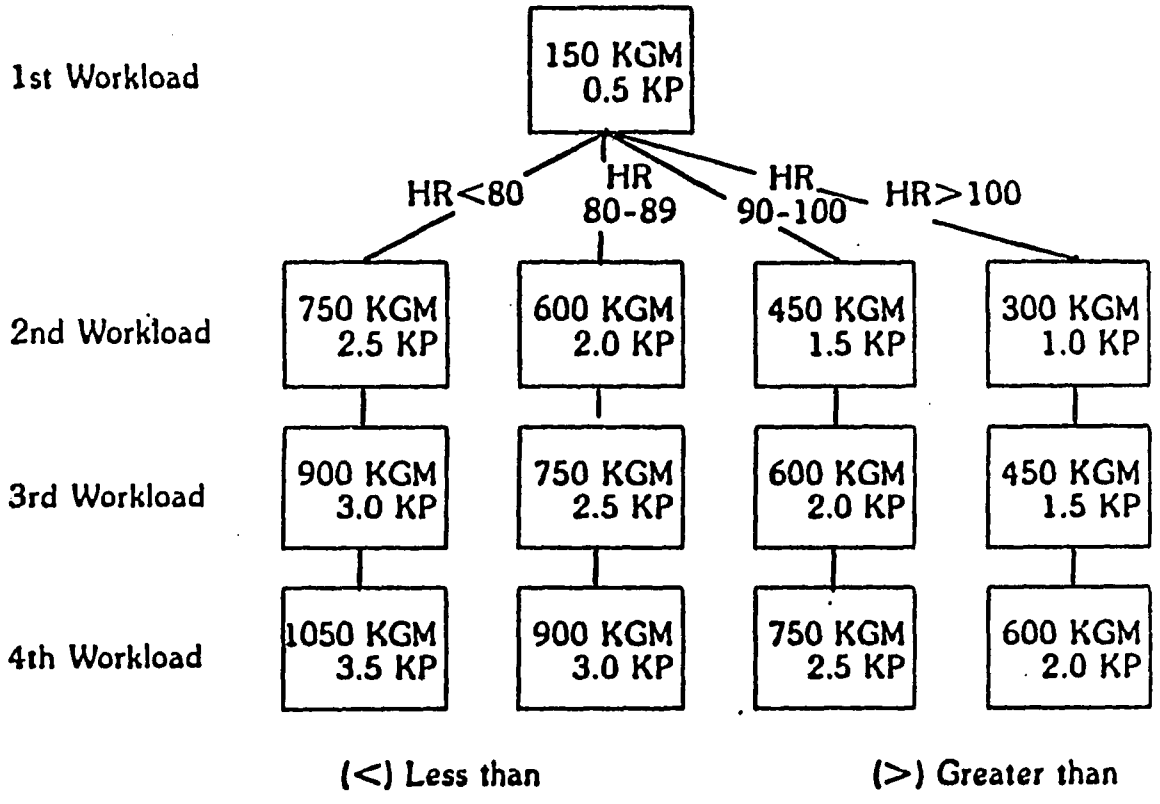
YMCA Guide to Setting Workloads

and

Physical Working Capacity Sheets

DIRECTIONS
1. Set the first work load at 150 KGM/min (0.5 KP)
2. If HR in third min is:
   Less than (<) 80, set 2nd load at 750 KGM (2.5 KP)
   80 to 89, set 2nd load at 600 KGM (2.0KP)
   90 to 100, set 2nd load at 450 KGM (1.5 KP)
   Greater than (>) 100, set 2nd load at 300 KGM (1.0KP)
3. Set third and fourth (if required) loads according to the loads in colum below second loads

FIGURE I



Graph 4-6

Y's WAY TO PHYSICAL FITNESS — TEST BATTERY

MAXIMUM PHYSICAL WORKING CAPACITY PREDICTION.

NAME _____ AGE _____ WEIGHT _____ LB. _____ KG SEAT HEIGHT _____

PREDICTED MAX. HR _____

	DATE	1st WORKLOAD HR USED	2nd WORKLOAD HR USED	MAX. WORKLOAD	MAX O_2(L/min)	MAX O_2(ml/kg)
TEST 1						
TEST 2						
TEST 3						

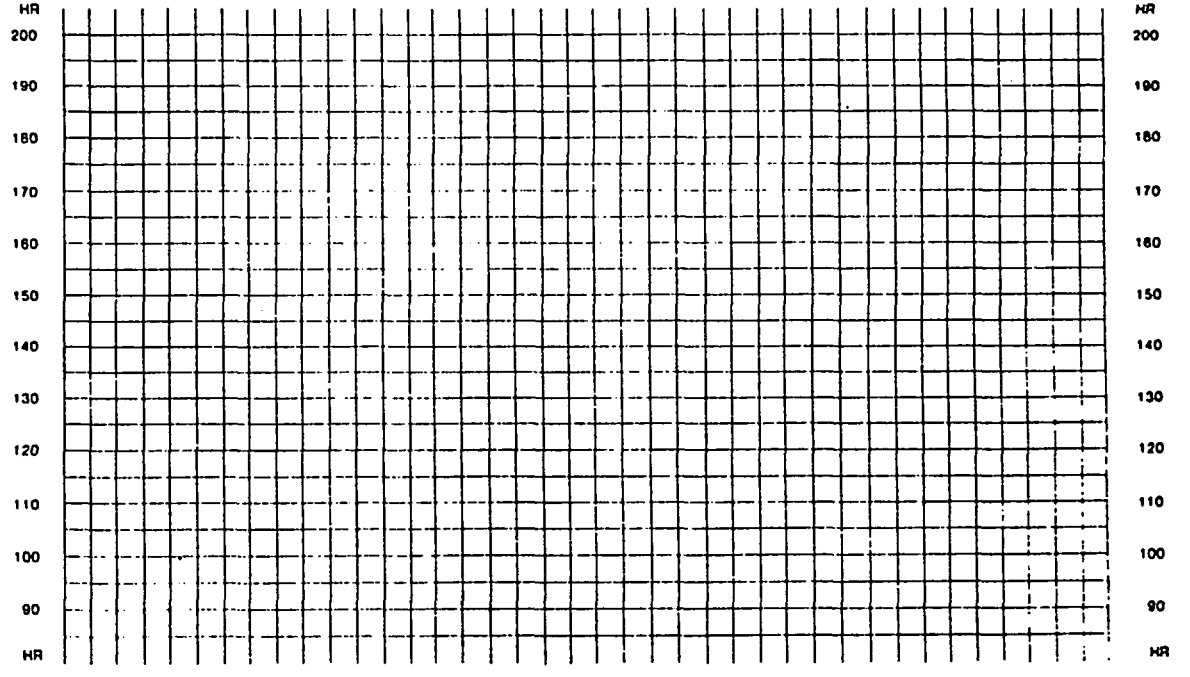
DIRECTIONS

1 Plot the HR of the 2 workloads versus the work (kgm/min)

2 Determine the subject's max HR. e by subtracting subject's age from 220 and draw a line across the graph at this value

3 Draw a line through both points and extend to the max HR line for age

4 Drop a line from this point to the baseline and read the predicted max. workload and O_2 uptake



WORKLOAD (kgm/min)	150	300	450	600	750	900	1050	1200	1350	1500	1650	1800	1950	2100
MAX O_2 UPTAKE (L/m)	0.6	0.9	1.2	1.5	1.8	2.1	2.4	2.8	3.2	3.5	3.8	4.2	4.8	5.0
KCAL USED (Kcal/m)	3.0	4.5	6.0	7.5	9.0	10.5	12.0	14.0	16.0	17.5	19.0	21.0	23.0	25.0
APPROX. MET LEVEL (for 132) lbs.)	3.3	4.7	6.0	7.3	8.7	10.0	11.3	12.7	14.0	15.3	16.7	18.0	19.3	20.7
APPROX. MET LEVEL (for 176 lbs.)	3.0	4.0	5.0	6.0	7.0	8.0	9.0	10.0	11.0	12.0	13.0	14.0	15.0	16.0

PHYSICAL WORKING CAPACITY TEST

NAME_____

SEAT HEIGHT_____

Workload	Heart Rate	
	_____2nd min	
150 kgm	_____3rd min.	1st WORKLOAD
	_____	
	_____2nd min.	
___ kgm	_____3rd min.	2nd WORKLOAD
	_____	
	_____2nd min.	
___ kgm	_____3rd min.	3rd WORKLOAD
	_____	
	_____2nd min.	
___ kgm	_____3rd min.	4th WORKLOAD
	_____	

APPENDIX E

Descriptive Data and Fitness Results on Las Vegas Diabetics

Means and Standard Deviations on all Data from Combined Diabetic Population

Means and Standard Deviations on all YMCA Data

Percentile Rankings for Diabetics and Apparently Healthy

Raw Data on Diabetics

DESCRIPTIVE DATA on LAS VEGAS DIABETICS

FEMALES

Subject's Initials	Age	Wt (lbs)	RBPS	RBPD	RHR	% Fat
JB	65	142	118	88	96	31.2
OD	58	221	142	90	66	**
MEF	47	144	90	62	66	29.6
LH	62	151	122	70	76	37.7
TH	58	125	118	70	88	25.0
CK	63	183	140	90	84	40.4
ML	56	179	140	82	78	40.6
LJ	63	128	118	70	84	32.7
VR	62	207	150	90	72	31.7
NU	59	169	130	80	72	31.2
MN	67	162	138	78	80	35.0
SS	54	186	122	84	72	37.1
BL	54	243	140	82	108	**
BW	55	176	110	70	68	**
MEAN	58.8	172.6	127.0	79	79.3	33.8
SD	5.3	34.4	16.0	9.2	12.0	4.8

MALES

Subject's Initials	Age	WT (lbs)	RBPS	RBPD	RHR	% Fat
AA	57	194	170	106	74	26.6
JD	61	154	132	64	72	18.7
WD	61	153	158	86	58	19.7
FD	63	183	120	76	68	23.6
SD	69	159	120	80	70	27.5
CG	63	174	150	92	80	25.3
GH	62	177	130	82	68	25.1
SS	62	185	150	84	70	25.1
JM	60	204	140	70	60	28.8
NT	53	243	138	94	84	26.3
WT	58	182	138	84	68	20.1
EG	61	182	122	72	88	27.6
MEAN	60.8	182.5	139	82.5	71.7	24.6
SD	3.9	24.5	15.7	11.5	8.9	3.5

* Resting blood pressures--systolic and diastolic
** could not be obtained

FITNESS RESULTS on LAS VEGAS DIABETICS

FEMALES

Subject's Initials	PWC Max	$\dot{V}O_2$ Max	Flexi-bility	Bench Press	Sit-ups	Step Test
JB	320	15.5	19.5	9	13	160
OD	***	***	13.0	24	0	*
MEF	628	23.8	12.0	0	10	108
LH	600	21.9	16.0	7	0	124
TH	704	30.1	13.0	1	0	*
CK	738	21.4	10.0	15	0	*
ML	495	15.9	13.5	17	4	*
LJ	394	18.8	10.0	1	0	*
VR	548	14.9	14.0	5	0	*
NU	854	26.2	10.0	16	4	*
MN	1060	32.6	5.0	1	0	*
SS	500	14.2	0.0	2	6	145
BL	*	*	12.0	10	0	*
BW	1000	28.7	13.5	10	13	124
MEAN	653.4	22.0	11.5	8.4	3.4	132.2
SD	229.0	6.4	4.5	7.1	4.8	18.2

MALES

AA	818	41.8	8.0	10	10	*
JD	713	47.3	6.0	1	0	118
WD	744	25.8	11.0	6	12	90
FD	**	**	2.0	1	0	**
SD	469	17.2	15.0	1	12	135
CG	588	18.7	6.0	5	15	121
GH	1400	38.6	13.0	6	16	109
SS	979	26.9	9.0	1	0	134
JM	***	***	15.0	27	9	106
NT	970	20.3	15.0	14	5	143
WT	711	20.9	18.0	0	17	102
EG	800	22.9	11.0	2	0	135
MEAN	819.2	28.0	10.8	6.2	8.0	119.3
SD	256.2	10.7	4.5	7.5	6.4	17.4

*--could not complete due to fatique
**--not completed as subject was taking a beta blocker
***--advised against completing due to numerous PVCs

MEANS and STANDARD DEVIATIONS ON ALL DIABETICS

FEMALES

	N	Mean	SD
Age	88	55	8.1
Wt	87	174	36.3
Resting HR	88	78	13.5
Resting BP Systolic	87	135	12.8
Resting BP Diastolic	85	84	7.9
% Fat	80	33	6.7
PWC max	84	767	203.1
$\dot{V}O_2$ max	83	26	6.5
Flexibility	87	15	4.3
Bench press	84	15	8.7
Situps	86	10	10.0
Step test one-minute recovery HR	73	123	18.7

MEANS and STANDARD DEVIATIONS ON ALL DIABETICS

MALES

	N	Mean	SD
Age	138	56	8.4
Wt	136	196	29.8
Resting HR	138	75	11.9
Resting BP Systolic	138	140	14.0
Resting BP Diastolic	126	85	8.9
% Fat	132	25	6.3
PWC max	121	1103	369.1
$\dot{V}O_2$ max	122	32	11.4
Flexibility	136	12	3.9
Bench press	136	10	6.4
Situps	138	17	9.3
Step test one-minute recovery HR	123	119	18.0

MEANS and STANDARD DEVIATIONS ON ALL YMCA DATA

FEMALES

	N	Mean	SD
Age	2000	53	9.1
Wt	1934	147	25.9
Resting HR	1929	72	10.3
Resting BP Systolic	1917	121	14.8
Resting BP Diastolic	1936	77	9.6
% Fat	1740	31	6.9
PWC max	1021	767	264.1
$\dot{V}O_2$ max	1012	30	9.3
Flexibility	1841	16	4.1
Bench press	1286	13	9.3
Situps	1491	16	9.5
Step Test one-minute recovery HR	627	117	18.8

MEANS and STANDARD DEVIATIONS ON ALL YMCA DATA

MALES

	N	Mean	SD
Age	2226	53	9.3
Wt	2098	186	27.7
Resting HR	2151	71	11.2
Resting BP Systolic	2177	129	14.7
Resting BP Diastolic	2178	81	9.4
% Fat	1932	24	5.6
PWC max	1220	1171	382.2
$\dot{V}O_2$ max	1208	34	10.4
Flexibility	2101	12	5.2
Bench press	1534	13	8.8
Situps	1989	23	10.6
Step Test one-minute recovery HR	795	109	19.4

PERCENTILES ON DIABETIC FEMALES

Weight, Resting BP and HR, and % Fat

Percentile	Wt	RBPS	RBPD	RHR	% Fat
5	124	118	70	59	20
10	129	119	72	61	22
15	136	122	74	64	26
20	145	123	78	65	28
25	150	124	80	67	28
30	153	127	80	68	30
35	155	130	82	69	32
40	160	132	82	72	32
45	166	134	84	75	32
50	171	138	84	77	33
55	175	138	86	79	34
60	177	139	88	81	35
65	179	139	90	84	36
70	182	140	90	85	37
75	186	140	90	87	38
80	193	144	90	88	39
85	212	150	92	89	40
90	238	151	92	99	42
95	254	158	94	101	43
100	260	172	98	110	45

Skinfold Measurements

Percentile	Chest	Abdomen	Ilium	Axilla	Scapula	Tricep	Thigh
5	16	14	14	15	11	15	18
10	19	20	16	18	15	16	19
15	20	24	18	20	18	17	23
20	23	26	20	22	19	19	25
25	25	29	22	23	21	20	27
30	27	32	23	24	22	20	29
35	27	33	24	27	25	21	30
40	28	34	26	30	25	23	31
45	29	37	27	31	26	24	31
50	29	38	28	32	27	25	33
55	32	40	31	34	29	27	34
60	36	42	32	35	29	27	35
65	37	44	36	36	30	28	35
70	39	48	39	38	31	29	37
75	41	49	42	39	33	31	38
80	44	50	43	40	34	35	39
85	47	53	44	44	35	36	47
90	48	56	50	50	37	39	50
95	59	58	52	52	46	43	51
100	66	66	60	56	56	50	56

PERCENTILES ON DIABETIC FEMALES (continued)

PWC max, $\dot{V}O_2$ max, Flexibility, Bench Press, Sit-ups, and Step Test

Percentile	PWC max	$\dot{V}O_2$ max	Flexibility	Bench Press	Sit-ups	Step Test
5	457	15	7	0	-0	91
10	512	17	7	4	-0	98
15	557	19	9	6	-0	103
20	602	21	11	8	0	106
25	632	21	11	8	0	112
30	654	21	13	10	0	115
35	676	23	13	10	2	117
40	695	23	13	12	4	120
45	713	24	13	14	6	121
50	735	25	13	14	6	123
55	757	25	15	14	10	126
60	781	27	15	16	12	128
65	813	27	15	18	12	130
70	851	27	17	20	14	134
75	895	29	17	20	18	136
80	932	31	17	20	20	140
85	985	33	19	24	22	144
90	1032	33	19	24	22	148
95	1167	35	21	32	28	152
100	1317	46	24	34	36	160

PERCENTILES ON YMCA FEMALES

Weight, Resting BP and HR, and % Fat

Percentile	Wt	RBPS	RBPD	RHR	% Fat
5	112	98	61	56	18
10	118	101	65	59	21
15	122	106	68	61	22
20	126	110	69	63	24
25	128	111	70	64	25
30	131	113	70	65	26
35	134	116	73	68	27
40	136	117	74	69	28
45	139	118	76	71	29
50	142	119	77	72	30
55	145	122	80	73	31
60	149	124	80	73	32
65	152	126	81	76	33
70	155	129	82	77	34
75	161	131	82	79	35
80	167	134	85	80	36
85	173	137	88	83	38
90	183	141	89	85	39
95	200	146	92	89	41
100	236	168	110	106	50

Skinfold Measurements

Percentile	Chest	Abdomen	Ilium	Auxilla	Scapula	Tricep	Thigh
5	4	11	6	6	7	10	14
10	5	15	9	9	10	13	18
15	7	18	11	10	11	15	21
20	8	20	13	12	11	17	23
25	9	22	14	13	12	18	25
30	11	24	17	14	14	19	27
35	12	26	18	16	15	19	29
40	13	27	19	17	16	21	30
45	15	28	21	18	18	22	31
50	16	31	22	20	18	22	33
55	17	32	23	21	19	23	34
60	19	34	25	21	20	25	35
65	20	35	26	22	22	26	37
70	21	36	27	24	23	27	39
75	24	39	30	26	24	29	41
80	25	40	31	28	26	30	43
85	28	43	34	30	28	31	46
90	29	47	38	32	32	34	50
95	33	51	42	36	25	38	54
100	46	68	56	46	48	48	68

PERCENTILES ON YMCA FEMALES (continued)

PWC max, $\dot{V}O_2$ max, Flexibility, Bench Press, Sit-ups, and Step Test

Percentile	PWC max	$\dot{V}O_2$ max	Flexibility	Bench Press	Sit-ups	Step Test
5	399	16	7	0	0	86
10	467	19	9	1	2	92
15	505	20	11	2	5	96
20	543	22	11	4	8	100
25	577	23	13	5	9	103
30	611	24	13	6	10	106
35	642	25	13	8	12	109
40	671	27	15	9	13	111
45	701	27	15	10	14	114
50	729	28	15	12	16	117
55	757	30	17	12	17	119
60	790	31	17	13	18	121
65	824	32	17	14	20	124
70	864	33	17	17	21	126
75	913	35	19	18	22	129
80	963	36	19	21	24	132
85	1022	39	19	22	26	136
90	1125	42	21	25	29	141
95	1275	47	21	30	32	148
100	1784	69	28	46	46	178

PERCENTILES ON DIABETIC MALES

Weight, Resting BP and HR, and % Fat

Percentile	Wt	RBPS	RBPD	RHR	% Fat
5	153	119	70	55	14
10	157	123	71	58	16
15	162	126	75	61	17
20	170	128	78	65	19
25	174	130	79	67	20
30	178	131	80	69	20
35	181	132	82	70	21
40	185	138	83	71	22
45	189	139	86	73	23
50	193	140	86	74	24
55	195	140	87	77	25
60	198	143	88	77	26
65	201	144	90	78	26
70	206	148	91	79	27
75	213	150	91	81	28
80	221	152	92	83	29
85	231	154	95	87	30
90	241	158	96	90	32
95	257	160	99	94	36
100	272	180	108	112	43

Skinfold Measurements

Percentile	Chest	Abdomen	Ilium	Axilla	Scapula	Tricep	Thigh
5	8	11	11	8	11	5	7
10	12	17	13	12	11	5	9
15	12	18	14	14	15	5	9
20	14	21	15	14	17	7	11
25	16	23	18	14	17	7	11
30	18	25	19	16	17	9	11
35	18	26	21	18	19	9	13
40	20	27	22	18	19	9	13
45	20	29	25	18	21	9	13
50	20	30	26	20	21	11	15
55	22	31	27	22	23	11	15
60	22	33	28	24	23	13	15
65	24	34	31	24	25	15	17
70	24	37	33	24	27	15	19
75	26	39	35	26	29	15	19
80	28	46	37	28	31	17	19
85	30	49	39	30	33	17	21
90	34	53	43	32	35	19	27
95	36	58	49	36	37	21	29
100	40	68	56	44	46	32	38

PERCENTILES ON DIABETIC MALES (continued)

PWC max, $\dot{V}O_2$ max, Flexibility, Bench Press, Situps, and Step Test

Percentile	PWC max	$\dot{V}O_2$ max	Flexibility	Bench Press	Situps	Step Test
5	575	17	4	-0	0	83
10	720	21	6	0	2	95
15	750	21	8	2	4	99
20	780	22	8	4	8	103
25	810	23	8	4	10	105
30	867	25	8	6	12	109
35	928	26	10	6	14	114
40	988	27	10	8	16	119
45	1041	29	10	10	18	120
50	1087	30	12	10	18	121
55	1131	33	12	10	20	122
60	1168	34	12	10	20	123
65	1206	35	14	10	22	125
70	1257	37	14	12	22	127
75	1312	38	14	12	24	129
80	1363	41	14	14	24	133
85	1413	43	16	14	26	137
90	1553	47	16	16	30	139
95	1688	51	18	20	32	144
100	2414	67	20	30	34	172

PERCENTILES ON YMCA MALES

Weight, Resting BP and HR, and % Fat

Percentile	Wt	RBPS	RBPD	RHR	% Fat
5	144	107	64	53	14
10	151	109	70	57	17
15	157	113	71	60	18
20	161	116	72	61	19
25	165	119	75	62	20
30	168	120	78	64	21
35	171	121	78	65	22
40	176	123	79	66	22
45	179	125	80	68	23
50	182	126	80	69	24
55	186	127	82	72	24
60	189	129	83	73	25
65	192	132	84	74	26
70	197	135	86	76	27
75	201	138	88	77	28
80	206	139	90	80	29
85	206	141	91	82	30
90	220	146	92	85	31
95	232	156	98	92	33
100	312	214	132	118	38

Skinfold Measurements

Percentile	Chest	Abdomen	Ilium	Axilla	Scapula	Tricep	Thigh
5	7	14	8	9	10	5	7
10	8	16	11	12	10	7	9
15	10	19	14	12	11	7	10
20	12	22	15	13	12	9	11
25	14	23	16	14	14	9	11
30	14	24	18	16	15	9	13
35	15	26	19	17	15	9	13
40	16	27	20	18	16	11	14
45	18	28	22	20	18	11	15
50	19	30	23	20	19	11	15
55	20	31	24	21	19	13	17
60	20	32	26	22	20	13	18
65	22	34	27	24	22	13	18
70	23	36	28	25	23	15	19
75	24	38	31	25	24	15	21
80	27	40	32	26	26	17	22
85	28	43	35	29	28	19	23
90	32	46	38	32	31	19	27
95	36	51	43	36	35	23	31
100	48	64	56	46	44	32	44

PERCENTILES ON YMCA MALES (continuted)

PWC max, $\dot{V}O_2$ max, Flexibility, Bench Press, Sit-ups, and Step Test

Percentile	PWC max	$\dot{V}O_2$ max	Flexibility	Bench Press	Sit-ups	Step test
5	632	18	2	0	5	76
10	749	21	4	2	9	83
15	805	24	6	4	12	88
20	862	25	8	5	13	92
25	914	26	10	6	14	95
30	956	27	10	8	17	98
35	999	29	10	8	18	100
40	1041	30	10	9	20	102
45	1085	31	12	10	21	106
50	1127	33	12	12	22	110
55	1170	34	12	13	24	112
60	1212	36	14	14	25	114
65	1266	37	14	16	26	117
70	1320	38	14	17	28	119
75	1375	39	16	20	29	122
80	1454	42	16	21	30	125
85	1535	44	16	22	33	129
90	1655	47	18	25	37	132
95	1864	52	20	29	41	141
100	2792	76	40	42	54	162

RAW DATA ON LAS VEGAS DIABETIC FEMALES--SKINFOLD MEASUREMENTS, WORKLOADS AND HEART RATES

Skinfolds

ID	Chest	Abdomen	Ilium	Axilla	Scapula	Tricep	Thigh
JB	29	34	26	25	31	22	30
OD	27	42	*	34	44	34	40
MEF	18	37	20	14	14	16	34
LH	41	42	38	34	28	29	39
TH	20	21	17	20	18	19	27
CK	19	50	42	24	44	35	37
ML	37	43	32	47	47	43	49
LJ	24	31	25	24	27	26	38
VR	33	22	24	25	27	32	37
NU	29	37	21	22	24	21	34
MN	28	41	30	*	40	30	31
SS	36	46	35	31	37	33	32
BL	17	34	28	27	29	29	*

*--could not be obtained

Workloads and heart rates

ID	1st wkld over 110 HR (kgm)	HR at 1st wkld	2nd wkld over 110 HR (kgm)	HR at 2nd wkld
JB	150	142	300	164
OD	**	**	**	**
MEF	300	114	450	140
LH	150	110	300	126
TH	150	114	300	126
CK	150	110	300	122
ML	150	118	300	138
LJ	150	113	300	140
VR	300	125	450	145
NU	300	113	450	126
MN	150	112	300	120
SS	150	126	300	138
BL	**	**	**	**
BW	300	120	450	130

**--could not complete the bicycle test

RAW DATA ON LAS VEGAS DIABETIC MALES--SKINFOLD MEASUREMENTS, WORKLOADS AND HEART RATES

Skinfolds

ID	Chest	Abdomen	Ilium	Axilla	Scapula	Tricep	Thigh
AA	26	35	26	29	29	18	16
JD	8	19	24	24	28	26	9
WD	18	14	18	18	28	19	9
FD	19	27	20	22	22	16	13
SD	20	26	23	19	20	21	20
CG	21	29	26	18	15	10	19
GH	37	22	39	27	23	14	9
SS	25	33	23	22	23	12	16
JM	36	41	40	28	30	9	14
NT	50	40	36	33	43	10	11
WT	15	18	25	19	23	12	9
EG	19	34	28	26	25	19	16

Workloads and Heart rates

ID	1st wkld over 110 HR (kgm)	HR at 1st wkld	2nd wkld over 110 HR	HR at 2nd wkld
AA	450	114	600	134
JD	300	115	450	130
WD	450	109	600	135
FD	*	*	*	*
SD	300	115	450	147
CG	150	122	300	134
GH	300	114	450	120
SS	150	111	450	128
JM	**	**	**	**
NT	150	126	450	141
WT	300	110	450	129
EG	300	118	450	132

*--on beta blocker (Inderol)
**--advised against continuing as subject began having numerous PVCs

DIABETIC SUBJECT RAW DATA

Column 1: identifier code
Column 2: age
Column 3: weight (lbs)
Column 4: systolic blood pressure
Column 5: diastolic blood pressure
Column 6: resting heart rate (bpm)
Column 7: chest skinfold
Column 8: abdomen skinfold
Column 9: ilium skinfold
Column 10: axilla skinfold
Column 11: scapula skinfold
Column 12: tricep skinfold
Column 13: thigh skinfold
Column 14: 1st workload on submax bicycle test
Column 15: heart rate at 1st workload
Column 16: 2nd workload on submax bicycle test
Column 17: heart rate at 2nd workload
Column 18: flexibility (in)
Column 19: bench press
Column 20: 1 minute timed situps
Column 21: step test (1 minute recovery heart rate)

FEMALES

89110	36	130	120	80	70	20	19	19	15	20	13	20	300	110	450	135	24.0	15	25	104
89110	36	129	120	82	70	0	19	9	0	12	13	17	300	118	450	133	24.0	15	25	104
89110	39	170	150	0	90	30	45	40	40	35	42	45	150	120	300	135	13.0	9	10	135
89110	40	180	140	90	85	35	50	40	42	30	45	50	150	113	300	132	14.0	10	9	138
89110	41	155	150	90	90	40	44	38	30	35	30	45	300	121	450	140	18.0	15	2	115
89110	41	140	130	75	100	15	13	16	15	12	20	20	150	110	300	130	20.0	19	23	146
89110	41	154	150	92	87	0	44	28	0	26	28	44	300	121	450	135	18.0	15	0	115
89110	41	140	126	72	99	0	13	6	0	12	20	18	150	113	300	127	20.0	19	23	146
89110	42	257	160	95	100	65	65	55	50	35	35	40	150	120	300	145	8.0	35	1	153
89110	42	257	158	94	108	0	0	44	0	33	34	50	150	128	300	137	8.0	35	0	153
89110	42	257	158	94	108	0	0	44	0	33	34	50	150	128	300	137	8.0	35	0	153
89110	43	327	130	80	100	60	55	60	45	50	30	35	150	125	300	148	8.0	20	1	140
89110	43	130	120	75	80	15	15	20	20	15	15	30	150	110	300	125	18.0	20	28	115
89110	43	237	128	80	105	0	50	45	0	24	27	28	150	127	300	142	18.0	20	0	139
89110	43	128	118	74	78	0	12	18	0	9	15	31	150	112	300	123	18.0	20	28	114
89110	45	190	138	88	86	38	57	42	40	28	41	55	150	111	300	128	22.0	14	6	140
89110	45	160	135	95	100	40	40	45	40	35	20	25	150	120	300	149	20.0	10	15	135
89110	45	190	138	88	86	0	57	42	0	25	41	54	150	111	300	128	22.0	14	6	140
89110	45	160	132	92	100	0	39	30	0	26	19	19	150	125	300	144	22.0	10	15	133
89110	47	144	90	62	66	18	37	20	14	14	16	34	300	114	450	140	12.0	0	10	108
89110	49	155	102	72	64	30	41	24	29	24	25	28	300	110	450	126	13.0	99	15	100
89110	49	155	102	72	64	0	41	24	0	24	25	28	300	110	450	126	13.0	0	16	95
89110	50	185	150	90	90	50	50	36	35	30	30	35	300	112	450	135	12.0	20	1	130
89110	50	186	148	92	90	0	50	26	0	29	29	33	300	114	450	128	12.0	27	0	127
89110	51	176	138	78	62	30	38	33	32	22	22	18	450	118	600	135	15.0	25	13	0
89110	52	240	142	82	88	0	65	52	0	29	39	36	300	114	450	122	8.0	9	99	140
89110	52	227	122	86	63	38	65	53	40	35	20	50	300	115	450	128	13.0	99	5	110
89110	52	227	122	86	63	0	0	53	0	35	19	50	600	110	750	120	13.0	0	5	0

89110	53	170	125	85	70	25	26	23	25	23	15	27	450	110	600	125	16.0	30	30	102
89110	53	153	125	90	75	40	39	32	30	30	25	35	300	111	450	135	23.0	12	36	129
89110	53	170	122	82	65	0	26	23	0	23	15	27	450	110	600	125	16.0	30	30	102
89110	53	153	124	84	75	0	39	22	0	20	22	36	300	111	450	125	23.0	12	36	124
89110	54	150	140	90	85	30	35	20	35	25	20	35	150	111	300	130	17.0	20	20	125
89110	54	175	145	99	90	50	56	45	40	30	22	30	150	115	300	129	13.0	10	1	130
89110	54	173	144	99	88	0	56	43	0	29	12	23	150	115	300	129	13.0	10	0	128
89110	54	186	122	84	72	36	46	35	31	37	33	32	150	126	300	138	0.0	2	6	145
89110	54	243	140	82	108	17	34	29	27	29	29	0	0	0	0	0	12.0	10	0	0
89110	55	118	122	68	75	20	23	16	20	19	15	23	300	121	450	130	14.0	8	10	108
89110	55	145	135	80	90	35	37	24	30	25	25	38	150	113	300	128	19.0	21	18	124
89110	55	205	140	90	70	45	55	56	50	35	35	40	450	130	600	150	12.0	33	6	130
89110	55	118	122	68	75	0	23	16	0	19	15	23	300	121	450	130	14.0	8	10	108
89110	55	205	140	88	67	0	53	26	0	32	35	56	450	128	600	145	12.0	33	6	0
89110	55	145	132	80	92	0	37	24	0	15	25	38	150	113	300	128	19.0	21	18	124
89110	55	176	110	70	68	22	30	26	24	32	22	0	300	120	450	130	13.5	10	13	124
89110	56	180	150	90	85	50	50	50	45	50	25	35	300	110	450	130	13.0	12	1	119
89110	56	181	150	88	84	0	50	47	0	23	22	29	300	115	450	128	13.0	12	0	114
89110	56	179	140	82	78	37	43	32	47	47	43	49	150	118	300	138	13.5	17	4	0
89110	57	175	140	90	75	45	55	55	50	50	20	35	300	110	450	130	6.0	10	3	121
89110	58	160	135	85	77	50	45	40	50	55	35	45	300	111	450	127	10.0	11	8	130
89110	58	186	140	90	80	0	52	45	0	27	25	54	150	120	300	131	16.0	4	2	115
89110	58	221	142	90	66	27	42	0	34	44	34	40	0	0	0	0	13.0	24	0	0
89110	58	125	118	70	88	20	21	17	20	18	19	27	150	114	300	126	13.0	1	0	0
89110	59	175	130	80	60	45	48	31	40	28	23	35	300	110	450	130	16.0	20	21	134
89110	59	150	140	85	85	40	42	38	35	35	20	35	300	120	450	150	10.0	24	19	137
89110	59	250	120	90	55	60	65	0	55	60	50	40	450	115	600	135	13.0	20	1	140
89110	59	256	120	88	51	0	0	0	0	36	49	32	450	116	600	125	15.0	50	0	0
89110	59	173	130	78	59	0	48	31	0	18	23	32	300	113	450	129	16.0	20	21	134
89110	59	145	138	84	86	0	42	18	0	18	19	9	300	125	450	146	10.0	24	19	137
89110	59	256	120	88	51	0	0	0	0	36	49	32	450	116	600	125	15.0	50	0	0
89110	59	169	130	80	72	29	37	21	22	24	21	34	300	113	450	126	10.0	16	4	0
89110	60	150	140	90	75	28	29	30	26	25	30	30	300	120	450	140	12.0	13	11	145
89110	60	185	140	90	80	50	52	45	50	35	25	30	150	120	300	141	16.0	4	2	115
89110	60	176	138	78	62	0	38	33	0	22	22	18	450	118	600	135	15.0	25	0	72
89110	61	137	160	92	98	0	28	19	0	14	21	23	150	115	300	129	16.0	19	13	152
89110	62	151	135	80	73	25	30	29	25	26	29	37	300	120	450	143	13.0	20	12	150
89110	62	125	140	90	69	25	32	20	20	19	18	28	300	122	450	140	15.0	5	99	97
89110	62	180	150	85	70	30	25	25	30	30	25	35	450	110	600	135	17.0	9	19	104
89110	62	125	140	92	69	0	32	20	0	19	18	28	300	122	450	140	15.0	5	0	97
89110	62	151	136	78	73	0	30	29	0	26	29	37	300	120	450	143	13.0	20	12	150
89110	62	179	148	84	66	0	25	25	0	24	25	23	450	110	600	120	17.0	9	19	104
89110	62	151	122	70	76	41	42	38	34	28	29	39	150	110	300	126	16.0	7	0	124
89110	62	207	150	90	72	33	22	24	25	27	32	37	300	125	450	145	14.0	5	0	0
89110	63	190	130	85	70	46	56	44	35	30	29	30	450	110	600	135	6.0	15	1	94
89110	63	188	128	84	69	0	56	44	0	30	29	32	450	119	600	127	6.0	15	0	94
89110	63	183	140	90	84	19	50	42	24	44	35	37	150	110	300	122	10.0	15	0	0
89110	63	128	118	70	84	24	31	25	24	27	26	38	150	113	300	140	10.0	1	0	0
89110	64	180	140	0	82	30	32	17	35	28	37	50	300	118	450	129	19.0	24	21	122
89110	64	160	130	85	60	30	34	35	30	35	30	25	300	110	450	130	17.0	15	15	130
89110	64	130	130	75	70	15	12	17	15	20	7	20	450	115	600	145	18.0	17	22	120
89110	64	185	140	90	70	27	27	30	35	35	17	25	450	110	600	136	11.0	6	4	115
89110	64	180	140	99	82	0	32	17	0	28	37	50	300	118	450	129	19.0	24	21	122
89110	64	240	142	82	88	0	0	52	0	29	39	36	300	114	450	122	8.0	9	0	89
89110	64	155	128	82	58	0	34	24	0	21	29	35	0	0	0	0	17.0	15	15	0
89110	64	126	128	76	68	0	12	7	0	12	21	35	450	118	600	135	18.0	17	22	118
89110	65	175	150	0	85	30	35	28	30	25	35	45	300	120	450	130	13.0	20	22	125
89110	65	160	172	94	66	0	36	25	0	29	30	33	450	115	600	124	17.0	7	7	83
89110	65	142	118	88	96	29	34	26	25	31	22	30	150	142	300	164	19.5	9	13	160
89110	67	162	138	78	80	28	41	30	0	40	30	31	150	112	300	120	5.0	1	0	0

89110	35	170	120	70	65	13	23	13	8	11	5	7	600	113	750	133	16.0	16	33	135
89110	35	168	120	70	62	13	23	13	39	11	5	7	600	113	750	133	16.0	16	33	135
89110	36	200	126	78	79	15	25	24	15	24	15	22	300	143	450	155	16.0	17	30	130
89110	36	200	126	78	79	15	25	24	15	24	15	22	300	143	450	154	26.0	17	30	130
89110	40	173	110	72	71	8	18	17	10	10	12	13	600	120	750	125	15.0	10	25	106
89110	40	250	150	90	90	25	55	30	25	35	15	15	300	121	600	128	13.0	10	3	120
89110	40	185	120	85	77	23	20	20	15	11	15	12	600	115	750	125	14.0	11	22	115
89110	40	173	110	72	71	8	18	17	10	12	10	13	600	118	750	124	15.0	11	22	106
89110	42	180	120	80	75	10	20	20	15	11	15	12	600	118	750	124	14.0	11	22	110
89110	45	248	148	88	96	23	57	33	24	34	14	13	300	120	600	126	14.0	9	0	119
89110	45	180	120	80	54	13	35	30	22	26	6	17	750	118	900	138	7.0	10	25	96
89110	45	145	145	80	82	9	21	10	9	21	6	12	600	122	750	132	14.0	9	20	125
89110	45	183	124	86	54	13	35	30	22	20	6	7	750	118	900	135	7.0	10	25	96
89110	45	145	148	78	82	9	21	10	9	21	6	12	600	122	750	132	14.0	9	20	0
89110	45	248	148	88	96	23	57	33	24	34	14	13	300	120	600	126	14.0	9	0	119
89110	46	200	140	95	75	22	40	37	26	26	13	15	600	117	750	133	7.0	15	20	120
89110	46	195	150	95	85	14	31	28	30	35	19	25	300	113	600	130	14.0	31	23	125
89110	46	200	140	98	75	22	40	37	26	26	13	15	600	117	750	133	7.0	15	20	120
89110	46	195	150	96	83	14	31	18	17	15	9	16	300	113	600	127	14.0	31	23	125
89110	47	240	126	80	75	36	65	50	30	45	15	16	450	115	600	135	14.0	30	21	100
89110	47	225	132	78	45	15	17	24	12	18	9	11	0	114	1200	134	11.0	26	30	87
89110	47	198	145	72	80	21	38	35	25	22	18	19	600	125	750	140	9.0	5	4	125
89110	47	245	150	90	71	35	55	40	30	30	15	25	750	110	900	135	10.0	10	20	107
89110	47	160	130	75	60	19	33	35	30	35	19	20	600	112	750	128	8.0	0	30	82
89110	47	240	126	78	74	36	0	50	31	35	12	16	450	115	600	130	14.0	30	21	0
89110	47	225	132	78	46	15	17	24	12	18	9	11	0	114	1200	124	11.0	26	30	87
89110	47	198	144	72	80	21	37	34	25	22	18	18	0	0	0	0	9.0	0	4	0
89110	47	156	126	72	57	9	33	22	10	18	8	14	600	114	750	122	8.0	0	34	82
89110	48	210	140	85	70	27	30	22	16	19	19	18	600	114	750	125	5.0	15	16	113
89110	48	260	150	89	71	37	59	43	32	34	9	15	750	117	900	137	10.0	10	20	106
89110	48	222	148	92	77	34	50	45	34	36	16	28	600	131	750	145	15.0	19	16	143
89110	48	195	145	90	82	20	35	35	30	35	20	40	300	110	600	130	10.0	15	15	120
89110	48	222	148	92	72	34	50	45	34	30	16	28	600	131	750	145	15.0	19	16	143
89110	48	210	140	88	70	27	30	22	16	19	14	18	600	114	750	124	5.0	15	16	113
89110	48	260	150	88	71	37	59	43	32	34	9	14	750	117	900	127	10.0	10	20	106
89110	50	225	150	90	78	35	45	48	35	35	20	30	600	130	750	148	13.0	13	15	140
89110	51	165	140	90	58	25	25	25	17	16	6	18	750	120	900	135	16.0	10	25	100
89110	51	206	122	82	72	22	45	37	23	23	9	13	600	115	750	127	14.0	15	31	124
89110	51	206	122	82	72	22	45	37	23	23	9	13	600	115	750	127	14.0	15	31	124
89110	51	165	144	90	55	24	26	26	17	16	6	15	750	120	900	130	16.0	10	25	100
89110	52	212	140	90	77	27	45	43	32	35	7	15	300	110	600	140	8.0	5	30	129
89110	52	198	135	95	85	22	35	28	18	25	20	22	600	125	900	145	13.0	10	18	128
89110	52	191	110	68	59	15	33	15	17	22	11	15	750	116	900	128	14.0	15	31	103
89110	52	191	110	68	59	15	33	15	17	22	11	15	750	116	900	128	14.0	15	31	103
89110	52	212	138	88	77	27	52	43	32	35	7	15	300	101	600	133	8.0	5	30	129
89110	53	262	152	95	57	29	0	53	30	31	15	13	0	0	0	0	9.0	0	0	0
89110	53	260	155	0	89	28	55	55	40	41	20	28	600	120	750	135	10.0	10	20	130
89110	53	262	152	94	57	29	0	53	30	31	15	13	600	88	750	100	0.0	20	20	0
89110	53	243	138	94	84	50	40	36	33	43	10	11	150	126	450	141	15.0	14	5	143
89110	54	204	130	88	80	24	34	27	20	27	16	19	600	122	750	130	14.0	13	32	138
89110	54	160	155	90	75	15	12	10	10	15	10	15	600	115	750	128	10.0	9	29	99
89110	55	180	130	80	91	24	39	36	24	20	16	20	300	111	600	125	12.0	5	23	141
89110	55	150	157	89	70	10	10	10	8	11	8	9	600	111	750	125	9.0	4	32	91
89110	55	208	142	0	105	20	30	35	25	30	20	25	300	110	450	128	8.0	2	18	120
89110	55	176	130	75	75	17	37	19	17	27	10	15	600	120	750	138	8.0	13	24	131
89110	55	150	158	88	69	10	10	10	8	11	8	9	600	111	750	125	9.0	4	32	91
89110	55	204	130	88	80	24	34	27	20	27	16	19	600	122	750	130	14.0	13	32	138
89110	55	180	130	80	91	24	39	36	22	20	16	20	300	111	600	123	12.0	5	23	141
89110	55	208	142	98	109	20	30	21	18	17	5	7	300	116	450	124	10.0	0	15	0
89110	56	170	130	90	75	14	15	15	15	15	5	8	600	112	750	122	17.0	12	25	120
89110	56	185	130	80	80	30	30	25	25	20	10	18	450	114	600	135	13.0	1	10	120
89110	56	195	144	86	77	20	28	26	15	18	7	11	300	112	600	129	9.0	10	18	122
89110	56	170	128	88	76	14	15	15	15	15	5	8	600	113	750	121	17.0	12	25	119
89110	56	184	128	82	80	29	28	36	22	18	10	8	450	114	600	125	13.0	0	10	118
89110	57	151	125	85	65	8	9	10	9	9	7	7	900	125	1050	141	12.0	5	25	120
89110	57	200	130	90	100	12	15	16	14	25	9	10	300	133	600	154	5.0	1	19	167
89110	57	300	180	0	80	40	65	65	60	55	35	45	150	112	300	140	13.0	12	4	140
89110	57	195	150	90	75	30	35	40	25	35	20	25	450	112	600	140	8.0	4	8	135
89110	57	151	126	80	64	8	9	10	9	9	7	7	900	127	1050	131	12.0	5	28	79
89110	57	201	128	88	104	12	15	16	14	25	9	9	300	133	600	154	5.0	0	19	167
89110	57	315	180	0	79	32	0	53	36	41	32	30	0	0	0	0	16.0	12	0	0
89110	57	194	170	106	74	26	34	26	29	29	18	16	450	114	600	134	8.0	10	10	0

89110	58	176	128	66	74	17	37	28	19	17	10	15	600	124	750	135	8.0	13	24	131
89110	58	182	138	84	68	15	18	25	19	23	12	9	300	110	450	129	18.0	0	17	102
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89110	60	178	126	65	90	19	29	25	19	17	6	12	450	118	600	128	20.0	10	15	123
89110	60	198	155	0	72	37	46	29	37	45	26	23	300	110	600	142	15.0	9	10	123
89110	60	165	140	95	70	18	18	13	14	21	10	10	300	112	450	129	16.0	13	26	112
89110	60	210	140	90	80	21	54	50	39	25	18	30	600	125	900	148	3.0	20	22	140
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89110	60	178	126	62	90	19	29	25	19	17	6	12	450	118	600	128	20.0	10	15	123
89110	60	198	154	98	72	37	46	29	37	45	26	13	450	82	600	90	15.0	9	10	77
89110	60	165	140	94	65	18	18	13	14	21	10	10	300	112	450	119	16.0	13	26	112
89110	60	210	140	90	81	21	54	30	29	25	8	13	600	125	900	146	3.0	20	22	140
89110	60	210	122	88	77	22	49	31	18	21	12	20	750	115	900	122	12.0	10	20	111
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89110	61	175	126	80	65	24	25	22	20	22	15	15	750	115	900	135	3.0	7	23	130
89110	61	190	145	0	55	34	31	25	25	20	17	15	900	110	1050	135	16.0	1	20	80
89110	61	236	138	88	86	10	35	39	25	33	15	12	300	113	600	140	18.0	10	15	154
89110	61	265	144	88	91	29	0	42	32	29	36	33	450	113	600	122	14.0	17	0	120
89110	61	173	126	80	66	24	25	22	19	22	5	13	750	115	900	125	3.0	7	23	0
89110	61	187	144	98	56	34	31	26	26	19	17	11	900	115	1050	124	16.0	0	21	78
89110	61	154	132	64	72	8	19	24	24	28	6	9	300	115	450	130	6.0	1	0	118
89110	61	153	158	86	58	18	14	18	18	28	19	9	450	109	600	135	11.0	6	12	90
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89110	62	201	130	80	65	27	27	24	17	32	13	17	900	115	1050	128	9.0	2	18	101
89110	62	220	158	99	63	25	50	34	25	37	9	18	0	0	0	0	10.0	10	0	0
89110	62	201	130	78	61	27	27	24	17	32	13	17	900	115	1050	122	9.0	2	18	101
89110	62	177	130	82	68	37	22	39	27	23	14	9	300	114	450	120	13.0	6	16	109
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89110	63	172	160	88	72	17	26	14	12	17	6	9	300	111	600	135	19.0	10	17	133
89110	63	183	120	76	68	19	27	20	22	22	16	13	0	0	0	0	2.0	1	0	0
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89110	64	180	140	70	60	21	24	23	24	23	13	22	750	120	900	135	12.0	11	24	119
89110	64	254	160	0	60	35	55	45	35	16	35	35	150	110	300	136	6.0	10	3	140
89110	64	182	155	80	69	13	36	16	14	12	10	9	300	99	450	107	15.0	8	13	88
89110	64	160	130	98	66	23	18	16	15	17	6	14	600	140	750	155	12.0	8	20	141
89110	64	218	140	98	93	29	45	39	24	23	7	10	300	121	450	127	12.0	17	13	141
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89110	64	254	160	99	58	31	55	48	35	35	16	32	0	0	0	0	6.0	0	4	0
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89110	67	200	160	90	75	20	22	28	25	22	37	29	600	115	900	148	11.0	20	24	120
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89110	68	170	150	90	80	25	30	15	18	20	10	25	450	120	600	135	13.0	11	20	120
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89110	68	189	168	99	80	21	24	19	18	18	7	12	450	115	600	130	7.0	4	7	101
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89110	69	240	135	90	85	30	65	65	45	55	27	35	300	109	450	135	9.0	6	5	125
89110	69	160	140	80	90	22	22	29	20	18	22	20	300	115	450	132	10.0	6	10	120
89110	69	190	148	82	69	22	28	22	24	19	9	11	450	122	600	137	10.0	7	10	98
89110	69	240	134	92	85	30	0	0	22	32	7	37	300	109	450	134	9.0	6	0	0
89110	69	158	138	78	94	12	12	9	10	12	8	10	300	118	450	122	10.0	0	10	0
89110	69	159	120	80	70	20	26	23	19	20	21	20	300	115	450	147	15.0	1	12	135

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