

FINAL REPORT

USM SHORT TERM GRANT

GRANT NO : 304/PPSP/6131115



**AN INTERVENTIVE AND KAP STUDY ON
HEALTHY LIFESTYLE OF OVERWEIGHT
AND UNCONTROLLED DIABETIC PATIENTS
IN KOTA BHARU, KELANTAN**

1 JUN 2000 – 30 NOVEMBER 2002

RESEARCHERS

**ASSOC PROF AZIZ AL-SAFI ISMAIL (HEAD)
PROF MAFAUZY MOHAMED
PROF RUSLI NORDIN
PROF WAN ABDUL MANAN WAN MUDA
DR NOOR IBRAHIM MOHD SAKIAN**

2004

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And Uncontrolled Diabetic Patients In Kota Bharu, Kelantan.**

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PUSAT PENGAJIAN

TABLE OF CONTENTS

ABSTRACT	xi
Chapter 1 INTRODUCTION	
1.1 Introduction	1
1.2 Study Hypothesis	3
1.3 Objectives	3
1.4 Study Expectations	4
Chapter 2 LITERATURE REVIEW	
2.1 Diabetes Mellitus	5
2.2 Classification of Diabetes Mellitus	5
2.3 Diagnosis of Diabetes Mellitus	9
2.4 Management of Diabetes Mellitus	9
2.4.1 Diet	10
2.4.2 Oral Hypoglycaemic Agents	10
2.4.3 Insulin	11
2.5 Type 2 Diabetes Mellitus	11
2.5.1 Pathogenesis and Natural History	11
2.5.2 Associated Disorders	12
2.5.3 Physical Activity and Type 2 Diabetes	15
2.5.4 Recommendations For Exercise in Type 2 Diabetes	18
2.5.5 Exercise Prescription for Individuals with Type 2 Diabetes	18
2.5.6 Monitoring Intensity of Exercise	22

2.5.7	Resistance training	22
2.5.8	Warm-up and Cool-down	23
2.6	Exercise Recommendations for Blood Glucose Management	23
2.7	Managing Diabetic Complications	24

Chapter 3 MATERIAL AND METHODS

3.1	Subjects	26
3.2	Sampling	26
3.3	Study Design	27
3.4	Assessments	27
3.4.1	Anthropometric and Blood Analysis	27
3.4.2	Cardiorespiratory Fitness Assessment	28
3.5	Exercise Programme	29
3.5.1	Warm-up Phase	30
3.5.2	Exercise Phase	30
3.5.3	Cool-down Phase	31
3.6	Statistical Analysis	31

Chapter 4 RESULTS

4.1	Subjects	32
4.2	Knowledge, Attitude and Practice Questionnaire Results	34
4.2.1	Knowledge Scores	34
4.2.2	Attitude Scores	35
4.2.3	Practice of Exercise Scores	35

4.3	Anthropometric, Body composition and Biochemical Results	40
4.3.1	Body Mass Index	40
4.3.2	Body Fat Percentage and Fat Free Mass	40
4.4	Blood Pressure	42
4.5	Fasting Plasma Glucose	42
4.6	Glycosylated Hemoglobin	43
4.7	Lipid Profile	43
4.8	Fasting Insulin	44
4.9	Fasting C-Peptide	44
4.10	Cardiorespiratory Fitness	44
4.11	Resting Heart Rate	45

Chapter 5 DISCUSSION

5.1	Body Mass Index and Body Composition	46
5.2	Blood Sugar Control	48
5.3	Fasting Plasma Insulin and C-Peptide Level	49
5.4	Lipid Profiles	50
5.5	Blood Pressure	50
5.6	Cardiorespiratory Fitness	52
5.7	Knowledge, Attitude and Practice Study	52
5.8	Limitations of Study	53

CONCLUSION AND RECOMMENDATIONS

BIBLIORAPHY

APPENDICES

Appendix I	:	Progress Report	67
Appendix II	:	Abstract Submission Form	70
Appendix III	:	Exercise Log Book	71
Appendix IV	:	Statement Report	72
Appendix V	:	Abstract	73

ABSTRACT

OBJECTIVES: To assess the effects of a regular exercise programme on metabolic control and also knowledge, attitude and practice (KAP) of exercise and physical activity among Type 2 diabetes mellitus patients.

MATERIAL AND METHODS: Forty two Type 2 diabetes patients from the community medicine and medical specialist clinics in USM were assigned to two groups; The interventional group consisted of 13 males and 14 females, mean age 52.6(8.7) (mean (SD)), body mass index 27.2 (3.9) participated in regular group exercise sessions (3 times a week, 1 hour sessions) for 7 weeks. Exercise intensity was predetermined based on the predicted target heart rate for each subject. The control group consisted of 7 males and 8 females, mean age 56.5(11.1), body mass index 25.8(2.73). Pre and post-intervention anthropometric measurements and blood investigations were done. These include height, weight, body mass index, body fat percentage and fat free mass (FFM), blood pressure, cardiorespiratory fitness level, fasting plasma glucose level (FPG), HbA1c level, fasting lipid profile, insulin and C-peptide level.

RESULTS: In the intervention group, FFM increased from 45.98(9.71) to 47.93(10.53) kg.; percentage body fat decreased from 32.37(11.02) to 30.14(10.73)%. Diastolic pressure reduced from 91(11.2) to 83.3(7.5) mmHg, cholesterol level decreased from 6.23(1.03) to 5.74(1.23) mmol/l, HDL level increased from 0.97(0.31) to 1.19(0.28) mmol/l. Estimated VO_2 max increased from 21.6(7.04) to 27.7(8.72) ml/kg/min. Resting heart rate declined significantly from 80(7) to 73(11) bpm. There was no improvement in body mass index (BMI), fasting plasma glucose, insulin and C-peptide levels. Knowledge and attitude scores were higher than practice scores.

CONCLUSION: Seven weeks of exercise intervention among type 2 diabetes patients resulted in an improvement in body composition, cardiorespiratory fitness, lipid profile and a reduction in blood pressure. However there is a disparity between knowledge and practice of exercise and physical activity.

CHAPTER 1

INTRODUCTION

1.1

Recent figures from *Diabetes Atlas 2000* published by the International Diabetes Federation (IDF) revealed that over 150 million adults around the world have diabetes, an 11% increase in just 5 years (1995-2000)(IDF, 2000). The prevalence of diabetes is presently higher in developed countries than in developing countries, but the developing world will be more seriously affected by the escalating diabetes epidemic in the future. Even more alarming is the increasing numbers of younger (and productive) diabetics seen especially in developing countries. This substantial rise in diabetes is contributed to the urbanisation, westernisation and economic development in developing countries (Jerval, 1997). The above changes inadvertently bring about concomittant changes in lifestyle and diet, the two most important external contributing factors for type 2 diabetes. Morbidity and mortality in type 2 diabetes is mainly due to cardiovascular disease, as a result of hypertension, dyslipidemia, smoking, obesity and lack of exercise (Fuller et al, 1983). In Malaysia, the prevalence of diabetes ranges from 4-8% (Mustaffa, 1990) and is expected to exceed 10% by the year 2020 (Anuar, 1997). This means that with a 21 million population estimate in 1996, the number of diabetics in Malaysia would number about 1.7 million. Of these, about 95% would be having type 2 diabetes mellitus (Ministry of Health 1997). Management of this serious health threat requires effective preventive and therapeutic measures. Prevention of diabetes either at the primary or secondary level is directed primarily towards a change in lifestyle and diet i.e. from a sedentary to a more physically active lifestyle coupled with a healthy and balanced diet. Primary treatment of type 2 diabetes is mostly non pharmacological i.e. diet therapy combined with exercise. This treatment modality has in fact been

recommended for the treatment of diabetes for over 2000 years and is still effective when optimally utilised (Eriksson, 1999). Exercise is also one of the three main approaches (including diet and education) to managing type 2 diabetes and impaired glucose tolerance (IGT) patients in Malaysia (Ministry of Health, 1997). A trial of therapy for at least 3-6 months of prudent exercise, diet and a healthy lifestyle should be advocated before any oral hypoglycaemic agents (OHA) are prescribed. Even when OHA are being used, emphasis should still be put on a proper diet, regular exercise and compliance at all levels.

Guidelines on a suitable diet for type 2 diabetics are usually given by the attending physician or when available by a resident dietitian. However advice on exercise is usually provided by the attending physician based on the guidelines provided by the Ministry of Health (Ministry of Health, 1997). As there will be different levels of involvement in physical activity or exercise in different community groups (Walker et al, 1999), the exercise programme prescribed has to be 'tailored' to suit the different groups, especially so for the majority of type 2 diabetics who would most likely be elderly and/or obese. The experience of the Universiti Sains Malaysia (USM) obesity clinic has shown that obese subjects who underwent a proper weight reduction programme in the form of dietary counselling, behaviour modification and exercise achieved a significant amount of weight loss (Wan Manan et al, 1998). In another similar study, dietary and physical activity intervention resulted in significant increase in VO_2 max and decrease in weight, body mass index (BMI), fasting glucose levels, insulin, total cholesterol, triglycerides and systolic blood pressure. In this study the type of exercise used was brisk walking, step aerobics or line dancing, 5 days a week (Wing et al, 1998).

The current project aims at evaluating the effects of a structured exercise programme in a group of type 2 diabetic patients at USM Kubang Kerian.

1.2 Study hypothesis

A well-managed and structured exercise programme can improve metabolic control, body composition, lipid profiles and cardiorespiratory fitness of type 2 diabetes mellitus patients.

1.3 Objectives

1. To determine the proportion of overweight (BMI > 25 kg/m²) and obesity (BMI > 30 kg/m²) among type 2 diabetes patients.
2. To determine the proportion of uncontrolled diabetes (HbA1c > 8%) or fasting plasma glucose (FPG) > 7 mmol/l in type 2 diabetes patients.
3. To determine the proportion of hypertension (systolic blood pressure (SBP) > 140 mm Hg, diastolic blood pressure (DBP) > 90 mm Hg).
4. To determine the levels of fasting plasma insulin and C-peptide in type 2 diabetes patients.
5. To determine the levels of triglyceride, total cholesterol and high density lipoproteins in type 2 diabetes patients.
6. To estimate the cardiovascular fitness of type 2 diabetes patients.
7. To assess the effects of a regular structured exercise programme on all the above mentioned parameters.
8. To assess the knowledge, attitude and practice of exercise in type 2 diabetes patients.

1.4 Study Expectations

Exercise interventional studies done at other centers have shown positive changes in metabolic control (FPG, HbA1c, insulin, C-peptide levels), lipid profile, body composition (body mass index (BMI), percentage body fat, fat free mass) and reduction in blood pressure (Mourier et al, 1997; Yamanouchi et al, 1995; Lehmann et al, 1995). The present study is an attempt at establishing a suitable and effective exercise programme which could be easily followed by the local subjects and thus could consequently produce similar positive changes in the metabolic control of type 2 diabetes patients.

CHAPTER 2

LITERATURE REVIEW

2.1 Diabetes Mellitus

Diabetes mellitus is a group of metabolic disorders characterized by chronic hyperglycemia resulting from abnormalities in insulin secretion, insulin action or both.

Diabetes is associated with long-term complications that affect almost every part of the body. It is associated with long-term damage, dysfunction and failure of multiple organs especially the eyes, kidneys, heart and blood vessels. The disease often leads to blindness, heart and blood vessel disease, strokes, kidney failure, diabetic gangrene, and nerve damage. Uncontrolled diabetes can complicate pregnancy, and birth defects are more common in babies born to women with diabetes. Signs and symptoms of marked hyperglycemia include polyuria, polydipsia, weight loss, and blurred vision. Growth may be impaired and patients are more susceptible to infections. Acute emergencies of diabetes include hyperglycemia with ketoacidosis or non ketotic hyperosmolar syndrome. Patients with diabetes have an increased incidence of atherosclerotic cardiovascular, peripheral vascular and cerebrovascular disease. Hypertension and abnormalities of lipoprotein metabolism are often found in diabetic patients. The emotional and social impact of diabetes and the demands of therapy may cause significant psychosocial dysfunction in patients and their families.

2.2 Classification of Diabetes Mellitus

The World Health Organisation (WHO) Expert Committee on diabetes in 1980 and later the WHO Study Group on Diabetes Mellitus (1985) recognized two major forms of diabetes; insulin-dependent diabetes mellitus (IDDM, type 1 diabetes) and non-insulin –

dependent diabetes mellitus (NIDDM, type 2 diabetes) including three other distinct types viz. gestational diabetes mellitus (GDM), malnutrition-related diabetes mellitus (MRDM) and other types (WHO 1985). The WHO and the National Diabetes Data Group (NDDG) highlighted the heterogeneity of the diabetic syndrome which share hyperglycemia in common. The degree of hyperglycemia may change over time, depending on the extent of the underlying disease (Figure 2.1) (American Diabetes Association (ADA) Expert Committee Report, 1998).

The vast majority of cases of diabetes falls into two broad categories. In one category (type 1 diabetes), the cause is an absolute deficiency of insulin secretion. Individuals at increased risk of developing this type of diabetes can often be identified by serological evidence of an autoimmune pathologic process occurring in the pancreatic islets and by genetic markers. In the other, much more prevalent category (type 2 diabetes), the cause is a combination of resistance to insulin action and an inadequate compensatory insulin secretory response. In this category, a degree of hyperglycemia sufficient to cause pathologic and functional changes in various target tissues, but without clinical symptoms, may be present for a long period of time before diabetes is detected. During this asymptomatic period it is possible to demonstrate an abnormality in carbohydrate metabolism by measurement of plasma glucose in the fasting state or after a challenge with an oral glucose load. The main characteristics of the two main types of diabetes (type 1 and 2) are listed in TABLE 2.1.

TABLE 2.1 Characteristics of Type 1 and Type 2 Diabetes Mellitus

Characteristics	NIDDM	IDDM
Patient at risk	Positive family history Obese Hypertensive	Positive family history
Onset	Insidious Older age group at onset >40 years	Acute Younger age at presentation, <40 years
Classical symptoms	Sometimes absence of classical symptoms Absence of ketonuria or ketoacidosis	Polyuria Ketonuria Ketoacidotic Loss of weight
Immune markers	C-peptide positive GAD antibody usually negative	Presence of islet cell antibody (for 18 months or so after diagnosis) GAD antibody positive
Genetic markers	No characteristic HLA type	Particular HLA type: DQ, DR3, DR4, DR3/DR4
Treatment	Diet, oral hypoglycaemic agent or sometimes insulin	Need insulin for life

2.3 Diagnosis of Diabetes Mellitus

Clinical diagnosis of diabetes is based essentially on presence of hyperglycemia. Based on WHO diagnostic criteria (World Health Organisation, 1985), a fasting plasma glucose (FPG) concentration greater than 7.8 mmol/l or a random plasma glucose concentration greater than 11.1 mmol/l is diagnostic of diabetes. If blood glucose levels are borderline, and there is cause for doubt about the presence of diabetes, a glucose tolerance test (GTT) should be performed. In symptomatic patients, a single abnormal blood or plasma glucose level is adequate for the diagnosis of diabetes; but if symptoms are absent, then two abnormal levels are needed.

2.4 Management of Diabetes

The goal of diabetes management is to relieve symptoms, to enable the patient to lead a normal life, to prevent acute and chronic complications, to reduce mortality and to treat accompanying disorders. The principal means to achieve the above objectives is by keeping blood glucose levels as close to the normal range as safely possible. Studies of patients with type 1 and type 2 diabetes have provided clear evidence that therapeutic approaches should target the lowest blood glucose levels attainable without unacceptable hypoglycemia or other adverse effects. Long-term results of the Diabetes Control and Complications Trial (DCCT) on type 1 diabetes (DCCT, 1993) as well as the United Kingdom Prospective Diabetes Study (UKPDS, 1998) and the Japanese Kumamoto Study on type 2 diabetes (Ohkubo *et al*, 1995) has shown that intensive therapy aimed at achieving tight glycaemic control reduces the micro- and macrovascular complications associated with diabetes. The main therapeutic tools commonly used to achieve this target include lifestyle modifications (diet and physical

activity/ exercise), oral hypoglycemic agents and/ or insulin. These are often prescribed singly or in combination to diabetic patients to achieve optimal glycaemic control.

2.3.1 Diet

Diet modification is the prime treatment for type 2 diabetes, whereas for type 1 diabetics it is an important secondary treatment option. The mainstay of diet therapy for Type 2 diabetics who are obese is restriction of saturated fat <10 % of total dietary energy, reduction of energy intake and lowering of body weight. Dietary management of type 1 diabetics should aim in the short term to prevent hypoglycemia and ultimately reduce the risks of chronic diabetic complications and improve life expectancy. Dietary advice must take into account the individual preferences, attitudes and lifestyle in order for it to be effective (Pickup and Williams, 1997).

2.4.2 Oral Hypoglycemic Agents (OHA)

These are primarily used to treat type 2 diabetes and are used to correct the underlying metabolic disorders, namely insulin resistance and inadequate insulin secretion. The three main groups are the sulphonylureas, the biguanide metformin and the α -glucosidase inhibitor acarbose. Sulphonylureas such as glibenclamide and gliclazide lower blood glucose primarily by stimulating the second phase of insulin secretion. Metformin lowers blood glucose levels via several mechanisms, including inhibition of gluconeogenesis in the liver and thus suppression of hepatic glucose production; improving peripheral insulin sensitivity and promoting glucose uptake and storage in muscle. Metformin also causes weight loss (unlike sulphonylureas) and is therefore useful for obese patients. The α -glucosidase inhibitor acarbose, delays carbohydrate absorption from the gut and is usually used together with the other OHA. Other newer

drugs include the insulin-sensitising thiazolidinediones and insulin secretagogues (Pickup and Williams, 1997).

2.4.3 Insulin

Insulin is used in various forms and regimes to treat both type 1 and type 2 diabetes.

Insulin via injection or an insulin pump are the basic therapies for type 1 diabetes. The amount of insulin must be balanced with food intake and daily activities. Only type 2 diabetics who fail to respond to combinations of OHA are treated with insulin injections.

2.5 Type 2 Diabetes Mellitus

In Malaysia, the majority of diabetic patients, up to 95%, fall into this category of diabetes (Ministry of Health Malaysia, 1997). The actual aetiology of this syndrome is not known. However, epidemiological data indicate that both genetic and environmental factors are involved. Identified risk factors for type 2 diabetes are age, gender, inheritance, obesity (degree and duration), body fat distribution, physical inactivity, dietary components (Perry *et al*, 1993), together with hyperinsulinemia and insulin resistance (Lundgren *et al*, 1990; Boyko *et al*, 1991).

2.5.1 Pathogenesis and Natural History

Type 2 diabetes can be viewed as the consequence of a series of pathophysiologic changes; each of which makes the patient vulnerable to subsequent disruption of normal glucose homeostasis. The pathogenesis of type 2 diabetes involves the development of insulin resistance associated with compensatory hyperinsulinemia, followed by progressive β -cell impairment that results in decreasing insulin secretion and

hyperglycemia. Insulin resistance denotes the inability of insulin to produce its usual biological effects at circulating concentrations that are effective in normal subjects. Hyperglycemia itself causes additional inhibition of insulin secretion and more insulin resistance (glucose toxicity), which further accentuates the hyperglycemia. Thus the development of type 2 diabetes is usually characterized by impaired insulin action (insulin resistance) and deficient insulin secretion (β -cell abnormalities) (Groop *et al*, 1993). The above suggested natural history of type 2 diabetes is illustrated in Figure 2.2. The mode of insulin secretion during this pathological process has been referred to as the 'Starling curve of the pancreas' (Figure 2.3). Another contributing metabolic defect is an elevated hepatic glucose production due to hepatic insulin resistance (De Fronzo *et al*, 1992). These pathological processes may take several years and are not necessarily irreversible. Ideally the treatment modality chosen should be able to modify all of the abovementioned abnormalities. However this kind of treatment is still not available although various interventions such as weight loss, increased physical activity and also insulin sensitising drugs are able to reduce the likelihood of progression to overt type 2 diabetes (Eriksson, 1999).

2.5.2 Associated Disorders

Type 2 diabetes is often associated with other metabolic abnormalities which Reaven in 1988 called them as Syndrome X. The metabolic syndrome is a multifaceted syndrome characterised by five major abnormalities: obesity, hypertension, insulin resistance, glucose intolerance (impaired glucose tolerance/type 2 diabetes) and dyslipidemia (hypertriglyceridemia and low HDL-cholesterol). In addition, a number of other abnormalities such as microalbuminuria, hyperuricemia, hyperfibrinogenemia and

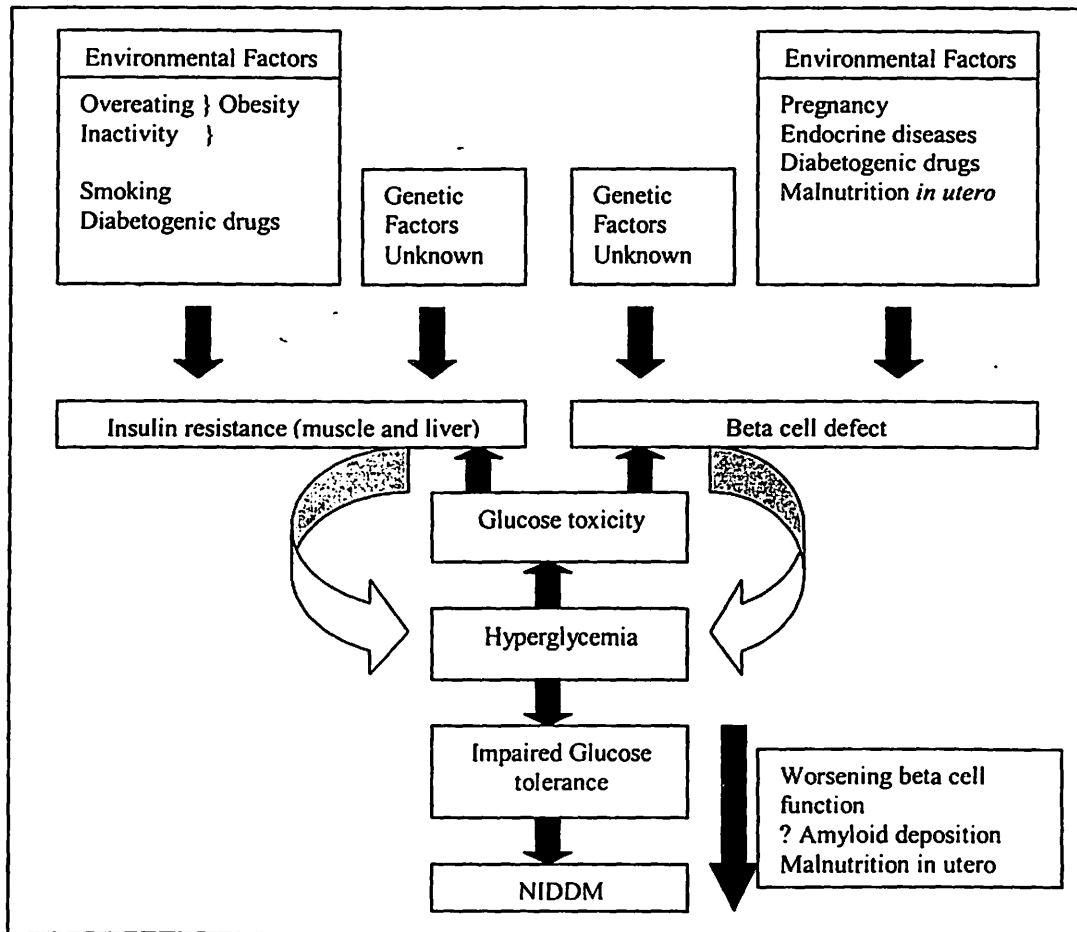


Figure 2.2 Hypothetical scheme of the natural history of type 2 diabetes showing possible interactions between insulin resistance and beta cell function (Pickup and Williams, 1997)

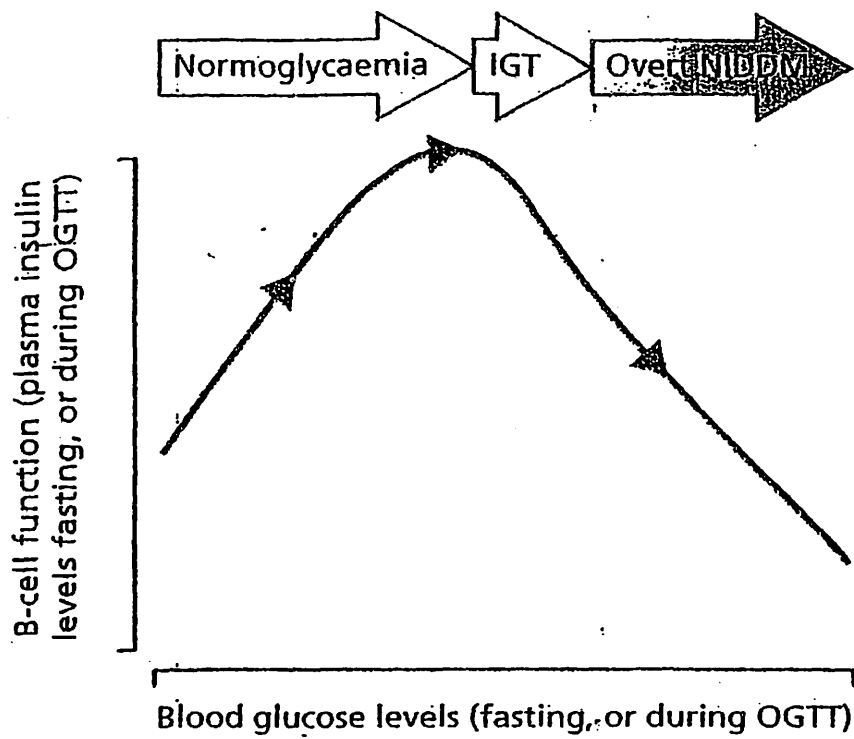


Figure 2.3 The 'Starling curve of the pancreas'. Insulin secretion increases initially to overcome insulin resistance, but reaches a plateau and then fails. Blood glucose levels rise initially within the normal range, then into the category of impaired glucose tolerance (IGT), and finally into overt type 2 diabetes. (Pickup and Williams, 1997)

increased levels of plasminogen activator inhibitor I and low concentrations of tissue plasminogen activator are often associated with the syndrome. The clinical importance of this syndrome is the clustering of simultaneously occurring atherosclerotic risk factors in the same individual (Eriksson *et al*, 1997). Thus when discussing treatment for type 2 diabetes we should not only concentrate on the reduction of hyperglycemia, but also consider all the other usually coexisting metabolic abnormalities (e.g. obesity, dyslipidemia, hypertension). In other words, the treatment goal is to correct all metabolic abnormalities and to lower the increased morbidity and mortality associated with the disease.

2.5.3 Physical Activity and Type 2 diabetes

The World Health Organisation (WHO) defines physical activity as all movements in everyday life, including work, recreation, exercise and sporting activities (WHO, 1997). Why exercise? The reason is simple, because exercise has numerous benefits ranging from a feeling of overall well being to achieving specific health benefits. Indeed high physical activity levels have been associated with lower risk of developing cardiovascular disease (Paffenbarger *et al*, 1994) and a reduced chance of developing type 2 diabetes (Manson *et al*, 1992). Furthermore it has been shown in several recent epidemiological studies that the level of physical activity is associated with a lowered incidence of type 2 diabetes (Manson *et al*, 1991, 1992; Perry *et al*, 1993). A lower prevalence of diabetes was also found among female former college athletes as compared with non-athletes (Frisch *et al*, 1986). A ten year study of 200 type 2 diabetic patients enrolled in a physical training program showed an increase in VO_2 max, reduction in blood pressure, heart rate, body weight, fasting plasma glucose, glycosylated hemoglobin and triglycerides (Schneider *et al*, 1992). Exercise has a

pronounced effect on substrate utilisation and muscle glucose uptake can increase by up to 20-fold during exercise (Eriksson, 1999). Exercise-induced stimulation of glucose uptake may involve several factors. Firstly, increased muscle blood flow enhances insulin delivery to muscle by opening previously non-perfused capillaries, thus increasing both the effect of insulin and the surface area for glucose transport. Secondly, muscle contraction can stimulate glucose transport, possibly through translocation of glucose transporters (GLUT-4) from an intracellular pool into the cell membrane. Exercise and insulin have additive effects on muscular glucose transport, indicating two separate glucose transport systems; one insulin dependent and the other contraction dependent (Pickup and Williams, 1997).

Physical training enhances whole-body as well as hepatic insulin sensitivity (DeFronzo *et al*, 1987). The mechanisms contributing to the enhanced insulin sensitivity in endurance trained athletes are increased muscle blood flow, elevated muscle glucose transport protein (GLUT-4) levels and glycogen synthase activity (Houmard *et al*, 1991). This improvement in glucose disposal lasts for months after the training programme ends. During intensive training programmes, glycaemic control also improves. A period of as short as 1 week of intense physical training (68% VO_2 max) has been shown capable of improving oral glucose tolerance in type 2 diabetic men (Rogers *et al*, 1988). Glycosylated hemoglobin levels may fall by 14% after 6 weeks of training, due to the cumulative effects of individual bouts of exercise (Schneider *et al*, 1984). Physical training also improves cardiovascular risk profiles: 20% decrease in triglycerides, 23% increase in high-density lipoprotein levels, reduces systolic and diastolic blood pressures (Horton, 1988; Lehmann *et al*, 1995) and reduces visceral adipose tissue (VAT) (Mourier *et al*, 1997). However habitual, less intensive physical exercise does not necessarily improve glycaemic control (Vanninen *et al*, 1992) nor

insulin, lipid profile or lipoprotein levels (Lampman *et al*, 1987). Improved glycemic control as reflected in glycosylated hemoglobin or fasting plasma glucose levels has also been documented after 6 to 12 weeks of an aerobic exercise programme (Devlin, 1992). This enhanced glycemic control appears to result not only from the acute effects of exercise (via increased skeletal muscle glucose disposal and repeated cycles of glycogen depletion and resynthesis) but also from a true training effect. A single exercise session has been shown to increase insulin sensitivity for 16 hours or longer (Devlin *et al*, 1987) and a particularly intense bout (85% of VO₂ max, continued to exhaustion) is able to increase glucose utilisation 12 hours later to a level comparable to what is achievable after 12 weeks of moderate training (Devlin, 1992). However, insulin sensitivity appears to correlate with increased capillarisation of skeletal muscle (Richter *et al*, 1992) and increased capillary density (by reducing in diffusion distance) may also enhance positive metabolic changes in obese diabetics who exercise (Maynard, 1991). With repeated exercise, muscle hypertrophy (with increased glucose metabolism) and increases in VO₂ max can occur. In addition, psychological benefits associated with exercise such as improvements in mood, self-esteem, and quality of life are particularly important for people having chronic diseases such as diabetes (Rodin, 1989). Exercise and organised sports allow diabetic patients to participate in social activities. Mature diabetic patients can enjoy the company of similarly aged participants in an exercise programme or while walking in the park. This active participation promotes socialization, peer acceptance and personal esteem. Having exercise as part of their treatment regimen allows patients to take an active and positive role in management of their disease.

2.5.4 Recommendations for Exercise in Type 2 diabetes

Undoubtedly exercise training plays an important role in the treatment of type 2 diabetes. It however remains a problematic treatment option. Just as diet and drug therapy, exercise has to be prescribed for the individual depending on the overall condition of the patient. The exercise prescribed should ideally bring about a regular and permanent change in physical activity levels as only then will it benefit the patient. Frequently asked questions are, “How much exercise is enough?” and “What type of exercise is best for developing and maintaining fitness?”. Before these questions can be answered a distinction has to be made between physical activity as it relates to health versus fitness. The American Colleges of Sports Medicine (ACSM, 1998) pointed out that the quantity and quality of exercise needed to attain health-related benefits may differ from what is recommended for fitness improvements. Lower levels of physical activity (particularly intensity) are generally required to exert positive metabolic changes and reduce risks for certain chronic degenerative diseases (ACSM, 1998). Even regular daily walking combined with diet therapy not only reduced body weight but also improved insulin sensitivity (Yamanouchi *et al*, 1995). Advice should thus be tailored to the individual’s need taking into account his or her fitness and interests.

2.5.5 Exercise Prescription for Individuals With Type 2 Diabetes

Before beginning an exercise program, individuals with diabetes should have a medical evaluation to screen for macrovascular and microvascular complications that may be exacerbated by exercise. The examiner should screen for cardiovascular health, peripheral arterial disease, retinopathy, nephropathy, and peripheral and autonomic neuropathy. A graded exercise test is recommended to screen for cardiovascular disease

in individuals who meet any of the criteria in Table 2.2 and who wish to participate in moderate- to high-intensity exercise programs (American Diabetes Association, 2000).

The American College of Sports Medicine (ACSM) recommendations (Table 2.3) for all individuals (including those without diabetes) is that aerobic physical activity be done a minimum of 3 to 5 days a week, for 20 to 60 minutes at 40% to 85% of maximum oxygen uptake reserve (VO_2R or heart rate reserve [HRR]), or at 55% to 90% of maximal heart rate (MHR)(Pollock *et al*, 1998). For less conditioned individuals, exercise can be done at the lower intensity level for a longer duration, at least until a higher level of fitness can be achieved. Individuals with type 2 diabetes should especially be encouraged to progress to a higher total duration of exercise (e.g. 1 hour daily) to facilitate fat loss.

TABLE 2.2 Criteria* for Assessing the Need for Graded Exercise Testing Among Patients Who Have Type 1 or Type 2 Diabetes (American College of Sports Medicine (ACSM) Recommendations 1998)

Age > 35 years
Type 2 diabetes > 10 years duration
Type 1 diabetes > 15 years duration
Presence of any additional risk factor for coronary artery disease
Presence of microvascular disease (proliferative retinopathy or nephropathy, including microalbuminuria)
Peripheral vascular disease
Peripheral and autonomic neuropathy

*If an individual meets any one of these criteria, exercise testing is recommended prior to participation in moderate- to high-intensity exercise programs.

TABLE 2.3 American College of Sports Medicine Guidelines for Aerobic Exercise Programs (American College of Sports Medicine (ACSM) recommendations 1998)

Exercise Characteristic	Recommendation*
Mode	Continuous, rhythmic, prolonged activities using the large muscle groups of the arms and/or legs
Intensity	Range of 55%-90% of maximal heart rate, 40%-85% of VO ₂ R or HRR, or RPE of 12-16 (somewhat hard to hard)
Duration	Minimum of 20-60 min of continuous aerobic activity to improve fitness and endurance capacity
Frequency exercise	Minimum of 3-5 days/wk, with frequency determined by duration and intensity
Rate of progression mo,	Initial conditioning of 4-6 wk, improvement phase lasting 4-5 mo, and maintenance thereafter

*Resistance-type and flexibility training are recommended 2-3 days per week.

VO₂R = VO₂ reserve; HRR = heart rate reserve; RPE = rating of perceived exertion

2.5.6 Monitoring Intensity of Exercise

Exercise intensity can be prescribed and monitored in several ways. Oxygen uptake reserve (VO₂R) is a percentage of the difference between maximal and resting oxygen uptake (VO₂) (Swain and Leutholtz, 1997). If metabolic data from an exercise stress test are available, a target VO₂ can be determined at a desired percentage of VO₂R and then translated into a workload for a given exercise. For practical purposes, exercise intensity is usually established using heart rate.

Calculation of a training heart rate or range using the heart rate reserve (HRR) method (Karvonen equation) is done with the following formula:

$$\text{Training HR} = \text{intensity fraction} \times (\text{HR}_{\text{max}} - \text{HR}_{\text{rest}}) + \text{HR}_{\text{rest}}$$

where the intensity fraction is chosen between 0.40 and 0.85 (the lower end of the range is used for patients with low initial fitness) and HR rest is the resting heart rate. A known value of maximum heart rate (HR_{max}), obtained from a maximal stress test, should be used if available; otherwise, HR_{max} may be estimated by subtracting the patient's age from 220.

For example, calculation of a training HR at 50% of HRR for a patient with a maximal HR of 170 beats per minute (bpm) and a resting HR of 80 bpm would result in a training HR of 125 bpm :

$$\text{Training HR} = 0.50 \times (170 - 80) + 80 = 45 + 80 = 125 \text{ bpm.}$$

2.5.7 Resistance training

More recently resistance training has been shown to have a positive effect on metabolic profile of type 2 diabetes patients (Eriksson et al, 1997; Honkola et al, 1997). Resistance or weight training that includes at least one set of each of 8 to 10 different exercises using the major muscle groups is recommended 2 to 3 days a week. Each set should

consist of 8 to 12 repetitions, with the amount of weight increased when the individual can complete 12 or more repetitions. For those age 50 or older who have diabetes or preexisting health concerns such as hypertension, more repetitions (12 to 15) done at a lower weight may be more suitable. Flexibility training should be incorporated into the overall fitness routine a minimum of 2 to 3 days per week to develop and maintain joint range of motion and to minimise the potential loss of flexibility resulting from glycosylation of various joint structures. Stretching for 5 to 10 minutes should be done either after an aerobic warm-up or following the completion of an exercise session.

2.5.8 Warm-up and Cooldown

No matter what type of activity is done, the standard recommendation for all individuals (with or without diabetes) is to include proper warm-up and cooldown periods. A warm-up consists of 5 to 10 minutes of a lower-intensity aerobic activity that uses the same muscles that will be exercised at a higher intensity (e.g. walking before starting to jog). The cooldown consists of 5 to 10 minutes of less intense activity. Such activities may ease the cardiovascular transition between rest and exercise and help prevent muscle and joint injuries.

2.6 Exercise Recommendations for Blood Glucose Management

For individuals with type 2 diabetes who are not using supplemental insulin, the risks for hypoglycemia are less compared to patients with type 1 diabetes. Supplemental carbohydrates are generally not needed in these patients; however, blood glucose monitoring will reveal which individuals may need additional carbohydrates to prevent hypoglycemia during and following exercise. Use of certain oral hypoglycemic agents such as sulfonylureas carries a higher risk of exercise-induced hypoglycemia due to their longer half-lives. Other recommendations concern safe participation for type 2

exercisers with existing or developing diabetes-related complications such as cardiovascular disease, hypertension, neuropathy, or microvascular changes.

2.7 Managing Diabetic Complications

People with diabetes may experience a variety of macrovascular and microvascular complications that can complicate exercise. Any individual with a high risk for underlying cardiovascular disease should undergo a graded exercise test before beginning a moderate- to high-intensity exercise program (Swain and Leutholtz, 1997). For exercise involving the feet, precautionary measures are recommended, especially if peripheral neuropathy is present. Use of silica gel or cushioned midsoles, polyester or synthetic-blend socks, and proper footwear is essential to prevent blisters, to keep the feet dry, and minimise or prevent trauma. Non-weight-bearing exercise such as aqua aerobics can be substituted for weight-bearing activities. Hypotension and hypertension following vigorous exercise are more likely to develop in individuals with autonomic neuropathy. Their thermoregulatory capacity may be inadequate, and special care is needed to maintain adequate hydration. Active, strenuous exercise is contraindicated for individuals with active vitreous hemorrhages due to unstable proliferative retinopathy. Intense exercise involving straining, jarring, or Valsalva-like maneuvers should be avoided as well. When nephropathy is present, individuals may have a reduced exercise capacity, but low to moderate-intensity activities can be done. Although the above general exercise recommendations can be helpful, physicians may have to aid patients in modifying diet and/or insulin regimens because the recommendations require tailoring to meet individual needs. Furthermore, if a patient's pre-exercise metabolic control is poor or diabetes-related complications are present, clinical recommendations should be followed to prevent worsening or onset of complications. Exercise can be