INTERNATIONAL JOURNAL OF INNOVATIVE RESEARCH & DEVELOPMENT

ISSN 2278 – 0211 (Online)

Mango Leaves in Treating Diabetes: A Strategic Study

Dr Rajnandan Patnaik
Professor, IMT Nagpur, India

Abstract:
Type 2 Diabetes Mellitus (DM) results from insulin resistance, a condition in which cells fail to use insulin properly, sometimes also with an absolute insulin deficiency. This form was previously referred to as non insulin-dependent diabetes mellitus (NIDDM) or "adult-onset diabetes". Prevention and treatment often involve: a healthy diet, physical exercise, not using tobacco, and being a normal body weight (WHO, 2013). Type 2 diabetes may be treated with medications with or without insulin. Insulin and some oral medications can cause low blood sugar, which can be dangerous.
The methodology pertaining to the study - Effect of Mango Leaves on treating Diabetes, was sequenced under the heads, viz. selection of area, selection of sample population (patients), collection of data and conducting the study, diet counseling and questionnaire-cum-interview schedule, administration process of mango-leaf powder and finally the evaluation of blood glucose level, before and after, administration of diet counseling, medicines and mango-leaf powder.
The sample of 30 patients were broken down in three groups, Group-A (who were on counseled diet and medication), Group-B (who were on counseled diet and medication, were administered mango-leaf powder), and Group-C (who were on counseled diet and medication, and were administered a placebo in the form of mint-leaf powder). The blood glucose level of all the subjects (30 in number) in three groups were tested and recorded. Fasting and post-prandial sugar levels were recorded and assessed. The results of the three groups were compared to see the effectiveness of the mango-leaves in controlling diabetes.
Group A proves that there was a reduction of blood sugar level (FBS and PP correspondingly) of the patients after about a month, during which time they followed a strict diet and medication. Group B proves that there was remarkable reduction of blood sugar level (FBS and PP correspondingly) of the patients after about a month, during which time they followed a strict diet and medication and mango-leaf solution. Group C proves that there was a slight reduction of blood sugar level (FBS and PP correspondingly) of the patients after about a month, during which time they followed a strict diet, medication and placebo (mint-leaf). The study confirms that the subjects who were under diet counseling, medication and mango-leaf powder showed considerable reduction in blood sugar level.

1. Introduction

Diabetes Mellitus is Greek word, which means sweet fountain. Diabetes Mellitus is a metabolic disorder, which is caused by the deficiency of the hormone insulin or the inability of the body cells to use the available insulin produced from pancreas. The normal blood sugar in adult is between 70-120 mg/dl during fasting and 120-140 mg/dl during post prandial. If the blood sugar is consistently more than 120-140 mg/dl, it is called diabetes.

Diabetes is a major global health problem. The World Health Organization estimates that Diabetes primarily, Type 2, affects 4% of the world's adult population, amounting to about 135 million people. If current trends continue, they anticipate that number to essentially double, growing to 300 million people by 2025 (122% growth) about 75% of whom will live in developing countries. India now has the largest population of diabetes internationally (19 million). India is also expected to show the highest increase in prevalence internationally by 2025 (195% growth).

India leads the world with largest number of diabetic subjects earning the dubious distinction of being turned the diabetes capital of the world. According to the Diabetes Atlas (2006), published by the International Diabetes Federation, the number of people with diabetes in India currently around 40.9 million is expected to rise to 69.9 million by 2025, unless urgent preventive steps are taken.

About 630 deaths (about 4.4% of total deaths) in Odisha happened due to Diabetes and comparing this with the national figures, Odisha remains on the top part of the table. As urban cases of diabetes are more prevalent and Rourkela being the third largest urban city in Odisha, this study is not only pertinent, but also important.

2. Review of Literature

The classic symptoms of untreated diabetes are weight loss, polyuria (frequent urination), polydipsia (increased thirst), and polyphagia (increased hunger) Cooke & Plotnick, 2008). Symptoms may develop rapidly (weeks or months) in type 1 diabetes, while they usually develop much more slowly and may be subtle or absent in type 2 diabetes. Prolonged high blood glucose can cause glucose absorption in the lens of the eye, which leads to changes in its shape, resulting in vision changes. Blurred vision is a common complaint leading to a diabetes diagnosis. A number of skin rashes that can occur in diabetes are collectively known as diabetic dermadromes.

All forms of diabetes increase the risk of long-term complications. These typically develop after many years (10–20), but may be the first symptom in those who have otherwise not received a diagnosis before that time. The major long-term complications relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease (Emerging Risk Factors Collaboration, 2010). The main "macrovascular" diseases (related to atherosclerosis of larger arteries) are coronary artery disease (angina and myocardial infarction), stroke, and peripheral vascular disease. About 75% of deaths in diabetics are due to coronary artery disease (O'Gara et. al., 2013). Diabetes also damages the capillaries (causes microangiopathy) (Boussageon et. al., 2011). Diabetic retinopathy, which affects blood vessel formation in the retina of the eye, can lead to visual symptoms including reduced vision and potentially blindness. Diabetic nephropathy, the impact of diabetes on the kidneys, can lead to scarring changes in the kidney tissue, loss of small or progressively larger amounts of protein in the urine, and eventually chronic kidney disease requiring dialysis.

Another risk is diabetic neuropathy, the impact of diabetes on the nervous system — most commonly causing numbness, tingling, and pain in the feet, and also increasing the risk of skin damage due to altered sensation. Together with vascular disease in the legs, neuropathy contributes to the risk of diabetes-related foot problems (such as diabetic foot ulcers) that can be difficult to treat and occasionally require amputation. Additionally, proximal diabetic neuropathy causes painful muscle wasting and weakness.

Several studies suggest (Cukierman, 2012) a link between cognitive deficit and diabetes. Compared to those without diabetes, the research showed that those with the disease have a 1.2 to 1.5-fold greater rate of decline in cognitive function, and are at greater risk.

Diabetes mellitus is classified into four broad categories: type 1, type 2, gestational diabetes, and other specific types (Shoback, 2011). The "other specific types" are a collection of a few dozen individual causes (Shoback, 2011). The term "diabetes", without qualification, usually refers to diabetes mellitus.

There is no known preventive measure for type 1 diabetes (WHO, 2013). Type 2 diabetes can often be prevented by a person being a normal body weight and physical exercise (WHO, 2013).

Diabetes mellitus is a chronic disease, for which there is no known cure except in very specific situations. Management concentrates on keeping blood sugar levels as close to normal ("euglycemia") as possible, without causing hypoglycemia. This can usually be accomplished with diet, exercise, and use of appropriate medications (insulin in the case of type 1 diabetes; oral medications, as well as possibly insulin, in type 2 diabetes).

Patient education, understanding, and participation is vital, since the complications of diabetes are far less common and less severe in people who have well-managed blood sugar levels (Nathan, 2005). The goal of treatment is an HbA1C level of 6.5%, but should not be lower than that, and may be set higher (National Institute for Health and Clinical Excellence, 2008). Attention is also paid to other health problems that may accelerate the deleterious effects of diabetes. These include smoking, elevated cholesterol levels, obesity, high blood pressure, and lack of regular exercise (National Institute for Health and Clinical Excellence, 2008). Specialised footwear is widely used to reduce the risk of ulceration, or re-ulceration, in at-risk diabetic feet. Evidence for the efficacy of this remains equivocal, however (Cavanagh, 2004).

3. Methodology

The methodology pertaining to the study - Impact of Dietary Counseling and Administration of Mangifera Indica (Mango) Leaf Powder as Herbal Treatment for Diabetes: A Study on Patients in Rourkela, Odisha, was sequenced under the heads, viz. selection of area, selection of sample population (patients), collection of data and conducting the study, diet counseling and questionnaire-cum-interview schedule, administration process of mango-leaf powder and finally the evaluation of blood glucose level, before and after, administration of diet counseling, medicines and mango-leaf powder.

Using convenience sampling method, patients with moderate to high blood glucose level were selected from the Ayurvedic clinic of Dr Devraj Sahoo of Rourkela, Odisha for the better convenience in respect to availability of data or accessibility of the units. The study was conducted on three groups of samples with moderate to high blood sugar levels. Each group consisted of 10 (ten) members/subjects. Hence, the total number of subject for the study chosen was 30.

Group A: Diet and Medication subjects were on diabetic diet after counseling and medication. Group B: Diet, Medication and Mango-Leaf Powder subjects were on diabetic diet after counseling, medication and were administered mango-leaf powder. Group C: Diet, Medication and Placebo (Mint-Leaf) subjects were on diabetic diet after counseling, medication and were given a placebo in the form of mint-leaf powder.

A structured questionnaire, as given in Appendix A, was formulated by the investigator and the subjects were allowed to fill them. The proper filling of the questionnaire was ensured by the researcher. The information (clinical status, medical history, anthropometric measurements, biochemical analysis, lifestyle pattern, dietary pattern and overall preferences) of the selected subjects were collected. The factors such as Age, Gender, Occupation, Economic Status and Family Medical History played an important role as well to ascertain the subject's vulnerability towards diabetes.

3.1. Administration of Mango-Leaf Powder

The sample of 30 patients were broken down in three groups, Group-A (who were on counseled diet and medication), Group-B (who were on counseled diet and medication, were administered mango-leaf powder), and Group-C (who were on counseled diet and medication, and were administered a placebo in the form of mint-leaf powder). The blood glucose level of all the subjects (30 in number) in three groups were tested and recorded with the help of Accu-chek blood sugar measuring instrument. Fasting and post-prandial sugar levels were recorded and assessed. The results of the three groups were compared to see the effectiveness of the mango-leaves in controlling diabetes.

3.2. Choice of Parameters in Correlation with Blood Sugar Reduction

The questionnaire is divided into broadly five parts, with 23 questions. The five parts consisted of General Information, Anthropometric Data, Biochemical Data, Lifestyle Pattern and Dietary Pattern. In the analysis, every other part of the response is correlated with Biochemical data to ascertain the findings of this research. In General Information category, Age Group, Family Medical History and Clinical Status is correlated as these are most pertinent to ascertain the independent factors that affect sugar levels. In Anthropometric Data, BMI covers the remaining data asked and hence is considered here. In Lifestyle Pattern, Physical Activity is the most important factor that can affect sugar levels and hence taken for analysis. In Dietary Pattern, Sweet Consumption and Veg/Non-Veg Diet is considered as these are expected to affect sugar levels most. On the whole, seven factors are cross-tabulated with the Biochemical Data that shows change in blood sugar level, representing the management of diabetes.

4. Results and Findings

The results and findings of the present study on the Effect of Mango leaves on treating diabetes is consolidated and discussed under the following heads:

Data Comparison of FBS and PP - Before and After - Groupwise

Subject	FBS (mg/dl) (Before Diet Counseling & with Medication)	FBS (mg/dl) (After Diet Counseling & with Medication)	PP (mg/dl) (Before Diet Counseling & with Medication)	PP (mg/dl) (After Diet Counseling & with Medication)
A1	95	88	135	128
A2	86	80	126	122
A3	115	105	145	130
A4	77	75	122	120
A5	92	88	130	124
A6	84	82	125	122
A7	79	77	122	120
A8	80	78	120	119
A9	88	87	128	124
A10	90	87	132	125
B1	89	75	129	120
B2	98	85	127	121
B3	117	100	139	129
B4	93	80	137	125
B5	79	68	123	117
B6	80	71	126	119
B7	88	77	128	120
B8	106	95	132	123
B9	101	87	135	115
B10	89	80	118	113
C1	88	82	129	122
C2	85	81	127	119
C3	101	96	141	135
C4	97	90	129	124
C5	82	77	124	118
C6	113	112	143	144
C7	111	104	135	130
C8	93	88	123	120
C9	75	79	120	122
C10	80	75	126	118

Table 1: Data Comparison of FBS and PP – before and after - Groupwise

5. Analysis and Discussion

In Age Group a (Age<30 yrs), subject A3 showed excellent reduction in both sugar levels. In Age Group b (Age=30-49), 3 subjects A1, A2, and A5 showed good reduction in sugar levels, whereas 2 subjects A7 and A8 had below average fasting sugar reduction and

poor post prandial sugar reduction. In Age Group c (50-69), 2 subjects A6 and A10 showed good reduction in both sugar levels, whereas subject A4 showed poor post prandial sugar reduction. Age Group d (>70), subject A9 had poor fasting sugar level reduction. Overall, lower age group showed better results.

In Age Group a (Age<30 yrs), subject B9 showed excellent reduction in both sugar levels. In Age Group b (Age=30-49), 5 subjects B1, B2, B4, B6 and B7 showed excellent reduction in both sugar levels. In Age Group c (50-69), 2 subjects B5 and B8 showed excellent reduction in both sugar levels. Age Group d (>70), subject B10 also showed comparatively excellent reduction in both sugar levels. Overall, the whole group showed excellent sugar reduction results.

In Age Group a (Age<30 yrs), subject C2 showed above average in both sugar levels. In Age Group b (Age=30-49), 2 subjects C1 and C3 showed above average reduction in both sugar levels. In Age Group c (50-69), 3 subjects C5, C7 and C10 showed average reduction in both sugar levels; subject C8 showed below average post prandial sugar levels. In Age Group d (>70), subject C4 had very good fasting sugar level reduction; subject C6 had marginal reduction in fasting and negative reduction in post prandial; whereas C9 had negative reduction in both sugar levels. Overall, lower age group showed better results, except two cases in the higher age group.

In family medical history, Group a (Father diabetic), subjects A1 showed good reduction in both blood sugar levels. In Group b (Mother diabetic), subjects A3 showed good reduction in both sugar level, whereas A6 and A8 showed below average reduction in both sugar levels. In Group c (both Parents diabetc), A2 showed good reduction in FBS and average reduction in PP, whereas A4 showed below average reduction in both sugar level. In Group d (other family member diabetic), A5 showed average reduction in both sugar level, whereas A9 showed below average reduction in FBS. In Group e (no diabetes in family), A7 showed below average reduction in both sugar levels, whereas A10 showed below average reduction in both blood sugar level. Overall, the group showed diverging results.

In family medical history, Group a (Father diabetic), subjects B1, B5 and B6 showed good reduction in FBS levels, but showed below average reduction in PP sugar level. In Group b (Mother diabetic), subjects B3 and B7 showed excellent reduction in both sugar levels, whereas B10 showed below average reduction in PP levels. In Group c (both Parents diabetc), B2 showed excellent reduction in FBS and below average reduction in PP, whereas B8 showed average reduction in both sugar level. In Group d (other family member diabetic), B9 showed excellent reduction in both sugar level. In Group e (no diabetes in family), B4 showed excellent reduction in both sugar level. Overall, the group showed excellent FBS reduction and average PP reduction results.

In family medical history, Group a (Father diabetic), subjects C1 showed average reduction in both blood sugarl evels. In Group b (Mother diabetic), subject C2 showed avarege reduction in both FBSand PP results, whereas C6 showed marginal FBS reduction and negative PP reduction, whereas C9 showed negative reduction results in both sugar level. In Group c (both Parents diabetc), C3 showed average reduction in both sugar level, whereas C4 showed good FBS reduction and average PP reduction. In Group d (other family member diabetic), C5, C7 and C8 showed average reduction in both sugar level, except C8 that showed below average PP reduction. In Group e (no diabetes in family), C10 showed good reduction in both sugar level. Overall, the group showed average reduction in both sugar level.

In clinical status, Group a (controlled diabetes) has no subjects. Group b (uncontrolled diabetes) subjects A1 and A3 showed good reduction in both blood sugar levels; subjects A7 and A9 showed below average reduction in both sugar levels, whereas A10 showed below average FBS reduction and above average reduction in PP. In Group c (fluctuating sugar level), subjects A2 and A5 showed average reduction in both sugar level, whereas A4, A6 and A8 showed below average reduction in both sugar levels. Overall, the group showed average results.

In clinical status, Group b (uncontrolled diabetes) subjects B2, B3, B4, B5, B6, B7 and B8 showed excellent FBS and PP reduction, with the exception of B2, B5 and B6 who showed below average PP reduction results. In Group c (fluctuating sugar level), subjects B1 and B10 showed excellent reduction in FBS and average reduction in PP, whereas B9 showed excellent reduction in both sugar level. Overall, the group showed excellent results in FBS reduction and average reduction in PP sugar level.

In clinical status, Group b (uncontrolled diabetes) subjects C1, C4, C5 and C10 showed average reduction in both sugar levels; C8 showed below average PP reduction, whereas C9 showed negative reduction in both sugar level. In Group c (fluctuating sugar level), subjects C2, C3 and C7 showed average reduction in both FBS/PP, whereas C6 showed marginal FBS reduction and negative PP reduction. Overall, the group showed average results in FBS reduction and average reduction in PP sugar level, similar to Group A.

In Grade 1 obesity (BMI=25-29), subject A5 showed average reduction in sugar levels. In Grade 2 obesity (BMI=30-40), 3 subjects A1, A2 and A3 showed good reduction in sugar levels, 2 subjects A8 and A4 had poor post prandial sugar reduction and subject A9 had poor both fasting and post prandial sugar level reduction; while the remaining showed average reduction in sugar levels. Overall, the group showed average reduction in both sugar levels.

In Grade 1 obesity (BMI=25-29), subject B2 showed above average reduction in both sugar levels. In Grade 2 obesity (BMI=30-40), 8 subjects B1, B3, B4, B6, B7, B8, B9 and B10 showed excellent reduction in both sugar levels. Grade 3 obesity (BMI>40) subject B5 has shown excellent reduction in fasting blood sugar levels and average reduction in post prandial sugar levels. Overall, the group showed excellent reduction in both sugar levels.

In Grade 1 obesity (BMI=25-29), subject C1 showed above average reduction in sugar levels. In Grade 2 obesity (BMI=30-40), 6 subjects C2, C3, C4, C5, C7 and C8 showed above average reduction in sugar levels; subject C6 showed minimum reduction in fasting sugar levels and negative reduction in post prandial sugar levels; subject C9 showed negative reduction in both sugar levels. Subject C10 has BMI closer to Grade 3 obesity, showed above average reduction in both sugar levels. Overall, the group showed average reduction in both sugar levels, except two cases.

In Physical Activity Group b (Seldom exercise), subject A5 showed average reduction in both sugar levels. In Group c (once a week exercise), subject A2 showed good reduction in fasting sugar levels, whereas subject A7 and A8 had below than average post prandial sugar reduction. In Group d (2-3 times a week exercise), subjects A4 and A6 showed below average reduction in both sugar levels, whereas subject A9 and A10 showed poor fasting sugar reduction; subject A1 showed good result in reduction of both sugar levels. In Group e (exercise everyday), subject A3 showed very good result in reduction of both sugar levels. Overall, group with more physical exercise showed better results.

In Group a (never exercise) showed good reduction in fasting sugar levels; B2 and B7 below showed below average post prandial sugar reduction, whereas B4 showed excellent post prandial reduction. In Group d (2-3 times a week exercise), subjects B1, B6, B8 and B10 showed good reduction in both sugar levels, except subject B10 showed below average post prandial sugar reduction. In Group e (exercise everyday), subject B3 sand B9 showed excellent result in reduction of both sugar levels. Overall, group with more physical exercise showed excellent results.

In Physical Activity Group b (Seldom exercise), subjects C1 showed above average reduction in both sugar levels, whereas subject C9 had negative reduction in both sugar levels. In Group c (once a week exercise), subject C2, C3, and C4 showed average reduction in fasting sugar levels, whereas subject C6 had marginal reduction in fasting sugar and negative reduction in post prandial sugar. In Group d (2-3 times a week exercise), subjects C7, C8 and C10 showed reasonable reduction in both sugar levels. In Group e (exercise everyday), subject C5 showed very above average result in reduction of both sugar levels. Overall, group with more physical exercise showed better results.

In Sweet Consumption Group a (never consume sweets) subject A5 above average reduction in both sugar levels, whereas A10 showed below average fasting and above average post prandial sugar levels. In Group b (seldom take sweets), subjects A1, A2, and A3 showed good reduction in both sugar levels; subject A4, A6, A7, A8 and A9 showed below average reduction in both sugar levels. Overall, group that refrained from sweets showed better results.

In Sweet Consumption Group a (never consume sweets) subjects B2, B4 and B8 showed excellent reduction in both sugar levels. In Group b (seldom take sweets), subjects B1, B3, B6, B7 and B10 showed very good reduction in both sugar levels; whereas subject B9 showed excellent result in reduction of both sugar levels. In Group c (consume sweets frequently) subject B5 showed good reduction in fasting sugar levels, but showed below average reduction in post prandial sugar levels. Overall, Group a and B showed excellent results.

In Sweet Consumption Group a (never consume sweets) subjects C1, C3 and C5 showed good reduction in both sugar levels. In Group b (seldom take sweets), subjects C2, C4, C7 and C8 showed average reduction in both sugar levels; whereas subject C6 showed marginal reduction in fasting and negative reduction in post prandial sugar levels; subject C9 showed negative reduction in both sugar levels. In Group c (consume sweets frequently) subject C10 showed above average reduction in both sugar levels. Overall, this group showed mixed results.

In Food Consumption Group a (vegetarian) subjects A5 and A10 showed above average reduction in both sugar levels. In Group b (non-vegetarian), subjects A1 showed good reduction in both sugar levels. In Group c (consume both vegetarian/non-vegetarian food) subjects A2 and A3showed average reduction in both sugar levels, whereas A4, A6, A7, A8 and A9 showed below average reduction in one or the other sugar level. Overall, group that consumed vegetarian food showed better results and the group that consumed both showed mixed results.

In Food Consumption Group a (vegetarian) subjects B2 and B8 showed excellent reduction in both sugar levels, except B2 that showed below average reduction in post prandial sugar levels. In Group b (non-vegetarian), subjects B1 showed excellent reduction in both sugar levels, whereas subject B6 posted below average reduction in both sugar levels. In Group c (consume both vegetarian/non-vegetarian food) subjects B3, B4, B5, B7, B9 and B10 showed excellent reduction in both sugar levels, except B5 and B10 showed below average reduction in post prandial sugar level. Overall, the group that consumed both showed better results.

In Food Consumption In Group b (non-vegetarian), subjects C7 showed above average reduction in both sugar levels and C9 showed negative reduction in both sugar levels. In Group c (consume both vegetarian/non-vegetarian food) subjects C1, C2, C3, C4, C5, C8 and C10 showed average reduction in both sugar levels, whereas C6 showed marginal reduction in fasting and negative reduction in post prandial sugar levels. Overall, group that consumed vegetarian/non-vegetarian/both food showed conflicting results.

The classic symptoms of untreated diabetes are weight loss, polyuria (frequent urination), polydipsia (increased thirst), and polyphagia (increased hunger) Cooke & Plotnick, 2008). Symptoms may develop rapidly (weeks or months) in type 1 diabetes, while they usually develop much more slowly and may be subtle or absent in type 2 diabetes.

Prolonged high blood glucose can cause glucose absorption in the lens of the eye, which leads to changes in its shape, resulting in vision changes. Blurred vision is a common complaint leading to a diabetes diagnosis. A number of skin rashes that can occur in diabetes are collectively known as diabetic dermadromes.

People (usually with type 1 diabetes) may also present with diabetic ketoacidosis, a state of metabolic problems characterized by the smell of acetone, a rapid, deep breathing known as Kussmaul breathing, nausea, vomiting and abdominal pain, and altered states of consciousness.

A rare but equally severe possibility is hyperosmolar nonketotic state, which is more common in type 2 diabetes and is mainly the result of dehydration.

All forms of diabetes increase the risk of long-term complications. These typically develop after many years (10–20), but may be the first symptom in those who have otherwise not received a diagnosis before that time. The major long-term complications relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease (Emerging Risk Factors Collaboration, 2010). The main

"macrovascular" diseases (related to atherosclerosis of larger arteries) are coronary artery disease (angina and myocardial infarction), stroke, and peripheral vascular disease. About 75% of deaths in diabetics are due to coronary artery disease (O'Gara et. al., 2013).

Diabetes also damages the capillaries (causes microangiopathy) (Boussageon et. al., 2011). Diabetic retinopathy, which affects blood vessel formation in the retina of the eye, can lead to visual symptoms including reduced vision and potentially blindness. Diabetic nephropathy, the impact of diabetes on the kidneys, can lead to scarring changes in the kidney tissue, loss of small or progressively larger amounts of protein in the urine, and eventually chronic kidney disease requiring dialysis.

Another risk is diabetic neuropathy, the impact of diabetes on the nervous system — most commonly causing numbness, tingling, and pain in the feet, and also increasing the risk of skin damage due to altered sensation. Together with vascular disease in the legs, neuropathy contributes to the risk of diabetes-related foot problems (such as diabetic foot ulcers) that can be difficult to treat and occasionally require amputation. Additionally, proximal diabetic neuropathy causes painful muscle wasting and weakness.

Several studies suggest (Cukierman, 2012) a link between cognitive deficit and diabetes. Compared to those without diabetes, the research showed that those with the disease have a 1.2 to 1.5-fold greater rate of decline in cognitive function, and are at greater risk.

In the United States, there were approximately 675,000 diabetes-related emergency department (ED) visits in 2010 that involved neurological complications, 409,000 ED visits with kidney complications, and 186,000 ED visits with eye complications Washington et. al., 2013).

Diabetes mellitus is classified into four broad categories: type 1, type 2, gestational diabetes, and "other specific types" (Shoback, 2011). The "other specific types" are a collection of a few dozen individual causes (Shoback, 2011). The term "diabetes", without qualification, usually refers to diabetes mellitus.

Type 2 diabetes mellitus is characterized by insulin resistance, which may be combined with relatively reduced insulin secretion (Shoback, 2011). The defective responsiveness of body tissues to insulin is believed to involve the insulin receptor. However, the specific defects are not known. Diabetes mellitus cases due to a known defect are classified separately. Type 2 diabetes is the most common type.

In the early stage of type 2, the predominant abnormality is reduced insulin sensitivity. At this stage, hyperglycemia can be reversed by a variety of measures and medications that improve insulin sensitivity or reduce glucose production by the liver.

Type 2 diabetes is due primarily to lifestyle factors and genetics (Riserus, 2009). A number of lifestyle factors are known to be important to the development of type 2 diabetes, including obesity (defined by a body mass index of greater than thirty), lack of physical activity, poor diet, stress, and urbanization.[5] Excess body fat is associated with 30% of cases in those of Chinese and Japanese descent, 60-80% of cases in those of European and African descent, and 100% of Pima Indians and Pacific Islanders (Shoback, 2011). Those who are not obese often have a high waist–hip ratio (Shoback, 2011).

Dietary factors also influence the risk of developing type 2 diabetes. Consumption of sugar-sweetened drinks in excess is associated with an increased risk (Malik et. al., 2010). The type of fats in the diet is also important, with saturated fats and trans fatty acids increasing the risk and polyunsaturated and monounsaturated fat decreasing the risk (Riserus, 2009). Eating lots of white rice appears to also play a role in increasing risk (Hu et. al., 2012). A lack of exercise is believed to cause 7% of cases Lee et. al., 2012).

Higher insulin levels increase some anabolic ("building up") processes, such as cell growth and duplication, protein synthesis, and fat storage. Insulin (or its lack) is the principal signal in converting many of the bidirectional processes of metabolism from a catabolic to an anabolic direction, and *vice versa*. In particular, a low insulin level is the trigger for entering or leaving ketosis (the fat-burning metabolic phase).

If the amount of insulin available is insufficient, if cells respond poorly to the effects of insulin (insulin insensitivity or resistance), or if the insulin itself is defective, then glucose will not have its usual effect, so it will not be absorbed properly by those body cells that require it, nor will it be stored appropriately in the liver and muscles. The net effect is persistent high levels of blood glucose, poor protein synthesis, and other metabolic derangements, such as acidosis.

When the glucose concentration in the blood is raised to about 9-10 mmol/L (except certain conditions, such as pregnancy), beyond its renal threshold (i.e. when glucose level surpasses the transport maximum of glucose reabsorption), reabsorption of glucose in the proximal renal tubuli is incomplete, and part of the glucose remains in the urine (glycosuria). This increases the osmotic pressure of the urine and inhibits reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. Lost blood volume will be replaced osmotically from water held in body cells and other body compartments, causing dehydration and increased thirst.

As per the World Health Organization people with fasting glucose levels from 6.1 to 6.9 mmol/l (110 to 125 mg/dl) are considered to have impaired fasting glucose (WHO, 2006) people with plasma glucose at or above 7.8 mmol/L (140 mg/dL), but not over 11.1 mmol/L (200 mg/dL), two hours after a 75 g oral glucose load are considered to have impaired glucose tolerance. Of these two prediabetic states, the latter in particular is a major risk factor for progression to full-blown diabetes mellitus, as well as cardiovascular disease (Santaguida et. al., 2008). The American Diabetes Association since 2003 uses a slightly different range for impaired fasting glucose of 5.6 to 6.9 mmol/l (100 to 125 mg/dl) (Batolli et. al., 2011).

Glycated hemoglobin is better than fasting glucose for determining risks of cardiovascular disease and death from any cause (Selvin et. al., 2010).

The rare disease diabetes insipidus has similar symptoms to diabetes mellitus, but without disturbances in the sugar metabolism (*insipidus* means "without taste" in Latin) and does not involve the same disease mechanisms.

There is no known preventive measure for type 1 diabetes (WHO, 2013). Type 2 diabetes can often be prevented by a person being a normal body weight and physical exercise (WHO, 2013).

Diabetes mellitus is a chronic disease, for which there is no known cure except in very specific situations. Management concentrates on keeping blood sugar levels as close to normal ("euglycemia") as possible, without causing hypoglycemia. This can usually be accomplished with diet, exercise, and use of appropriate medications (insulin in the case of type 1 diabetes; oral medications, as well as possibly insulin, in type 2 diabetes).

Patient education, understanding, and participation is vital, since the complications of diabetes are far less common and less severe in people who have well-managed blood sugar levels (Nathan, 2005). The goal of treatment is an HbA1C level of 6.5%, but should not be lower than that, and may be set higher (National Institute for Health and Clinical Excellence, 2008). Attention is also paid to other health problems that may accelerate the deleterious effects of diabetes. These include smoking, elevated cholesterol levels, obesity, high blood pressure, and lack of regular exercise (National Institute for Health and Clinical Excellence, 2008). Specialised footwear is widely used to reduce the risk of ulceration, or re-ulceration, in at-risk diabetic feet. Evidence for the efficacy of this remains equivocal, however (Cavanagh, 2004).

There are roles for patient education, dietetic support, and sensible exercise, with the goal of keeping both short-term and long-term blood glucose levels within acceptable bounds. In addition, given the associated higher risks of cardiovascular disease, lifestyle modifications are recommended to control blood pressure (Alder et. al., 2000).

Metformin is generally recommended as a first line treatment for type 2 diabetes, as there is good evidence that it decreases mortality (Ripsin et. al., 2009). Routine use of aspirin, however, has not been found to improve outcomes in uncomplicated diabetes (Pignone et. al., 2010).

Type 1 diabetes is typically treated with combinations of regular and NPH insulin, or synthetic insulin analogs. When insulin is used in type 2 diabetes, a long-acting formulation is usually added initially, while continuing oral medications (Ripsin et. al., 2009). Doses of insulin are then increased to effect (Ripsin et. al., 2009).

In countries using a general practitioner system, such as the United Kingdom, care may take place mainly outside hospitals, with hospital-based specialist care used only in case of complications, difficult blood sugar control, or research projects. In other circumstances, general practitioners and specialists share care of a patient in a team approach. Home telehealth support can be an effective management technique (Polisena et. al, 2009).

So the present study clearly states that mango-leaf solution has some effect in reducing the blood sugar level, with no expected side effects. One to two teaspoon of mango-leaf can be dissolved (leached) in 150 ml of water and taken twice daily on empty stomach. There is no certain study that specifies the dosage of mango-leaf.

It is understood from this research that it has strong potential for further research with a larger sample that is representative of a larger population. This study clearly shows that mango-leaf solution, when taken with the regular diabetic diet and medication, reduces the sugar levels of the subjects, making diabetes control easy.

This study (research) and its outcome are not meant for insulin dependent subjects. For others, it is essential that they keep the Doctor informed about their consuming mango-leaf solution and should monitor their sugar levels frequently.

6. Conclusion

Group A proved that there was a reduction of blood sugar level (FBS and PP correspondingly) of the patients after about a month, during which time they followed a strict diet and medication. The average FBS reduction percentage was 4.19 and the average PP reduction percentage was 3.83, which shows the effect of counseling in strictly following diabetic diet and medication.

Group B proves that there was remarkable reduction of blood sugar level (FBS and PP correspondingly) of the patients after about a month, during which time they followed a strict diet and medication and mango-leaf solution. The average FBS reduction percentage was 12.95 and the average PP reduction percentage was 7.02, which is considerably higher than Group A and C.

Group C proves that there was a slight reduction of blood sugar level (FBS and PP correspondingly) of the patients after about a month, during which time they followed a strict diet, medication and placebo (mint-leaf). Two patients responded negatively to the placebo, which on the average the whole group benefited from the placebo, perhaps a more psychological factor that helped in minor reduction of sugar levels. The average FBS reduction percentage was 4.33 and the average PP reduction percentage was 3.48, which is approximately similar to Group A.

The study confirms that the subjects who were under diet counseling, medication and mango-leaf powder showed considerable reduction in blood sugar level. From this research, it is concluded that although diet and medication are effective in reducing blood sugar levels, but intake of mango-leaf solution in appropriate quantity has an additional beneficial effect in reducing blood sugar level that is long-lasting.

7. Recommendations

From the research on the effect of mango-leaves on treating diabetes and basing upon the above said conclusion, the following recommendations are given:

- More studies are needed on larger samples for longer period to understand the details about diabetes in relation with mango-leaf solution.
- Steps should be taken to impart knowledge on nutrition to the community to consume a strict diabetic diet as a preventive measure to control blood sugar level.

- To educate diabetic patients to consume mango-leaf solution (1-2 teaspoon in 150ml of water) twice daily on empty stomach reduces the blood sugar level, and thus should be used by nutritional fraternity and other medical professional to use mango-leaf solution as functional food.
- In long term management of diabetes, mango-leaf solution should be included as daily diet in community hospitals, nursing homes and wellness centers.
- The study is not meant for insulin dependent subjects (mostly Type 1 diabetes patients).
- It is recommended for all the subjects to undergo regular screening and monitoring healthy level of blood glucose, HbA1c, kidney health indicators, BP, blood lipids, indicator of heart disease and stroke prevention.
- Strength training and brisk walking is recommended for all diabetic subjects.
- Include low glycemic food, which includes fruits and vegetables (except potatoes and other root vegetables), grainy breads, legumes, pulses, milk and curd.
- Include fiber rich foods, such as apple, figs, guava, lemon and orange, as well as grains rich in fiber, such as barley, oatmeal, bajra and wholewheat.
- Choose a healthy lifestyle, choose food with less fat and salt; avoid alcohol and smoking. Eat raw mangoes (especially its leaves) for a healthier lifestyle.

8. References

1. "Diabetes Blue Circle Symbol". International Diabetes Federation. 17 March 2006.
2. "Diabetes". World Health Organization. Retrieved 4 April 2014.
3. Shoback, edited by David G. Gardner, Dolores (2011). Greenspan's basic & clinical endocrinology (9th ed.). New York: McGraw-Hill Medical. pp. Chapter 17. ISBN 0-07-162243-8.
4. "Diabetes Fact sheet N°312". WHO. October 2013. Retrieved 25 March 2014.
5. Williams textbook of endocrinology (12th ed.). Philadelphia: Elsevier/Saunders. pp. 1371–1435. ISBN 978-1-4377-0324-5.
6. Jump up to: [a] [b] "International Diabetes Foundation: Diabetes Atlas". Retrieved 4 April 2014.
7. Vos, T; Flaxman, AD et al (2012 Dec 15). "Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010.". Lancet 380 (9859): 2163–96. PMID 23245607.
8. Cooke DW, Plotnick L (November 2008). "Type 1 diabetes mellitus in pediatrics". Pediatr Rev 29 (11): 374–84; quiz 385. doi:10.1542/pir.29-11-374. PMID 18977856.
9. Emerging Risk Factors Collaboration (2010). "Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: A collaborative meta-analysis of 102 prospective studies". The Lancet 375 (9733): 2215–22. doi:10.1016/S0140-6736(10)60484-9. PMC 2904878. PMID 20609967.
10. O'Gara, PT; Kushner, FG; Ascheim, DD; Casey DE, Jr; Chung, MK; de Lemos, JA; Ettinger, SM; Fang, JC; Fesmire, FM; Franklin, BA; Granger, CB; Krumholz, HM; Linderbaum, JA; Morrow, DA; Newby, LK; Ornato, JP; Ou, N; Radford, MJ; Tamis-Holland, JE; Tommaso, CL; Tracy, CM; Woo, YJ; Zhao, DX; Anderson, JL; Jacobs, AK; Halperin, JL; Albert, NM; Brindis, RG; Creager, MA; DeMets, D; Guyton, RA; Hochman, JS; Kovacs, RJ; Kushner, FG; Ohman, EM; Stevenson, WG; Yancy, CW; American College of Cardiology Foundation/American Heart Association Task Force on Practice, Guidelines (2013 Jan 29). "2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines.". Circulation 127 (4): e362–425. doi:10.1161/CIR.0b013e3182742cf6. PMID 23247304.
11. Boussageon R, Bejan-Angoulvant T, Saadatian-Elahi M, Lafont S, Bergeonneau C, Kassaï B, Erpeldinger S, Wright JM, Gueyffier F, Cornu C (2011). "Effect of intensive glucose lowering treatment on all cause mortality, cardiovascular death, and microvascular events in type 2 diabetes: meta-analysis of randomised controlled trials". BMJ 343: d4169. doi:10.1136/bmj.d4169. PMC 3144314. PMID 21791495.
12. Cukierman, T (8 Nov 2005). "Cognitive decline and dementia in diabetes—systematic overview of prospective observational studies". Springer-Verlag. Retrieved 28 Apr 2013.
13. Jump up to: [a] [b] Washington R.E., Andrews R.M., Mutter R.L. Emergency Department Visits for Adults with Diabetes, 2010. HCUP Statistical Brief #167. November 2013. Agency for Healthcare Research and Quality, Rockville, MD. [1].
14. Lambert, P.; Bingley, P. J. (2002). "What is Type 1 Diabetes?". Medicine 30: 1–5. doi:10.1383/medc.30.1.1.28264. Diabetes Symptoms edit
15. Rother KI (April 2007). "Diabetes treatment—bridging the divide". The New England Journal of Medicine 356 (15): 1499–501. doi:10.1056/NEJMp078030. PMID 17429082.
16. "Diabetes Mellitus (DM): Diabetes Mellitus and Disorders of Carbohydrate Metabolism: Merck Manual Professional". Merck Publishing. April 2010. Retrieved 2010-07-30.
17. Dorner M, Pinget M, Brogard JM (May 1977). "Essential labile diabetes". MMW Munch Med Wochenschr (in German) 119 (19): 671–4. PMID 406527.
18. Risérus U, Willet W (January 2009). "Dietary fats and prevention of type 2 diabetes". Progress in Lipid Research 48 (1): 44–51. doi:10.1016/j.plipres.2008.10.002. PMC 2654180. PMID 19032965.

19. Malik, VS; Popkin, BM, Bray, GA, Després, JP, Hu, FB (2010-03-23). "Sugar Sweetened Beverages, Obesity, Type 2 Diabetes and Cardiovascular Disease risk". Circulation 121 (11): 1356–64. doi:10.1161/CIRCULATIONAHA.109.876185. PMC 2862465. PMID 20308626.
20. Malik, VS; Popkin, BM, Bray, GA, Després, JP, Willett, WC, Hu, FB (November 2010). "Sugar-Sweetened Beverages and Risk of Metabolic Syndrome and Type 2 Diabetes: A meta-analysis". Diabetes Care 33 (11): 2477–83. doi:10.2337/dc10-1079. PMC 2963518. PMID 20693348.
21. Hu, EA; Pan, A; Malik, V; Sun, Q (2012-03-15). "White rice consumption and risk of type 2 diabetes: meta-analysis and systematic review". BMJ (Clinical research ed.) 344: e1454. doi:10.1136/bmj.e1454. PMC 3307808. PMID 22422870.
22. Lee, I-Min; Shiroma, Eric J; Lobelo, Felipe; Puska, Pekka; Blair, Steven N; Katzmarzyk, Peter T (1 July 2012). "Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy". The Lancet 380 (9838): 219–29. doi:10.1016/S0140-6736(12)61031-9. PMC 3645500. PMID 22818936.
23. Jump up to: [a] [b] "Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications" (PDF). World Health Organisation. 1999.
24. Unless otherwise specified, reference is: Table 20-5 in Mitchell, Richard Sheppard; Kumar, Vinay; Abbas, Abul K.; Fausto, Nelson. Robbins Basic Pathology. Philadelphia: Saunders. ISBN 1-4160-2973-7. 8th edition.
25. Sattar N, Preiss, D, Murray, HM, Welsh, P, Buckley, BM, de Craen, AJ, Seshasai, SR, McMurray, JJ, Freeman, DJ (February 2010). "Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials". The Lancet 375 (9716): 735–42. doi:10.1016/S0140-6736(09)61965-6. PMID 20167359.
26. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: report of a WHO/IDF consultation. Geneva: World Health Organization. 2006. p. 21. ISBN 978-92-4-159493-6.
27. Vijan, S (March 2010). "Type 2 diabetes". Annals of Internal Medicine 152 (5): ITC31-15. doi:10.1059/0003-4819-152-5-201003020-01003. PMID 20194231.
28. ""Diabetes Care" January 2010". American Diabetes Association. Retrieved 2010-01-29.
29. Saydah SH, Miret M, Sung J, Varas C, Gause D, Brancati FL (August 2001). "Postchallenge hyperglycemia and mortality in a national sample of U.S. adults". Diabetes Care 24 (8): 1397–402. doi:10.2337/diacare.24.8.1397. PMID 11473076.
30. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia : report of a WHO/IDF consultation. World Health Organization. 2006. p. 21. ISBN 978-92-4-159493-6.
31. Santaguida PL, Balion C, Hunt D, Morrison K, Gerstein H, Raina P, Booker L, Yazdi H. "Diagnosis, Prognosis, and Treatment of Impaired Glucose Tolerance and Impaired Fasting Glucose". Summary of Evidence Report/Technology Assessment, No. 128. Agency for Healthcare Research and Quality. Retrieved 2008-07-20.
32. Bartoli, E; Fra, GP; Carnevale Schianca, GP (2011 Feb). "The oral glucose tolerance test (OGTT) revisited.". European journal of internal medicine 22 (1): 8–12. doi:10.1016/j.ejim.2010.07.008. PMID 21238885.
33. Selvin E, Steffes MW, Zhu H, Matsushita K, Wagenknecht L, Pankow J, Coresh J, Brancati FL (2010). "Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults". N. Engl. J. Med. 362 (9): 800–11. doi:10.1056/NEJMoa0908359. PMC 2872990. PMID 20200384.
34. Nathan DM, Cleary PA, Backlund JY, Genuth SM, Lachin JM, Orchard TJ, Raskin P, Zinman B; Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications (DCCT/EDIC) Study Research Group (December 2005). "Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes". The New England Journal of Medicine 353 (25): 2643–53. doi:10.1056/NEJMoa052187. PMC 2637991. PMID 16371630.
35. The Diabetes Control and Complications Trial Research Group (April 1995). "The effect of intensive diabetes therapy on the development and progression of neuropathy.". Annals of Internal Medicine 122 (8): 561–8. doi:10.1059/0003-4819-122-8-199504150-00001. PMID 7887548.
36. National Institute for Health and Clinical Excellence. Clinical guideline 66: Type 2 diabetes. London, 2008.
37. Cavanagh, P. R. (2004). "Therapeutic footwear for people with diabetes". Diabetes/Metabolism Research and Reviews 20: S51–S55. doi:10.1002/dmrr.435. edit
38. Adler AI, Stratton IM, Neil HA, Yudkin JS, Matthews DR, Cull CA, Wright AD, Turner RC, Holman RR (August 2000). "Association of systolic blood pressure with macrovascular and microvascular complications of type 2 diabetes (UKPDS 36): prospective observational study". BMJ 321 (7258): 412–9. doi:10.1136/bmj.321.7258.412. PMC 27455. PMID 10938049.
39. Ripsin CM, Kang, H, Urban, RJ (2009). "Management of blood glucose in type 2 diabetes mellitus". American family physician 79 (1): 29–36. PMID 19145963.
40. Pignone M, Alberts MJ, Colwell JA, Cushman M, Inzucchi SE, Mukherjee D, Rosenson RS, Williams CD, Wilson PW, Kirkman MS; American Diabetes Association; American Heart Association; American College of Cardiology Foundation (June 2010). "Aspirin for primary prevention of cardiovascular events in people with diabetes: a position statement of the American Diabetes Association, a scientific statement of the American Heart Association, and an expert consensus document of the American College of Cardiology Foundation". Diabetes Care 33 (6): 1395–402. doi:10.2337/dc10-0555. PMC 2875463. PMID 20508233.
41. Polisena J, Tran K, Cimon K, Hutton B, McGill S, Palmer K (2009). "Home telehealth for diabetes management: a systematic review and meta-analysis". Diabetes Obes Metab 11 (10): 913–30. doi:10.1111/j.1463-1326.2009.01057.x. PMID 19531058.

42. Wild S, Roglic G, Green A, Sicree R, King H (2004). "Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030". Diabetes Care 27 (5): 1047–53. doi:10.2337/diacare.27.5.1047. PMID 15111519.
43. Ripoll, Brian C. Leutholtz, Ignacio (2011-04-25). Exercise and disease management (2nd ed.). Boca Raton: CRC Press. p. 25. ISBN 978-1-4398-2759-8.
44. Editor, Leonid Poretsky, (2009). Principles of diabetes mellitus (2nd ed.). New York: Springer. p. 3. ISBN 978-0-387-09840-1.
45. Konstantinos Laios et al.; Karamanou, M; Saridaki, Z; Androutsos, G (2012). "Aretaeus of Cappadocia and the first description of diabetes". Hormones 11 (1): 109–113. PMID 22450352.
46. Oxford English Dictionary. diabetes. Retrieved 2011-06-10.
47. Harper, Douglas (2001–2010). "Online Etymology Dictionary. diabetes.". Retrieved 2011-06-10.
48. Jump up to: [a] [b] Dallas, John (2011). "Royal College of Physicians of Edinburgh. Diabetes, Doctors and Dogs: An exhibition on Diabetes and Endocrinology by the College Library for the 43rd St. Andrew's Day Festival Symposium".
49. Oxford English Dictionary. mellite. Retrieved 2011-06-10.
50. "MyEtimology. mellitus.". Retrieved 2011-06-10.
51. Oxford English Dictionary. -ite. Retrieved 2011-06-10.
52. Theodore H. Tulchinsky, Elena A. Varavikova (2008). The New Public Health, Second Edition. New York: Academic Press. p. 200. ISBN 0-12-370890-7.
53. Piwernetz K, Home PD, Snorgaard O, Antsiferov M, Staehr-Johansen K, Krans M (May 1993). "Monitoring the targets of the St Vincent Declaration and the implementation of quality management in diabetes care: the DIABCARE initiative. The DIABCARE Monitoring Group of the St Vincent Declaration Steering Committee". Diabetic Medicine 10 (4): 371–7. doi:10.1111/j.1464-5491.1993.tb00083.x. PMID 8508624.
54. Dubois, HFW and Bankauskaite, V (2005). "Type 2 diabetes programmes in Europe" (PDF). Euro Observer 7 (2): 5–6.
55. Stewart WF, Ricci JA, Chee E, Hirsch AG, Brandenburg NA (June 2007). "Lost productive time and costs due to diabetes and diabetic neuropathic pain in the US workforce". J. Occup. Environ. Med. 49 (6): 672–9. doi:10.1097/JOM.0b013e318065b83a. PMID 17563611.
56. "Diabetes mellitus". Merck Veterinary Manual, 9th edition (online version). 2005. Retrieved 2011-10-23.
57. Danaei G, Finucane MM, Lu Y, Singh GM, Cowan MJ, Paciorek CJ et al. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2.7 million participants. Lancet, 2011, 378(9785):31–40.
58. Global health risks. Mortality and burden of disease attributable to selected major risks. Geneva, World Health Organization, 2009.
59. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Med, 2006, 3(11):e442.
60. Global status report on noncommunicable diseases 2010. Geneva, World Health Organization, 2011.
61. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus. Geneva, World Health Organization, 1999 (WHO/NCD/NCS/99.2).
62. Morrish NJ, Wang SL, Stevens LK, Fuller JH, Keen H. Mortality and causes of death in the WHO Multinational Study of Vascular Disease in Diabetes. Diabetologia 2001, 44 Suppl 2:S14–S21.
63. Global data on visual impairments 2010. Geneva, World Health Organization, 2012.
64. Roglic G, Unwin N, Bennett PH, Mathers C, Tuomilehto J, Nag S et al. The burden of mortality attributable to diabetes: realistic estimates for the year 2000. Diabetes Care, 2005, 28(9):2130–2135.