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To Dispel Darkness Of Diabetes

DIET MANAGEMENT

EXERCISE

MEDICATION

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Motivating diabetic towards better adherence to diabetes care

Smita Shah, Hardik Chandarana

ABSTRACT

The education and motivation to diabetic patients is an essential therapeutic tool since the early 1920s and well accepted in 1970s. In the last decade it has generated great enthusiasm. The objective of the educational programs is to secure better adherence of patients towards diabetic care at different levels: skills required for treatment, knowledge of the disease, capacity to integrate therapy in everyday life. Most important goal of diabetes management is to achieve therapeutic goal of each patient that is prevention of acute and chronic complications. Motivating patients to adopt a healthful treatment plan during brief office visits is a major challenge facing health care providers. Therefore, effective communication strategies that can be successfully employed during time-pressured consultations are worthy of consideration. Traditional approaches to patient care often rely on advice-giving and direct persuasion. However, specific training, good communication, teaching skills, supportive attitude, readiness to listen and to negotiate is required in diabetes care. Patients adhere to diabetes care and motivation to learn is also greatly influenced by individual factors such as psychological, environmental and economical.

Key words: Diabetes, Environment, Economical, Normoglycemia, Psychological

INTRODUCTION

Diabetes is a chronic challenging disease to manage successfully. It requires major changes in lifestyle to optimize the management. Although the diabetes care regimen is complex, patients with good self-care knowledge and behaviours can achieve desired glycemic goals. However, many patients do not achieve good glycemic control and continue to suffer. Diabetes health care providers know that if only their patients adhered to their treatment recommendations, they could do well and avoid diabetes-related complications.[1]

The management of diabetes mellitus in primary care, with its rising prevalence associated with increasing obesity and sedentary lifestyle worldwide[2-5], has become a priority. It remains a challenging task, because effective interventions have to take into account multiple aspects of care[6], and involve a collaborative approach between health professionals[7-9], patients and communities.[7-10]

Motivating behaviour change in diabetic patients is one of the most important but also most frustrating experience for general practitioners. There is great diversity in patients' acceptance and understanding of diabetes. One's own diabetes is often perceived as less serious than that of others and short-term comfort may be prioritized over long-term consequences. Knowledge about diabetes is not well correlated with the personal use of that information and patients' goals may differs considerably from the doctor's.[11-14]

Patient compliance and adherence to medical treatment is low, especially in the case of chronic illness and prescribed self-administered medication. Low adherence is a growing concern, seriously undercutting the benefits of current medical care. Efforts to assist patients to follow treatments might improve the efficiency of care and substantially enhance benefits.[15-16]

Medical non-compliance has been identified as a major public health problem that imposes a considerable financial burden upon modern health care systems[15]. The medical and economic implications of compliance tend to go hand in hand, but are specific to the therapeutic field and drug concerned. Non-compliance with a drug that is crucial

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to maintenance of vital physiological functions is more likely to have serious consequences than to medicine intended for symptomatic relief[17]. Because compliance is a word with negative connotations, we would prefer to use the term adherence to incorporate the broader notions of concordance, co-operation and partnership. However, as the majority of medical papers still use the term compliance, we feel obliged to maintain its use when citing published material. As the terms have different nuances of meaning, the terms compliance, adherence and concordance cannot be used interchangeably.[15, 17]

Three decades have passed since the first workshop on compliance research. An enormous amount of research on the topic has been undertaken since then, of variable methodological quality, with still no gold standard definition of compliance[17]. More than 200 different doctor, patient and consultation-related variables have been studied since 1975, but neither socio-economic nor pathology-related factors consistently predict compliance[18-22]. Patients' own beliefs and the constraints of everyday life are important in determining compliance. The patient's perspective on health and illness has only recently been taken into account in compliance research. It is important to know what sense individuals make of the advice given to them. When they arrive at the consultation, patients hold sets of beliefs and theories about health and illness. When confronted with a particular disease they will first try to deal with it and not yield control over their bodies to the medication regimen. It appears that more and more patients want to take their own decisions as consumers. Though the most prevalent influences on adherence seem to be doctor/patient communication and the beliefs that people hold about medicine in general and about their illness in particular, the prescriber's point of view is also important. This has resulted in limited research using exploratory qualitative methods rather than quantitative surveys.[15, 17-23]

Adherence to the multi-component diabetic treatment regimen requires a daily care plan. Diabetics can live a relatively normal life but chronic complications (neuropathy, myocardial and foot ischemia, renal disease, retinopathy) can result in a substantial decline in quality of life. The Diabetes Control and Complications Trial (DCCT) confirmed that improved metabolic control was significantly associated with delayed onset and progression of microvascular complications, with a clear increasing risk related to poorer metabolic control.[24-25]

To maintain adequate glycemic control, patients typically follow a self-management regimen involving frequent self monitoring of blood glucose (SMBG), dietary modifications, exercise, education, and medications. Collaboration and negotiation with health care providers, family members, and others is essential so that such behaviour changes are optimally supported and encouraged.[26]

More than 95% of diabetes care is done by the patient, and health professionals have very little control over how patients manage their illness between office visits. Patients manage their diabetes on a daily basis within the context of the other goals, priorities, health issues, family demands, and other personal concerns that make up their lives; they have the right to set goals and decide how they will manage their illness because they have to carry out those decisions and live with the consequences.[27]

MOTIVATIONAL INTERVIEWING (MI)

Motivational interviewing (MI) has recently become a topic of great interest in the diabetes behavioural field, having been the focus of workshops and research presentations at national meetings such as the Society of Behavioural Medicine, the American Diabetes Association, the North American Association for the Study of Obesity, and the Behavioural Research in Diabetes Exchange. The Motivational Interviewing Network of Trainers (MINT) was founded in 1995 and sponsors a website (www.motivationalinterviewing.org) through which MI trainers and researchers share information and ideas. This site provides information, research findings, and training opportunities and is a good starting point for further exploration of MI.[28]

Brief motivational interviewing (BMI) is an approach which provides GPs with a broader range of communication skills that are tailored to the individual patient's readiness to change. In this article BMI is applied to the diabetic patient, but the principles can be applied to any consultation that involves behaviour change. BMI is a derivation of motivational interviewing (MI), which was originally developed by specialists working in the context of alcohol addiction. MI is intended to elicit behaviour change by helping patients to explore and resolve their own ambivalence about change. It is based on an underlying patient-centered consultation style. Resistance from the patient is interpreted as a sign that the GP is being too confrontational or is incongruent with the patient's readiness to change.[28]

Key Elements of MI

Four elements of MI address both what clinicians discuss with patients and how they discuss it:

1. Express empathy
2. Roll with resistance
3. Develop discrepancy
4. Support self-efficacy.

The first two elements pertain to the practitioner-patient relationship.

EXPECTED BEHAVIOUR OF DIABETIC PATIENTS

Diet

The first behavioural change is to know what, when and how much to eat. Which food components is suitable and sufficient, which ones more directly influence blood glucose levels? Type 1 diabetic patients will have to estimate accurately the amount of carbohydrates in each meal to match the insulin dose (and adjust either one). One, two or three snacks will be necessary, according to the amount of long-acting insulin used and the timing of exercise. Type 2 patients shall keep the total caloric intake under control and reduce it until an acceptable body weight has been reached. Recent recommendations of the ADA to reduce protein intake to 0.8 g/kg ideal body weight have slightly changed the proportions of nutrients to be prescribed[28-29]. Together with the advice to increase fiber ingestion, this may demand greater changes in dietary habits than one can imagine, particularly for the higher caloric intakes of young active people.

Exercise

The medical position about the role of physical exercise in diabetes treatment has slightly changed over the past few years. Exercise is no longer considered a corner-stone in the treatment of type 1 diabetes, since it has been realized that daily variations in its duration and intensity will complicate treatment rather than simplify it. However, even if not formally prescribed, the practice of sports is commonly allowed; the additional variable added to blood glucose control needs to be taken into account and incorporated into day-to-day treatment. For its cardiovascular and psychological benefits, an active lifestyle including regular moderate exercise is strongly advised. The more regular it is, the easier it will be to adapt insulin and diet to it. Conversely, for type 2 diabetes, exercise gains more and more importance. Therapeutic exercise need not be strenuous, but should last as long as possible on a daily basis and be adapted to the age and cardiovascular status of the patient. Caution is mandatory in its prescription to unfit, elderly people, who should be advised to walk 1 h per day. Those who cannot walk should learn to practice some gymnastics at home, even while only sitting or lying.[28]

Insulin injections

All type 1 and 20-30% type 2 diabetic patients require insulin, via daily subcutaneous injections. The technique is easy, but many patients tend to forget it and start injecting insulin incorrectly. This is a frequent cause of irregular absorption from the site of injection and consequent brittleness of blood glucose levels. The use of semi-automatic injectors (‘pens’) has freed those injecting insulin before each meal from the syringe. This has made the acceptance of insulin by many youngsters much easier. As far as treatment schemes are concerned, there are two main strategies. To use large proportions of long-acting insulin in one/two daily injections is less effective in terms of control and more demanding in terms of dietary adaptations to match the insulin absorption profile; it is indicated only for some type 2 patients. Three to four injections of mainly short-acting insulin allows better results with a much freer lifestyle and, for the same glycemic target, bring about less risk of hypoglycemia.[28]

Hypoglycemia

Hypoglycemia is a threat for insulin-treated patients, due to the failure of the automatic switch-off of circulating insulin when its need decreases for some reason. Usual causes are a decrease of ingested/absorbed carbohydrates or an increase of exercise, without a concomitant reduction of the insulin dosage. Although milder episodes of hypoglycemia can be considered a routine set-back more serious episodes with signs of neuroglycopenia should absolutely be avoided. Hypoglycemia is not usually dangerous in itself, but the decrease in alertness that it can induce may cause a fall or accident.

Prevention and immediate treatment are mandatory. Patients should learn how to match carbohydrate intake, exercise and insulin dose in all situations, especially the potentially dangerous ones, particularly when there are doubts about patients’ perception of hypoglycemia. Every insulin-treated patient should carry some glucose at all times and the relatives of those patients who have experienced a coma, or

have hypoglycemia unawareness, should be ready to inject glucagon in case of need.

Blood glucose monitoring

Blood glucose monitoring is the only way for patients and care providers to know exactly the results of treatment. It can be used to answer two kinds of questions: during episodes of hyper- or hypoglycemia, to see exactly how high or how low blood glucose levels are; and randomly during everyday life, to show the usual blood glucose levels at given times of the day and night.

Foot care

The alterations typical of the diabetic foot are mainly due to chronic complications of peripheral vascular disease and neuropathy. These lead to decreased pain sensation, muscular weakness and deformities, dry skin and fissures. However, the major accidents leading to gangrene, long hospitalizations and often amputations, are almost always precipitated by trivial episodes such as wearing shoes which are too tight, a little bleeding from a cut toenail or corn, or a burn due to a hot-water bottle or walking bare foot on a heated temple floor.

Even in the presence of advanced vasculopathy and neuropathy, adverse consequences are largely preventable by avoidance of any possible foot lesion, due to physical, chemical or biological agents. This implies starting to take care of one's feet as a mother does with her newborn and it can only be achieved through so many behavioural changes that are not always easy to learn and apply, especially for elderly people.[28]

MOTIVATING DIABETIC

Behavioural modification

Behavioural changes require knowledge, skill and motivation. Educational science teaches us how to improve people's knowledge and skill, but it is the common experience that these are not enough. In order to have somebody modify a given behaviour, it is necessary that the perceived advantages of the change are greater than the perceived disadvantages.

Educational motivation and improving skill and knowledge

The health care provider must give information to the patients which is really essential for diabetic care and better outcome with greater adherence.

Patient education should be a learner-centered process. The classical educator-oriented approach, based on the idea that what the teacher does causes the learner to learn, is wrong. Teachers should accept the fact that they can only influence their patients' learning and create an environment in which patients may learn.[30]

Objectives for education

To have clear-cut objectives, agreed among all the members of the educating team, is essential for effective teaching. The objectives can be subdivided in long-term (more general), medium-term and short-term (very specific and detailed). Short-term objectives should be defined in detail as behavior change to be achieved, i.e. at the end of the educational process, patients should be able to define, describe, explain (as far as theory is concerned, or choose, avoid, perform a correct sequence of gestures ,as far as practical tasks are concerned)[31]. Also, the objectives can be subdivided into more urgent or 'first aid' (e.g. insulin injection, self blood glucose monitoring) and less urgent (e.g. self adjustment of insulin dosage), making patient education a continuous process.

COMPLIANCE AND ADHERENCE

Most of the health care providers use the term "compliance" in place of "adherence," although these concepts are quite different. The use of term adherence, is not certainly new in medical treatment and it increased use in recent times has positively made an impact in diabetes care by making practitioners more aware of patient's independence in the decision-making process. However, the term adherence is having an advantage over compliance because it implies patients rational participation while compliance has been defined as "the extent to which a person's behaviour coincides with medical advice"[32].

Non compliance essentially means that patients ignore the advice of their healthcare providers. Patient noncompliance is attributed to personal qualities of the patients, such as forgetfulness, low will power or discipline, or illiteracy. The concept of noncompliance not only assumes a negative attitude toward patients, but also places patients in a passive, unequal role in relationship to their healthcare providers. Adherence has been defined as the "active, willed, and cooperative involvement of the patient in a

mutually acceptable course of behaviour to produce a therapeutic result"[33]. In adherence the patient and therapist is mutually evolve goal setting, treatment planning, and implementation of the regimen. Patients internalize treatment recommendations and then either adhere to these internal guidelines or do not adhere. The concept of adherence has been evaluated because of its focus on patients and the nature of the diabetes regimen itself, which is dynamic rather than static[34].

MOTIVATION TIPS FOR DIABETIC

Patients with diabetes mellitus can be frightened and frustrated at times. It is important to stay motivated to follow the treatment plan to control blood sugar. Adherence to the treatment plan will help reduce the risk for complications such as heart disease, foot problems, kidney or eye disease, and depression.[34-35]

Plan for successful outcome

Good action plan is required that fits easily into your life. This is the key to staying motivated.

- Set goals that are within your reach.
- Plan exactly what you want to do.
- As you become comfortable with your plan, try to do a little more over time.
- Ask your health care team to provide you with a meal planner, an exercise planner, a blood sugar log, and a medication schedule that will help you to
- Make healthy food choices – Eat the right portions
- Watch your calories
- Make sure you have some physical activity every day – Vary your physical activities to keep them interesting
- Monitor your blood sugar level, and keep it from being too high or too low
- Take your medications as directed

Advise patients to stay on track

Teach the patients to stay motivated to eat right, keep active, and watch your blood sugar levels:

- Remember that success comes one step at a time.
- Focus on your successes—make reaching your goals a part of your life!
- Become a problem solver—if you are having trouble following the plan, think of what the problem might be and think of one step you could take to get back on track.
- Teach your family and friends about type 2 diabetes so they can help you reach your goals.
- Stay in touch with your health care team between appointments.
- Get support from your family, friends, co-workers, and health care team.
- Medical association to run a diabetes program for its employees.

FACTORS RELATED TO ADHERENCE

To improve patient adherence, it is important to understand why non-adherence occurs. A substantial literature has documented a number of factors related to diabetes regimen adherence problems. It is helpful to consider demographic, psychological, and social factors, as well as health care provider, medical system, and disease and treatment related factors.

SUMMARY

Compliance or adherence is a common issue in diabetes management. Many factors are potentially related to these problems such as social, economical, demographical, psychological, health care provider and medical system, and disease- and treatment-related factors. Chronic illness like diabetes mellitus requiring multiplicity of self-management behaviours, more skillful approach can be delivered to patients by a patients centered collaborative model for the care recognizing patient autonomy to improving diabetes self care behaviours. Health care providers should develop patient-centered dealings that respect patient independence; provide continuity of care with interim telephone contacts; brainstorm and problem-solve with their patients; organize their clinic or office to be patient-friendly; talk collaboratively with patients about treatment rationales and goals; gradually implement and tailor the regimen; use self-monitoring, provide written instructions; social supports and reinforcement, and behavioural contracts; and routinely refer patients to behavioural health specialists.

The extra burden of low motivation, confusion about reasons for change or lack of support to make desirable change combine to make sustained change unlikely. There is a need for a different approach than simply instructing the patients to change or gently

giving yet more information in the hope they may do so in their own time. Guided, structured focus on the reasons for, and ways of, changing is likely to be of more help.

MI is a type of talk therapy, a technique that can be learned by people who can listen and who are motivated to help others or themselves to find new ways to change aspects of their behaviour. The challenge is to develop MI interventions that are useable in health consultations (which tend to be brief), are teachable, and are sufficiently specific to enable proper evaluation. With such interventions, patients are likely to feel listened to and understood by their health practitioner. Health practitioners, on the other hand, are likely to gain a greater sense of achievement from recognizing change in patients' readiness as important progress, rather than seeing concrete behaviour change as the only goal. Thus, MI interventions are likely to contribute to a greater sense of satisfaction for patients and practitioners, as well as help promote health behaviour change.

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Metformin - The Ace Drug

Brij Mohan Makkar, Shalini Jaggi***

INTRODUCTION

Metformin is an oral anti-diabetic, or more precisely anti-hyperglycemic agent belonging to biguanide class of drugs that helps in control of hyperglycemia in patients with type 2 diabetes. In addition, metformin has been shown to have a host of beneficial effects including weight reduction, reduction in cardiovascular risk and mortality, and reduction in risk of cancer.

Metformin, however, is thought to increase the risk of lactic acidosis, and has been generally contraindicated in many chronic hypoxemic conditions that may be associated with lactic acidosis, such as cardiovascular, renal, hepatic and pulmonary disease, and advancing age.

HISTORY

Biguanides class of drugs originate from the French lilac or goat's rue (Galega officinalis), a plant used in folk medicine for several centuries.[1] Metformin was first described in scientific literature in 1922, by Emil Werner and James Bell, as a product in the synthesis of N,N-dimethylguanidine.[2] Though blood sugar lowering properties of metformin were reported in literature in early 20th century, the results were completely forgotten and biguanides were themselves soon overshadowed by insulin.[3,4]

Finally it was French diabetologist Jean Sterne who reinvestigated the blood sugar lowering activity of metformin and was the first researcher to try metformin on humans for the treatment of diabetes. Stern coined the name "Glucophage" (glucose eater) for the drug and published his results in 1957.[5,6] Metformin became available in the British National Formulary in 1958.[6]

Metformin gained limelight with withdrawal of the other biguanides in the 1970s. It was approved in Canada in 1972, but did not receive approval by the U.S. Food and Drug Administration (FDA) for type 2 diabetes until 1994.[6] Now metformin is believed to have become the most widely prescribed antidiabetic drug in the world.[6]

ANTI-HYPERGLYCEMIC PROPERTIES

Metformin reduces blood glucose levels by inhibiting hepatic glucose production and reducing insulin resistance primarily in liver and to some extent skeletal muscle[7] through inhibition of gluconeogenesis and lipogenesis.[9] Plasma insulin levels may show no change or a small reduction8. Metformin also decreases intestinal absorption of glucose and increases insulin sensitivity thereby increasing peripheral glucose uptake and utilization. It is one of the most potent anti-hyperglycemic agents available and lowers HbA1c levels by approximately 1.5% without causing hypoglycemia when used as monotherapy.[10,11] It has been shown to have synergistic effects when used in combination with all available oral anti-diabetic agents including sulfonylureas, glitazones, alpha glucosidase inhibitors and DDP-4 inhibitors. A combination of bedtime insulin and metformin was shown to be more effective in reducing glucose levels with a significantly less weight gain compared with bedtime insulin plus glibenclamide, bedtime insulin plus metformin plus glibenclamide, or morning and bedtime insulin.[10-15] Similarly, Metformin add on to GLP-agonists exenatide or liraglutide significantly reduced HbA1c compared to GLP-1 agonists in combination with placebo.[16,17]

There is a linear relationship between the dosage and effect of metformin. The most effective dosage of metformin observed in studies was 2000mg/day.18 Increasing the metformin dose from 2000 to 3000 mg/day only reduced fasting blood glucose levels by a further 5%, while significantly raising the incidence of gastrointestinal side effects.

METFORMIN AND SAFETY PROFILE

Common side effects include gastrointestinal upset, i.e. diarrhoea, nausea, bloating and metallic taste, seen in 1-30% of patients. Initiating metformin therapy with lower dose and increasing the dose gradually can reduce the incidence of gastrointestinal side affects. The risk of hypoglycaemia is low, almost the same as in the placebo group.[8]

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Lactic acidosis is the most dangerous side effect, fortunately rare, with an incidence of 0-0.084 cases/1000 patient years.[11,19] Risk of lactic acidosis can be avoided by observing the labelled contraindications to metformin usage i.e. impaired renal function, severe liver disease, pancreatitis, alcoholism, hypoxic states, respiratory insufficiency, severe cardiac insufficiency (NYHA III/IV), cardiovascular shock, metabolic acidosis, diabetic ketoacidosis, consumptive diseases, low serum level of vitamin B_{12}, preoperative, perioperative and postoperative states, radiological procedures using contrast, advanced age, and calorie restrictions (<1000 cal per day).[11,20] A Cochrane database review of pooled data from 347 comparative trials and cohort studies revealed no cases of fatal or nonfatal lactic acidosis in 70,490 patient years of metformin use or in 55,451 patients-years in the non-metformin group. There was no difference in lactate levels, either as mean treatment levels or as a net change from baseline, for metformin compared to non-metformin therapies. The authors concluded that there is no evidence from prospective comparative trials or from observational cohort studies that metformin is associated with an increased risk of lactic acidosis, or with increased levels of lactate, compared to other anti-hyperglycemic treatments.[21]

CONTRAINDICATIONS

Current U.S. prescribing guidelines warn against the use of metformin in patients with a serum creatinine ≥133 mmol/L (≥1.5 mg/dL) in men or ≥124 mmol/L (≥1.4 mg/dL) in women.11 Metformin is eliminated renally, and cases of lactic acidosis have been described in patients with renal failure.11 There is an ongoing debate, however, as to whether these thresholds are too restrictive and that those with mild–moderate renal impairment would gain more benefit than harm from using metformin.[22,23] Current diabetes management guidelines and clinical practice recommendations generally allow use of metformin down to a GFR of 30 mL/min, with dose reduction advised at 45 mL/min.[24-26] Given the current widespread reporting of estimated GFR, these guidelines appear very reasonable. Recently an observational study of more than 50 thousand patients from Swedish National Diabetes Register reported that metformin treatment in patients with renal impairment showed no increased risk of CVD, all-cause mortality or acidosis or serious infection.[27]

Previously contraindicated in heart failure, metformin can now be used if the ventricular dysfunction is not severe, if patient's cardiovascular status is stable, and if renal function is normal.[28]

CURRENT STATUS IN THE MANAGEMENT OF TYPE 2 DIABETES

Metformin remains the most commonly used first line agent in the management of type 2 diabetes. It is generally agreed that metformin, if not contraindicated and if tolerated, is the preferred and most cost-effective first line agent, in addition to lifestyle interventions, for treatment of type 2 diabetes.[24-26]

METFORMIN AND BODY WEIGHT

Metformin therapy is largely weight neutral. While insulin secretagogues, glitazones and insulin usage for treatment of type 2 diabetes lead to weight gain, treatment with metformin usually results in no change in body weight or a modest weight loss, and in combination with other agents it may mitigate weight gain.[8,11,29-32] This may be partly due to anorectic effect of metformin.[9] Adding metformin in patients sub-optimally controlled on insulin therapy compared with intensification of the insulin dose by 20% resulted in lower body weight, lower body mass index, lower insulin dose and HbA1c at the end of the study in the metformin arm.[33] Modest weight loss with metformin has also been observed in subjects with impaired glucose tolerance enrolled in the Diabetes Prevention Program[34] and in the Indian Diabetes Prevention Program[35]. Follow up of Diabetes Prevention Program showed that over a follow up period of 7-8years metformin used for diabetes prevention is safe and well tolerated and also that weight loss with metformin use is related to adherence to metformin and is durable for at least 10 years of treatment.[36]

There is limited data on metformin for weight loss in non-diabetic patients. Recently published MOCA trial showed that metformin treatment over a 6 month period leads to significant reduction in body mass index SD score in obese non-diabetic children.[37]

A recent study reported that compared with placebo, metformin treatment caused a significant body weight reduction in adult non-diabetic patients treated with atypical antipsychotics and in children.[38]

METFORMIN AND CARDIOVASCULAR RISK REDUCTION

Patients with type 2 diabetes have a two- to four fold higher risk of heart disease and stroke and

a reduction in life expectancy of 5-10 years as compared to general population.[39] UKPDS clearly demonstrated that metformin treatment in patients with type 2 diabetes was associated with significant reductions in the risk of myocardial infarction and diabetes-related death compared with diet.[8] A number of observational studies have shown a strong cardioprotective effect of metformin in patients with a high prevalence of cardiovascular disease, including patients with prior coronary heart disease and heart failure.[40,41] Recently a randomized, double blind, placebo controlled showed that treatment with metformin for 3 years substantially reduced major cardiovascular events in a median follow-up of 5.0 years compared with glipizide.[42] Another large population based study involving 51675 patients followed up for 4 years, showed that metformin was associated with lower risk than insulin for CVD and all-cause mortality and slightly lower risk for all-cause mortality compared with other OHA.[43]

Besides the effects on the classic cardiometabolic risk factors (dysglycemia, insulin resistance, obesity, dyslipidemia and high blood pressure) observed in type 2 DM patients and demonstrated in several studies, metformin has other potential anti-atherothrombotic actions. Treatment with metformin improves endothelial function and improves other hemostatic parameters. Metformin has been shown to have anti-inflammatory effects and also decreases oxidative stress. It may reduce the risk of chronic complications of diabetes by reducing the production of advanced glycation endproducts (AGE) indirectly, by reduction of hyperglycemia, and directly by an insulin independent mechanism. Experimental studies suggest that metformin may have anti-atherosclerotic properties.[9]

METFORMIN AND LIPID PROFILE

Diabetes Prevention Program[34] showed that in non-diabetic persons and those with impaired glucose tolerance metformin had only a modest effect on lipid profile which was lesser than that of the intensive lifestyle interventions, suggesting that the macrovascular risk reduction attributed to metformin in the UK Prospective Diabetes Study (UKPDS)[8] may be associated with other mechanisms and not due to an improvement in lipids. A meta-analysis of 41 randomized controlled trials reported that metformin caused significant reductions in total cholesterol, LDL cholesterol and triglycerides compared to other treatments.[44] However, HDL-cholesterol was rarely improved by metformin.

METFORMIN AND POLYCYSTIC OVARY SYNDROME

Polycystic ovary syndrome (PCOS) is possibly the most common endocrine disease in young women, affecting 5-10% of those in the reproductive age group. Polycystic ovary syndrome (PCOS) is associated with insulin resistance and an increased risk of developing type 2 diabetes mellitus. The attendant hyper-insulinaemia is also thought to contribute to the mechanism of anovulation in PCOS.[45] A woman with PCOS may present with several features associated with insulin resistance, such as abdominal obesity, hypertension, hyper-insulinemia, low HDL-cholesterol, increased triglycerides, and impaired fibrinolysis, acanthosis nigricans resembling the metabolic syndrome9. Both metabolic and reproductive abnormalities are amplified by obesity and the treatment of first choice for overweight or obese women with PCOS is modification of diet and lifestyle. Nevertheless, changes in diet and exercise are, for many subjects, not easy to sustain and there is evidently an obvious place for insulin sensitizing agents in management of both reproductive and metabolic disturbances.[45] A meta-analysis of studies comparing metformin with placebo vs metformin alone in women with PCOS showed that metformin not only significantly reduced fasting plasma glucose, it also reduced both systolic and diastolic blood pressures and improved dyslipidemia.[46] A Cochrane meta-analysis of trials comparing metformin and oral contraceptive pill showed significant improvement in fasting insulin and triglycerides with metformin.[47] However, both these meta-analyses showed that metformin significantly improved the hyper-androgenic state. There is no doubt that diet and lifestyle changes are the first line of treatment in overweight and obese subjects with IGT, and that applies as much (if not more) to overweight women with IGT and PCOS.[10] However, metformin, whilst not as effective as lifestyle changes, does significantly reduce conversion from IGT to T2DM.[31,45] The data regarding the effects of metformin in adolescents with symptoms of PCOS are highly encouraging. Metformin alone, or in combination with anti-androgens, and/or the oral contraceptives, appears to improve many of the clinical, endocrine and metabolic features of PCOS. There is an intriguing possibility that interventions with insulin sensitizers in adolescence can alter the natural history of PCOS and prevent (or at least retard) progression.[45] Guidelines also suggest use of

metformin as initial pharmacological therapy for most women with PCOS, especially overweight and obese, or in addition to clomifene in clomifene-resistant anovulatory women.[48,49] Although metformin crosses the placenta, observational studies have not shown any adverse effects of metfomin on fetal or neonatal development. Metformin used during pregnancy decreased the risk of gestational diabetes in women with PCOS.[9]

Metformin possibly has both central and peripheral beneficial effects in PCOS, leading to reduction in serum LH level, reduction in hepatic gluconeogenesis, increased synthesis of sex hormone-binding globulin (SHBG), and consecutively decreasing free androgen levels. Metformin also increases insulin sensitivity in peripheral tissues, reduces free fatty acid oxidation, and reduces ovarian and adrenal secretion of androgens. Although a meta-analysis of studies in patients with PCOS treated with metformin suggests no significant effect of metformin on body weight compared to placebo, some studies have reported a mean weight loss (1.5-3.6 kg) during 8 months of treatment with metformin in obese women with PCOS.[9]

METFORMIN AND NAFLD

Nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH) are associated with insulin resistance and cardiovascular and metabolic risk factors.[9] Metformin has been shown to improve metabolic parameters in patients with NAFLD.[50] However, recently published guidelines on diagnosis and management of NAFLD do not recommend use of metfomin as a specific treatment for adults with NASH.[51]

METFORMIN AND ITS POTENTIAL FOR THE TREATMENT OF NEOPLASTIC DISEASE

A growing body of evidence has suggested that metformin potentially reduces the risk of cancer. UKPDS revealed that metformin treatment reduced the risk of death from cancer by 29% relative to diet. Metformin acts at the molecular level by activating the enzyme AMP Kinase, an enzyme which has a major role in protein synthesis and cell proliferation. Activation of this pathway is a possible basis for the additional role of the anti-diabetic drug metformin as an anti-tumour agent too.[9]

The role of metformin in reducing cancer incidence in diabetic patients was demonstrated in a pioneering work by Evans et al.[53] Experimental studies in mice showed a reduction of 72% in tumour burden in preventing tobacco-induced carcinogenesis with metformin.[54] Metformin has been shown to specifically target cancer cells, while the normal epithelial cells (of breast or prostrate) are resistant to its action.[55,56] Metformin enhanced the antiproliferative effects of anti-cancer drugs in lung, breast and prostrate cancer.[57-59] Metformin intake has also been shown to improve survival in patients with ovarian cancer.[60] A meta-analysis of 6 studies involving more than 21 thousand patients showed that use of metformin in diabetic patients was associated with significantly lower risks of cancer mortality and incidence.[61]

However, a recently published meta-analysis of currently available RCT data does not support the hypothesis that metformin lowers cancer risk by one-third.[62]

OTHER POTENTIAL USES

There is very limited data on use of metformin in type 1 diabetes patients. Metformin addition to insulin has been shown to reduce insulin-dose requirement in type 1 diabetes but it is unclear whether this is sustained beyond 1 year and whether there are benefits for cardiovascular and other key clinical outcomes.[52]

CONCLUSION

Metformin as a molecule has stood the test of time. Its well documented antihyperglycemic efficacy and positive metabolic and vasculoprotective effects, supported by a number of clinical trials and meta-analyses, have positioned metformin as the first line antidiabetic agent of choice across all the guidelines. Wide spread clinical experience of more than four decades and its safety and efficacy across board in all patients certainly earns metformin the title of an Ace Drug for the management of type 2 diabetes. Several studies have suggested that metformin has beneficial effects in a number of other disease conditions with marked insulin resistance such as obesity, PCOS, NASH, and certain malignancies. Though metformin is not currently indicated for the management of these conditions and further prospective randomized studies are needed to strengthen its role as a modality of their treatment, the emerging positive evidence does indicate that this ace molecule still has a long way to go with its immense potential for the future.

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Release of e-based Diabetes Educator Course (eDEC) Website: www.helpdefeatdiabetes.org

An e-based Diabetes Educator Course (eDEC) will be released in October,2013. This will be an online course to train Diabetes Educators. Dietitians, nurses, physiotherapists, pharmacists and doctors (MBBS or above) will be eligible for this course. It will enable an individual to secure jobs at government, community and private hospitals and clinics. Additionally, pharmaceutical and nutraceutical companies active in the diabetes area may also employ the educators.

Duration of the course

- ❖ Duration of course is 6 months, with final test taken on-line under the supervision of mentor.
- ❖ Hands- on 3 months experience with one of the 50 recognized Diabetologists who will serve as mentors.

This course will provide you with:

- More than 750 pages of course reading material, which will include History, Etiopathophysiology, Epidemiology, Diagnosis and Classification of diabetes, Management of DM, Co-morbid conditions in DM, Acute complications, Chronic complications, DM through lifecycle, Self management and day-to-day care, Counseling strategies, Communications skills, New frontiers in DM
- 19 audios
- 3 Audio visual topics
- Chat sessions
- Virtual Class

Upon successful completion of the course, a certificate of training by Help Defeat Diabetes Trust and Diabetes Endocrine Nutrition Management and Research Centre will be issued.

Barriers to Insulin Therapy

Kavita Gupta & Sunil Gupta***

Introduction

IDF estimates that India alone has 61.3 million people living with diabetes; this places India second to China. The South-East Asia region has one of the highest estimates of prevalence of type 1 diabetes in children with 1,11,500 affected. In 2011, an estimated 18,000 children under the age of 15 in the region developed type 1 diabetes.[1] Out of the total diabetics only 1.2 million of these are insulin users. India has one of the lowest expenditure on healthcare in the world (184 DKK/capita per year). Healthcare insurance is not mandatory in India and so people most often spend out of their own pockets.[2]

More than 25% of people with diabetes take insulin. The American Diabetes Association and the European Association for the Study of Diabetes recently issued a consensus algorithm for management of type 2 diabetes identifying insulin as the most effective glucose-lowering agent. Lower compliance with insulin regimens is associated with higher A1C levels and with higher rates of hospital admissions for diabetes related complications. Despite the importance of adhering to prescribed insulin regimens, little is known about the degree to which patients are adherent or about factors associated with adherence.[3]

Insulin is recognized as providing the most effective treatment for many people with type 2 diabetes, but is also widely considered to be the most challenging and time consuming.[4] Despiteimprovements in lipid and blood pressure control in recent years, achieving good glycemic control remains challenging.[5-7] Significant barriers to appropriate and timely insulin initiation still exist,[8-10] and the prospect of intensifying to more complex insulin regimens and the need for more frequent blood glucose monitoring may be daunting for both patients and health care professionals alike. Patient's ability to adhere to therapy is known to be a significant problem in the treatment of chronic illnesses, and particularly in conditions such as diabetes, where patients may be asymptomatic for many years, and only develop significant morbidity in the longer term.[11]

Global Insulin Users

A review of treatment of diabetes in the United States, (2007–2009) showed that among adults with diagnosed diabetes (type 1 or type 2), only12% take insulin, 14% take both insulin and oral medication, 58% take oral medication only and 16% do not take either insulin or oral medication.[12]

Benefits of Early Introduction of Intensive Glycemic Control

The American Diabetes Association(ADA) recommends a target glycosylated hemoglobin (HbA1c) level of 7% or less to indicate glycemic control in most patients with T2DM.[13] In the seminal United Kingdom Prospective Diabetes Study (UKPDS),[14] which helped determine current targets for HbA1c, lowering glycemic levels reduced the incidence of long-term microvascular and neuropathic complications. In the early stages of T2DM, disease management focuses on dietary and lifestyle changes and oral hypoglycemic. The resulting decrease in adherence to the prescribed treatment can disrupt continuity of care and increase the likelihood that the patient will be lost to follow-up.Although the effectiveness of OADs to maintain glycemic control often diminishes with the natural progression of disease and OADs are usually unable to maintain normoglycemia indefinitely, these agents play an important role in the treatment of patients with prediabetes and early in the course of Type 2 Diabetes.[15] The progressive nature of T2DM is such that beta cell function and physiologic insulin response continue to deteriorate over time, even in the presence of good glycemic management. Therefore, despite escalating regimens of oral medications,many patients eventually require insulin therapy to achieve adequate glycemic control.

Up to 60% of patients will require insulin within 6 to 10 years of initial diagnosis and even sooner if they

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have had long-standing undetected disease.[16, 17] The role of primary care physiciansin managing T2DM continues to expand. Primary care physicians are more likely to manage T2DM than are specialists, and they are increasingly responsible for the initiation and management of insulin therapy in patients.[16] Primary care physician's confidence in introducing treatment with insulin can be instrumental in improving the long-term health of their patients.

Various Barriers to Insulin Therapy

The majority of patients with diabetes are managed in the government facilities; the rest are treated by private general practitioners or take complementary and alternative medications. The three main categories of barriers are recognized & elaborated below: (Table1)

Table 1: Patient Barriers, Healthcare Professional Barriers and System barriers[18]

I. Patient barriers
✓ Fear of side effects and pain
✓ Misconceptions about insulin
• Insulin is lethal
• Insulin is a punishment
• Insulin is a stigma among family, friends, and co-workers
• Insulin is a medication for old people
• Insulin causes sexual dysfunction
• Insulin is unlawful for Muslims
• Once on Insulin always on Insulin
✓ Needle phobia
✓ Seeking alternative treatment
✓ Lack of knowledge and self-efficacy
✓ Negative influence from family members
✓ Cost Factor
✓ Hindrance in carrying out day to day activities
✓ Anxiety about proper injection technique
✓ Inconvenience of daily injections
✓ Concerns about potential hypoglycemia
✓ Uncertainty of the clinical benefits of insulin
✓ Misconception that insulin is unnecessary because other treatments have worked in the past
✓ Concern for weight gain
✓ Insulin is an indicator that disease has worsened hence fear ofserious complications (e.g., organ deterioration, blindness)
✓ Psychosocial causes of patient resistance include a sense of personal failure and discomfort about administering insulin in public
✓ From the patient's perspective, the need for insulin indicates that his or her inability to control diabetes mellitus through lifestyle changes, weight loss, or adherence to oral therapy has led to a more serious and invasive form of therapy

II. Healthcare Professional barriers
✓ Risk of Hypoglycemia
✓ Negative attitudes towards insulin
✓ Lack of motivation and confidence
✓ Training-related barriers
✓ Conflicting advice from the healthcare professionals who are not updated.
✓ Uncertain of potential benefits of insulin therapy
✓ Concerns about patient adherence to prescribed regimen
✓ Belief that there is increased cardiovascular risk.
✓ Belief that additional time will need to be dedicated if hypoglycemia episodes increase infrequency and severity
✓ Fear of losing the patient.

III. System barriers
✓ Lack of continuity of care
✓ Lack of manpower
✓ Lack of resources
✓ Language barriers
✓ Economic barrier

Barriers to Initiationof Insulin Therapyfor the Managementof Type 2 Diabetes Mellitus

I. Patients:-

Effective glycemic control is essential to minimize the long-term complications of type 2 diabetes mellitus (T2DM). However, many patients spend prolonged periods above the optimal glycemic range.

When lifestyle modifications and oral anti diabetic medications are not sufficient to maintain glycemic control in patients with type 2 diabetes, expert consensus guidelines and evidence-based algorithms recommend insulin initiation and subsequent insulin progression (switching from a basal insulin regimen to a premixed insulin, adding bolus doses, and/or increasing the frequency of dosing) to achieve hemoglobin A1C targets.[19]

Patients show attitudinal reluctance to initiate insulin therapy due to multiple concerns, starting from the fear of self-injection ,the pain, to its side effects, worries about potential weight gain,risk of hypoglycemia and many more.In addition, psychosocial causes of patient resistance include a sense of personal failure and discomfort about administering insulin in public. Psychological insulin resistance may manifest as resistance to initiation of insulin therapy and to escalation of the insulin regimen as the disease progresses. A sense of personal failure represents one of the strongest obstacles to patient acceptance of insulin. From the patient's perspective, the need for insulin indicates that his or her inability to control diabetes mellitus through life style changes, weight loss, or adherence to oral therapy has led to a more serious and invasive form of therapy.[20]

The risk of severe nocturnal hypoglycemia, is also one of the major dose-limiting effect of all insulins. Incidence of hypoglycemia is generally lower in T2D than in T1D patients, although the difference diminishes with longer disease duration.[21] Few studies using continuous glucose monitoring have proven that episodes of low glycemia (including nocturnal events) occur more frequently than was known from analysis depending on clinical symptoms and/or older monitoring approaches.[22,23] Insulin treatment may lead to an adverse consequence of weight gain, the physiological basis of this response is not well understood since normal insulin production does not cause weight gain but some authors have speculated that weight gain is related to the fear of hypoglycemia, because patients rightly or wrongly perceive a need to snack defensively to maintain euglycemia (Defensive Snacking).[24]

At the initiation of the patient-physician relationship it can be favorableif the patient is well informed that insulin is a part of the normal continuum of care as disease progresses.[25] Thus patients understandthat the need for insulin is not a drastic or punitive measure butrather an expected step stemming fromthe progressive nature of the disease itself. Thus the stigma of insulin therapy fades.[26] Several forms of insulin non-adherence can be distinguished by consulting pharmacy records. For instance, 'primary non-adherent' patients are those who simply fail to fill their first prescription. These are patients with poor persistence, which discontinue treatment at some point and fail to refill their prescription. Frequent dose omission, which may be deliberate or accidental, can account for a pattern of continuous but infrequent refills.[24]

In the survey by Polonsky et al,[9] the negative attitude that most strongly distinguished unwilling from willing insulin-naïve patients wasthe sense that they had failed to properlymanage their disease.

Only one-quarter of insulin-naïve patients express unwillingness to take insulin if prescribed, and another 25% are only slightly willing to take insulin (Table 2). Common excuses observed from the Polonsky et al[9] survey data for not initiating insulin therapy were "Insulin can cause harm, eg. Blindness, Personal Failure, Low efficacy…"

"Insulin means … My Diabetes has become more serious , once on insulin always on insulin…"

"Insulin is painful … will restrict my day to day life…."

II. Healthcare Providers:-

Almost all primary care physicians are mindful about the goals for glycemic control in patients with T2DM but still many of these physicians are resistant to initiate insulin.[25,27] Physicians resistance to prescribing insulin maybe due to either physiologic or practical concerns.

Physiologic concerns include apprehension about weight gain, hypoglycemia and the belief that insulin causes adverse metabolic effects. ***Practical concerns*** include patient's anxiety, intricacy in training patients and further if the patient is illterate.[28]

Physician Barriers

A web-based survey, of 600 physicians across 6 countries was done to explore the barriers to insulin progression.[21] About 40% of primary-care physicians and 30% of specialists found administration of insulin therapy difficult and required more support staff and resources to assist them. About one-half (49%) reported that doctors lack experience with available types of insulin and that educating patients regarding progression would be time consuming. 40% agreed

Table 2: Results of Survey of Insulin Naïve Subjects With Type 2 Diabetes Mellitus Who Were Unwilling to Take Insulin if Prescribed (N=708)*

Survey Item	Survey Respondents %			
	Unwilling	Willing	Total	*P* Value†
Expected harm: Insulin therapy can cause problems, such as blindness	16.7	8.0	10.1	0.005
Illness severity: Taking insulin means my diabetes will become a more serious disease	46.7	35.4	38.1	0.000
Restrictiveness: Insulin therapy would restrict my life; it would be harder to travel, eat out.	56.1	41.6	44.8	0.000
Lack of fairness: I've done everything I was supposed to; If I had to do insulin therapy. It just wouldn't be fair	41.5	21.9	26.8	0.000
Anticipated pain: I couldn't take the needle every day; It would be just too painful	50.8	30.2	34.7	0.000
Problematic hypoglycemia: Insulin therapy might cause serious problems with low blood sugar.	49.3	37.9	40.6	0.021
Low self-efficacy: I'm not confident I could handle the demands of Insulin therapy	58.1	39.7	43.9	0.000
Personal failure: Insulin therapy would mean I had failed, that I hadn't done a good enough job taking care of my diabetes.	55.0	33.6	38.4	0.000
Permanence: Once you start insulin, you can never quit.	53.1	42.6	44.9	0.000

* Data are percentages of subjects who agree (either mildly, moderately, or strongly) with each barrier.
† P values compare differences between willing and unwilling subjects.

that patients cannot cope with advanced regimens, there is a lack of guidance about insulin progression, and monitoringto show when type 2 diabetics require progression.

In a study that explored injection-related anxiety among 80 patients with type 1 and 35 with type 2 diabetics, found that nearly 30% of patients injecting insulin were reported of being troubled by the prospect of additional injections, and 14% reported avoiding injections altogether.[19]

Even physicians, who aim at an achievement of adequate glycemic control, do not initiate insulin under the exposure of an insufficient HbA1c level which can increase a risk of subsequent diabetes complications .Few physicians underestimate the significance of achieving a targeted HbA1c level recommended by the guidelines. In addition, the gap was observed between this HbA1c value and actual value at which insulin therapy was recommended to the patients. Patients were actually recommended insulin therapy at an HbA1c level of 9.6%.An assumption of patient resistance for insulin initiation may be one possible explanation delaying an introduction of insulin therapy to the patients. This discordance is referred to as clinical inertia. Clinical inertia is defined as the recognition of a problem with a patient's management but a failure to act. Factors contributing to clinical inertia can include an assumption of patient resistance, actual resistance by the patients, failure to set clear target level, or insufficient communication with patients.[29]

The Diabetes Attitudes, Wishes, and Needs (DAWN) study found that many US physicians, involved in primary care, delay initiation of insulin therapy. In fact insulin initiation is not triggered until a patient's HbA1c level reaches > 9%.[30] US physicians ranked lowest among physicians in all nations except Japan and India in their disposition to initiate insulin.[25]

The DAWN Study: Diabetes Attitudes, Wishes and Needs

The DAWN study explored attitudes, Wishes and needs among patients, physicians and health care politicians in 11 regions (Australia,France,Germany,India, Japan, Nether lands, Poland, Scandinavia, Spain, UK and USA). In interviews with 2061 non-insulin treated patients and 2681 physicians the DAWN study explored psychological resistance to insulin among patients and physicians. The study demonstrated that:

- The majority of patients (57 %) were worried about starting insulin
- 23% of patients thought insulin could help them improve their diabetes

- 48% of patients believed that starting insulin meant that they had not followed treatment recommendations
- Patients were more worried about starting insulin if they were younger, female or had complications
- 42% of physicians were reluctant to start insulin until absolute essential and 16% were unsure
- Physicians were less resistant to prescribe insulin if they were
  - Younger, specialists or located in a multidisciplinary setting or
  - If they were teaching diabetes for providers or lay persons.

The DAWN study concludes that a substantial proportion of patients are worried about starting insulin. This worry may be an important element in psychological resistance to using insulin. A substantial proportion of physicians are resistant to prescribe insulin and this resistance may be an important element in the delay of insulin use, and may keep patients unable to manage their diabetes effectively.[31]

III. System Barriers:-

The lack of manpower is apparent especially in the government hospitals and clinics. Despite recognizing the important role of a nurse educator in insulin counseling, only a small number of diabetes nurse educators and dieticians are trained in the government sector and, when present, they have to handle heavy patient loads.

While insulin is subsidized in the public clinics and hospitals, there is no financial assistance for glucometers and test strips. This hampers insulin initiation.[18]

Role of Healthcare Professionals and Diabetes Educators in Overcoming Insulin Barriers[32]

Tips for insulin initiation in type 2 diabetes

- Address need for future use of insulin at diagnosis/or early in the disease process.
- Emphasize not as a failure, but a treatmentupgrade.
- Do not see as a last resort and thus delay too long.
- Be ready to address barriers to initiation.
- Individualize therapy: choose the right insulin forthe patient.
- Adjust other oral or injectable therapies when appropriate

Insulin is an upgrade, not a failure - Removing the word "failure"

One unfortunate consequence of the late positioning of insulin within the current treatment paradigm is that it is often viewed as a treatment of "last resort." This iscompounded by references throughout diabetes literature that uses the term "failure" Every effort must be made to help patients to understand the progressive nature of diabetes. The need for advancing therapy reflects a disease state and not a "personal failure."

Patient misconceptions surrounding insulin

The successful understanding and application of any treatment, including insulin, requires the patient to be an enthusiastic and motivated partner in the treatment decision. This is of vital importance, as many patients often present with the preconception that insulin will not effectively provide glycemic control. Similarly, many patients attribute the complications of diabetes to insulin use, and not suboptimal glycemic control. Fear of potential side effects, particularly weight gain and hypoglycemia, also deteriorates patients from admitting the need for insulin initiation.

Patient misconceptions surrounding disease progression

Consequently, it is crucial that patients understand that progression of the disease is a natural feature of their diabetes and not a personal failure. Patients should be encouraged to feel that, despite the inevitability of disease progression, they are successfully addressing the need for aggressive treatment and that insulin is an essential component in this process.

Make it about patient control

Individualized patient education & proper counseling should be an integral part of the disease management process, during which the outcomes and clinical implications of insulin should be communicated in an easily understandable way (IDF, 2005).

Patient-driven algorithms

Patient-driven algorithms that empower patients to self-manage their diabetes have been shown to improve glycemic control and disease outcome. A study (Meneghini et al., 2007) found that empowering patients to self-manage their insulin dose, through the use of simple, patient-directed, titration algorithms, led to measurable improvements in their condition.

Because T2D is progressive, dose titration and treatment intensification are needed to ensure that insulin therapy confers long-term benefits.

Choosing the right insulin

Individualizing therapy by choosing initial insulin that best meets the patient's specific needs will help to build patient confidence. The different types of insulin available offer the opportunity for tailoring different treatment regimens. Numerous insulin preparations, with array of pharmaco dynamic properties, are available.[33] These preparations include insulin analogs that off ercertain advantages over non analog (i.e. human insulin) alternatives. Among these advantages are improved physiologic time-action profiles and lower incidence of hypoglycemia.[34] Insulin analogs include rapid-acting analogs (for prandial insulin coverage), long-acting analogs(for basal coverage), and premixed insulin analogs (which combine both a rapid acting and an extended-duration component in a single formulation).[35]

Choosing newer devices of Insulin delivery

Current insulin and injection devices are much better accepted. The risk of hypoglycemia is reduced and there is greater flexibility in dosing. The use of pen devices, Insulin Pumps coupled with sensitive counseling and support from the Healthcare providers can make insulin initiation an easy process for the patient.

Conclusion

Insulin is a vital tool in the treatment of diabetes and more must be done to identify and address the issues that prevent insulin being used in an optimal fashion. Whilst insulin is recognized as providing the most effective treatment in type 2 diabetes, it is also widely considered to be the most challenging and time consuming. The organization of existing healthcare services, the challenges faced by patients, and the treatments themselves contribute to suboptimal insulin management. In order to improve future diabetes care, it will be necessary to address these problem areas:

(1) Re-think the best use of existing human and financial resources to promote and support patient self-management, adherence and compliance to the treatment;

(2) Empower patients with necessary knowledge and awareness to participate more actively in treatment decision making, thereby, they can titrate or intensify their treatment adequately;

(3) Improve acceptance, persistence and adherence to therapy by continuing to refine insulin therapy and treatment regimens in terms of safety, simplicity and convenience; and

(4) Caregivers need to provide training or answer questions about insulin's risks and benefits, ongoing communication between patients and caregivers is the important bridge to good glycemic control.

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Cooking Oils: Strike The Right Balance

Sushila Sharangdhar

Introduction

India is already known as the diabetic capital of the world. The country currently has the highest number of 50.8 million people suffering from diabetes (China-43.2 million, US-26.8 million). With the unhealthy lifestyle and eating habits being the trend amongst the youths, there is a rapid increase in the number of Indians suffering from heart diseases. As per the World Health Organization (WHO) report, heart diseases would be the largest cause of death and disability in India by 2020. We also have the upcoming obesity epidemic and marked increase in cancers among the population.

Out of the hot topics on diet, the major culprit is "FATS", especially for what it has done to our ***waistlines and hearts!!*** Eating the right kind of fat is a critical issue as dietary fat gets much of the blame for causing heart disease. We use cooking oils for frying, baking, stir-fries, salads and marinades, but using the right one can be confusing. Consumers are bombarded with such a wide variety – not to mention all the different descriptors: "extra light", "cholesterol-free", "cold-pressed", "mono-" and "polyunsaturated" and "blended vegetable oil". Fats and oils have varying proportions of various fatty acids referred to as saturated(SFA), monounsaturated(MUFA) or polyunsaturated. (PUFA). A fat or oil usually contains all three types of fatty acids, but is characterized by the fatty acid found in the largest amount. Plant oils are good sources of monounsaturated and polyunsaturated fats. Polyunsaturated fats include omega-6 and omega-3. Let us understand more about these types of fatty acids, their sources and their effect on serum cholesterol as shown in Table 1.

Essential Fatty Acids

These fats have essential roles in the body and must be supplied in the diet as the body cannot make the PUFA's(omega-6s and omega-3s).

- The essential polyunsaturated fats are found in cell membranes and are converted to biologically active substances that play a role to help prevent chronic disease.
- The polyunsaturated fats are also critical in the development of the central nervous system. Omega-3 fats are found in high concentration in the brain and retina of the eye.

Table 1: Types of Dietary Fatty Acids

Type of fat	Important sources	State at room temperature	Effect on Cholesterol
Saturated	Whole milk, butter, cheese, and ice cream; red meat; chocolate; coconuts, coconut milk and coconut oil	solid	Raises both LDL and HDL
Monounsaturated	Olives and olive oil, canola oil, peanut oil; cashews, almonds, peanuts, and most under nuts; peanut butter; avocados	liquid	Lowers LDL; Raises HDL
Polyunsaturated	Corn, soybean, safflower ,and cottonseed oils, fish	liquid	Lowers LDL; Raises HDL
Trans	Most margarines, vegetable shortening; partially hydrogenated vegetable oil; deep fried fast foods most commercial baked goods	Solid or semi solid	Raises LDL*
* Compared to monounsaturated or polyunsaturated fat, trans fat increases LDL, decreases HDL, and increases triglycerides			

Eat, Drink and Be healthy ,Walter.C Willett, M.D. ,co-developed with the Harvard School of Public Health

LDL - Low Density Lipoprotein, **HDL** - High Density Lipoprotein

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- PUFA's (omega-6s and omega-3s) are found primarily in plant and fish oils.

About Omega-3 Fatty Acids (n3PUFA's)

- Omega 3 derived hormones include ones that regulate blood clotting, contraction and relaxation of artery walls and inflammation.
- Importantly, these fats have been shown to help prevent or treat heart disease and stroke.
- They help reduce serum triglycerides and total cholesterol levels.
- They may also help control lupus, eczema, and rheumatoid arthritis and may play protective roles in cancer and other conditions.

The three major types of omega-3 fatty acids are alpha-linolenic acid (ALA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA). ALA is found in seeds, vegetable oils (canola, flaxseed, and soybean), green leafy vegetables, nuts, and beans. ALA is converted, usually in small amounts, into EPA and DHA, after it is ingested. Fish oil and fatty fish such as salmon, mackerel, herring, and tuna are the primary sources of EPA and DHA (marine omega3's) Algae oils are a vegetarian source of DHA.

Many of the biological benefits attributed to omega-3 fat are from the "longer-chain" type found in "fatty" fish. The human body can convert the "plant-type" omega-3 fat to the "fish-type." However, the body's conversion to the longer-chain omega-3 fat is not very efficient. Thus, the "fish-type" omega-3 fat has a higher level of biological potency, but on a daily basis it is much easier to include oils containing "plant-type" omega-3 (such as flaxseed, canola or soybean).

Ratio of Omega-6 to Omega-3 Fat

Today's typical Indian diet and many diets around the world tend to contain 14 - 25 times more n6 than n3 fatty acids. The World Health Organization (WHO) recommends an n6:n3 ratio of 5-10:1. Getting the recommended amount of omega-3 fat in the diet is more difficult since omega-3 is found in smaller amounts than omega-6 in most oils. Achieving healthy intakes of both omega-6 and omega-3 FAs is an important component of the nutritional prevention and treatment of coronary heart disease.[1]

Of all the diets in the world, the Mediterranean diet has a healthier balance between omega-3 and omega-6 fatty acids. Many studies have shown that people who follow this diet are less likely to develop heart disease. The Mediterranean diet does not include much meat (which is high in omega-6 fatty acids) and emphasizes foods rich in omega-3 fatty acids, including whole grains, fresh fruits and vegetables, fish, olive oil, garlic, as well as moderate wine consumption.

Trans fats

Unlike other dietary fats, trans fats are not essential, and they do not promote good health. The consumption of trans fats increases one's risk of coronary heart disease by raising levels of "bad" LDL cholesterol and lowering levels of "good" HDL cholesterol. Trans fats from partially hydrogenated oils are more harmful than naturally occurring oils.

Several large studies indicate a link between consumption of high amounts of trans fat and coronary heart disease and possibly some other diseases. The United States Food and Drug Administration (FDA), the National Heart, Lung and Blood Institute and the American Heart Association (AHA) all have recommended limiting the intake of trans fats.

Choosing the Right Oil

For a normal adult **WHO** recommends 30% calories in diet from fats. (SF < 10%, PUFA-8%, MUFA-12%) Whilst it's important to note that all oils are similar as per their calorie content, (9 calories/gram) and should be used only sparingly, as part of an otherwise healthy diet, the oils do differ regarding like methods used for oil extraction, composition and heat tolerance. The methods used for extraction would determine whether an oil is refined (extracted from oil cakes involving solvent extraction) or unrefined (cold pressed). ***Unrefined oils, filtered*** are considered better and recommended due to the presence of a wide range of bioactive compounds, flavons and Vitamin E content, which tend to prevent rancidity in them. The differences between fats/oils (flavor, mouth feel, smoke point, stability, shelf life, and health attributes { best n6:n3 ratio, more MUFA than PUFA, least amount of saturated fat, nil amount of trans fat} are related to their chemical make up that is the way the chains of carbon, hydrogen and oxygen atoms are hooked together

Composition of Different Fats

To determine the usefulness and appropriateness of different oils in preparing food, let us analyse their compositions.

Olive Oil

- contains 75% MUFA,(highest of all the oils), along with 15% saturated fat, 9% omega-6 linoleic acid and 1% omega-3 linolenic acid.
- The Extra virgin olive oil is also rich in antioxidants.
- Since extra virgin, cold pressed olive oil has comparatively lower smoking point, it is best used as salad dressings or for light sautéing and stir frying.
- Olive oil has indeed withstood the test of time and is still considered one of the best.

Groundnut/Peanut Oil (GNO)

- contains 48% MUFA, 18% saturated fat and 34% omega-6 linoleic acid.
- Like olive oil, groundnut oil is relatively stable but again it has a good percentage of omega-6, so use of groundnut oil should be limited.

Sesame Oil

- contains 42% MUFA, 15% saturated fat, and 43% omega-6 linoleic acid.
- Sesame oil is similar in composition to groundnut oil.
- It can be used for frying because it contains unique antioxidants that are not destroyed by heat.
- However, the high percentage of omega-6 is again a major drawback.
- Sesame oil (SO) is known to be stable against oxidative deterioration and its keeping quality is mainly attributed to the presence of endogenous unsaponifiable components such as sesamolin, sesamol and sesamin (absent in other vegetable oils). A study conducted by Central Food Technological Research Institute (CFTRI), Mysore showed that sesame oil is very stable against oxidative deterioration compared to sunflower (SFO) and groundnut oils (GNO) at room temperature over a period of time.
- Blending of SO with GNO and SFO increased the shelf life of blended oils at room temperature and heated oils and the oxidative stability of blended oils.[2]

Safflower, Corn, Sunflower, Soybean and Cottonseed Oils

- All contain over 50% omega-6 and, except for soybean oil, only minimal amounts of omega-3.
- They have much lesser MUFA's. Of these, Safflower oil contains the most omega-6 (almost 80%).
- Researchers are just beginning to discover the dangers of excess omega-6 oils in the diet, whether rancid or not.
- Use of these oils should be strictly limited.
- They should never be consumed after they have been heated, as in cooking, frying or baking.[3]

Canola Oil / Rapeseed / Mustard

- Canola oil contains 5% saturated fat (the least among all commercially available oils), 57% oleic acid, 23% omega-6 and 10%-15% omega-3(so high MUFA along with beneficial omega-3) .
- The original rapeseed plant was high in erucic acid, which is an unpalatable fatty acid having negative health effects in high concentrations. 'Canola' is genetically altered and improved version of rapeseed. 'Canola' is a registered trade mark of Canadian Oil Association and contains less than 1 percent erucic acid.[4]
- Actually, another name for canola oil is LEAR (Low Erucic Acid Rapeseed) oil. In India, the "Hyola" is only hybrid 'canola' quality gobhisarson notified by Govt. of India recently after extensive trials by Indian Council of Agricultural Research (I.C.A.R.).
- The Indian kachi ghani mustard oil has a very close composition to canola oil (given below). It has anti-oxidants, allyl-isothiocynates, phenolics (anti-bacterial properties), phytins and also absence of trans fatty acids (as it is cold pressed oil) and presence of Vitamin E, makes the mustard oil heart healthy and one of the best oils for cooking.

Rice bran oil: Rice bran oil (RBO) is not a popular oil worldwide, but it is in steady demand as a so-called "healthy oil" not only in Japan but also in Asian countries, particularly India.

As shown above in **Table 2**, RBO has a nonspecific fatty acid profile, but it does contain a detectable amount of -linolenic acid, ranging from 1 to 3% (mean value, 2%). This amount may be enough to increase the content of (n-3) highly polyunsaturated fatty acids such as EPA and DHA in tissue phospholipids compared with other vegetable oils such as corn oil.[5] The remaining major fatty acid in RBO is palmitic acid, (17%). Rice bran oil lowers serum total

Table 2: Comparison of Fatty Acid available in different Edible Oils

Fats/Oil	Saturated Fatty Acid (SFA)	Mono-unsaturated Fatty Acid (MUFA)	Linoleic Fatty Acid (PUFA), Omega 6	Alpha-Linolenic Fatty Acid (PUFA), Omega 3	Ratio of n6:n3	Predominant Fatty Acid
Mustard	8	70	12	10	6:5	MUFA
Soybean	15	27	53	5	10.6:1	PUFA
Palm Oil	45	44	10	<0.5	20:1	SFA+MUFA
Red Palm	50	40	9	<0.5	18:1	SFA+MUFA
Groundnut	24	50	25	<0.5	50:1	MUFA
Sunflower	13	27	60	<0.5	120:1	PUFA
Rice Bran	22	41	35	1.5	23:1	MUFA+PUFA
K.S.Oils Limited						

Ideally the ratio of n6:n3 should not exceed 10:1

and low density lipoprotein cholesterol and apo B levels in nonhuman primates.[6]

In recent years, studies have indicated that Ferulic Acid (FA) found in cell walls, prevents LDL from oxidation, exhibits inhibitory effects on tumor promotion, and protects against certain chronic diseases such as coronary heart disease and some cancers.[7] Numerous studies show rice bran oil reduces LDL without reducing good cholesterol- HDL. In those studies, Oryzanol is reported as the key element responsible for that function. Tocotrienol, on the other hand, is highlighted as the most precious and powerful vitamin E existing in nature and is said to have an anti-carcinogenic effect, too. As a Vitamin-E source, rice bran oil is rich not only in alpha Tocopherol but also has the highest amount of Tocotrienol in liquid form vegetable oils.

Suzuki and Oshima[8] studied the effect of blending different vegetable oils on serum cholesterol levels of healthy young women.[9-10] The most interesting observation was that the blend of 7 parts of RBO with 3 parts of safflower oil unexpectedly enhanced the cholesterol-lowering potential of RBO.

Flax Seed Oil contains 9% saturated fatty acids, 18% oleic acid, 16% omega-6 and 57% omega-3. With its extremely high omega-3 content, flax seed oil is an excellent top-up for the lack of n3 and n-6/n-3 imbalance so prevalent globally today. New extraction and bottling methods have minimized rancidity problems. It should always be kept refrigerated, never heated, and consumed in small amounts in salad dressings and spreads.[3]

Ghee: Though Ghee has a higher percentage of SFA's, its n6:n3 ratio is about 4, which is very good. A study on 63 healthy physically active adult volunteers (52 men and 11 women) was conducted at AIIMS, New Delhi following a randomized controlled parallel design. The experimental group was provided ghee and mustard oil in diet for 8 weeks. Their serum total cholesterol and HDL cholesterol level increased while LDL cholesterol level did not show any change. The study did not indicate any adverse effect of ghee on lipoprotein profile.[11]

Chicken Fat is about 31% saturated, 49% monounsaturated (including moderate amounts of antimicrobial palmitoleic acid) and 20% polyunsaturated, most of which is omega-6 linoleic acid.[12]

Lard or pork fat is about 40% saturated, 48% monounsaturated (including small amounts of antimicrobial palmitoleic acid) and 12% polyunsaturated. Lard is stable and a preferred fat for frying widely used in Asian countries. It is a good source of vitamin D, especially in third-world countries where other animal foods are likely to be expensive.[12]

Tropical Oils are more saturated than other vegetable oils.

Coconut oil is 92% saturated with over two-thirds of the saturated fat is in the form of medium-chain fatty acids (MCT). Of particular interest is lauric acid, found in large quantities in both coconut oil and in mother's milk. This fatty acid has strong antifungal and antimicrobial properties. Coconut oil protects

tropical populations from bacteria and fungus so prevalent in their food supply; as third-world nations in tropical areas have switched to polyunsaturated vegetable oils, the incidence of intestinal disorders and immune deficiency diseases has increased dramatically.(3) A report published in the American Soc.for Nutritional Sciences, showed that MCT may increase energy expenditure, may result in faster satiety and facilitate weight control when included in the diet as a replacement for fats containing Long Chain Triglycerides.(13) However, more work is required to establish whether prolonged consumption of MCT helps decrease in body weight or helps control weight gain.

Palm oil is about 50% saturated, with 41% MUFA and about 9% linoleic acid. Currently palm oil is a major edible oil commodity in more than 132 countries worldwide.

Highly saturated tropical oils do not contribute to heart disease but have nourished healthy populations for millennia. Human beings have been consuming saturated fats from animals products (meat, milk, butter) and the tropical oils for thousands of years; In fact, it is the advent of modern processed vegetable oil accompanied with lack of physical activity that is associated with the epidemic of modern degenerative disease. The saturated fat scare has forced manufacturers to abandon these safe and healthy oils in favor of hydrogenated soybean, corn, safflower and sunflower oils.

Cooking with oil

Heating oil changes its characteristics. Oils that are healthy at room temperature can become unhealthy when heated above certain temperatures. When choosing cooking oil, it is important to match the oil's heat tolerance with the cooking method.[14]

A 2001 parallel review of 20-year dietary fat studies in the United Kingdom, the United States of America, and Spain[15] found that polyunsaturated oils like soya, canola, sunflower, and corn oil degrade easily to toxic compounds when heated. Prolonged consumption of burnt oils led to atherosclerosis, inflammatory joint disease, and development of birth defects. The scientists also questioned global health authorities' recommendation that large amounts of polyunsaturated fats be incorporated into the human diet without concomitant measures to ensure the protection of these fatty acids against heat- and oxidative-degradation.

Palm oil contains more saturated fats than canola oil, corn oil, linseed oil, soybean oil, safflower oil, and sunflower oil. Therefore, palm oil can withstand the high heat of deep frying and is resistant to oxidation compared to highly unsaturated vegetable oils.[16] Since about 1900, palm oil has been increasingly incorporated into food by the global commercial food industry because it remains stable in deep frying or in baking at very high temperatures 17, 18 and for its high levels of natural antioxidants.[19]

Know the smoke point(20)

Because of their chemical makeup, some oils are better suited for lower-heat cooking than others. This is important because heating oil above its smoke point — the tempera ture at which the oil begins to smoke — produces toxic fumes and harmful free radicals (the stuff we're trying to prevent in the first place). *A good rule of thumb: The more refined the oil, the higher its smoke point.*

High smoke point

Best suited for searing, browning and deep frying (although the latter is not a recommended practice where heart health is concerned).

Oil	% Mono	% Poly	% Sat	Nutrition Notes
Almond	65	28	7	Distinctive nutty flavor
Avocado	65	18	17	Sweet aroma
Hazelnut	82	11	7	Bold, strong flavor
Palm	38	10	52	High in saturated fat. Not recommended
Sunflower	79	7	14	Seek out high-oleic versions, which are higher in monounsaturated fat
"Light" olive/ refined olive	78	8	14	The more refined the olive oil, the better its all-purpose cooking use. "Light" refers to color

Medium-high smoke point

Best suited for baking, oven cooking or stir frying.

Oil	% Mono	% Poly	% Sat	Nutrition Notes
Canola	62	31	7	Contains low levels of omega-3
Grapeseed	17	73	10	High in omega-6
Macadamia nut	84	3	13	Bold flavor
Extra virgin olive	78	8	14	Best-pick oil
Peanut	48	34	18	Great for stir frying

Medium smoke point

Best suited for light sautéing, sauces and low-heat baking.

Oil	% Mono	% Poly	% Sat	Nutrition Notes
Corn	25	62	13	High in omega-6. High-oleic (monoun-saturated fat) versions coming soon
Hemp	15	75	10	Good source of ome-ga-3. Keep refrigerated
Pumpkinseed	32	53	15	Contains omega-3
Sesame	41	44	15	Rich, nutty flavor. Keep refrigerated
Soybean	25	60	15	High in omega-6
Walnut	24	67	9	Good source of omega-3
Coconut	6	2	92	High in saturated fat. Not recommended

No-heat oils*

Best used for dressings, dips or marinades.

Oil	% Mono	% Poly	% Sat	Nutrition Notes
Flaxseed	65	28	7	Excellent source of alpha-linolenic acid, a form of omega-3
Wheat Germ	65	18	17	Rich in omega-6. Keep refrigerated

* *Toasted sesame, extra virgin olive and walnut oils also work well.*

Portion control

A gram of fat provides 9 Kcalories. Hence portion control is important though you may choose the right fat and the right cooking method for that fat are. Too much of a good thing is also not healthy, so always make sure you include healthy unsaturated fats as a part of a diet rich in plant foods — fruits, vegetables, legumes, nuts, seeds and whole grains — and low in animal fats.

Fats at a glance

Remember the following strategies when it comes to cooking with fats:

Saturated fats – Bottom line: the fewer the better. Less than 7 percent of your daily calories should come from saturated fats. Eliminate whole and 2 percent dairy, and limit red meat and other animal protein at meals (reduce frequency, portion size or both).

Trans fats – Eliminate from your diet by avoiding foods that contain hydrogenated or partially hydrogenated oils. (Read the ingredient list!) Shortening and stick margarine contain trans fat.

Monounsaturated fats – Ramp up your intake of olives, avocados and nuts, and use olive and canola oils for most of your cooking and baking, respectively.

Polyunsaturated fats – You're may already be getting enough omega-6, so focus on increasing your intake of omega-3 foods such as salmon and walnuts.

Summary

The appropriate amount and type of fat as a component of daily food consumption is the topic of some controversy. No single oil has the optimum combination of the desired fatty acids. The best way is to use a variety of oils for different cooking preparation e. g, olive oil for salads and light sautéing; canola, sesame, rice bran or groundnut oil for high temperature cooking and sesame and mustard oil for other preparations. Let us look back and see what our ancestors used –a small quantity of traditional oils.-ghee, coconut oil, and organic home made butter. Topping up on EFA's is best done through dietary measures such as nuts, seeds, soyabean (tofu), flaxseed, canola oil, and fish.

Also blending oils as discussed above provides health benefit. Dr. Ghafoorunissa, renowned lipid biochemist presented details of oil combinations which can promote better n-3 and n-6 ratios.[21]

Emphasis should be placed on MUFA's and on omega-3 PUFA' s to replace both SFAs and omega-6 PUFA's. Though Omega 6 is definitely essential, but in limited quantities.

Reusing oil minimally is fine so long as the fat is not rancid and deteriorated to the point it produces undesirable flavors and odors (at this point the oil contains free radicals that are potentially carcinogenic). Each time the oil is re-used, the smoke point gets lowered.

Last but not the least, what ever good quality oil, the golden word is "limit"…….

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Obesity and Diabetes: Interrelationship

Manju Panda, Sujeet Jha***

Abstract

Diabetes epidemic can be attributed to the increasing incidence of obesity, especially in India. It is estimated that about 60-90% of all the patients with type 2 diabetes are obese (BMI ≥ 30kg/m2) or overweight (BMI ≥ 25kg/m^2). A number of mechanisms involved in the pathogenesis of obesity have been proposed which play an important role in the development of diabetes by causing insulin resistance or hypersecretion of insulin. Excessive storage of fat in obese people leads to the release of excessive fatty acids resulting in insulin resistance and hyperglycemia. Insulin resistance is also a consequence of elevated secretion of cytokines (TNF-α, IL-6, complement C3, MIF and leptin) by the adipose tissue resulting in the development of diabetes. There is a close association of diabetes and obesity with low plasma concentrations of adiponectin. However, further experimental studies are required to establish the role of adiponectin. Development of both the diseases can be prevented to a large extent by increasing physical activities and maintaining a healthy weight.

Keywords: Obesity, Type 2 diabetes, Adipokines, FFAs, Cytokines

Introduction

Diabetes is one of the major health problems affecting large number of individuals and has reached epidemic proportion worldwide. The World Health Organization (WHO) has projected that around 300 million people will suffer from diabetes by 2025.[1] Among the well-known risk factors of diabetes, a major risk factor is obesity (BMI ≥ 30kg/m^2),[2] whose prevalence is also rising at a higher rate in developing countries, including India.[3,4] Around 60-90% of all patients with type 2 diabetes are obese.[5,6] The increasing incidence of diabetes can thus be attributed to the global epidemic of obesity.[7] A number of clinical studies have reported an association between obesity and insulin resistance in adults as well as children[8,9,10] and reports are also present which suggest that weight loss is associated with a decrease in insulin concentration and an increase in insulin sensitivity in adults and adolescents.[11,12] This co-morbid condition of obesity in patients with type 2 diabetes is very common and is often termed as "Diabesity".[13,14] Sedentary lifestyle and changing food habits could be the main reasons for continuously increasing incidence of obesity and diabetes both in the urban as well as the rural areas in India. The rising prevalence of these two diseases is of concern as they may act as major risk factors for other fatal conditions like coronary artery disease (CAD).

Pathophysiology of Obesity and Type 2 diabetes

Pathogenesis of obesity appears to play a central role in the dysregulation of cellular mechanism that accounts for insulin resistance, which is the state of reduced responsiveness of liver, muscle and adipose tissue to insulin in type 2 diabetes. The stored fat is required for survival during nutritionally deprived states, however during state of prolonged abundance of food, excessive fat storage results in obesity.[15,16] This excessive storage of fat eventually leads to the release of elevated levels of fatty acids (FFAs) from enhanced lipolysis. The release of these FFAs then induces lipotoxicity, as lipids and their metabolites create oxidant stress in the endoplasmic reticulum and mito-chondria. This affects adipose as well as non-adipose tissues resulting in insulin-receptor dysfunction. The consequence is an insulin-resistant state which then creates hyperglycemia with compensated hepatic gluconeogenesis.[17,18] Insulin resistance is a key factor for type 2 diabetes. FFAs also decrease utilization of insulin-stimulated muscle glucose, contributing further to hyperglycemia. Lipotoxicity from excessive FFAs also decreases secretion of pancreatic β-cell insulin, which eventually results in β-cell exhaustion. *(Figure 1)*

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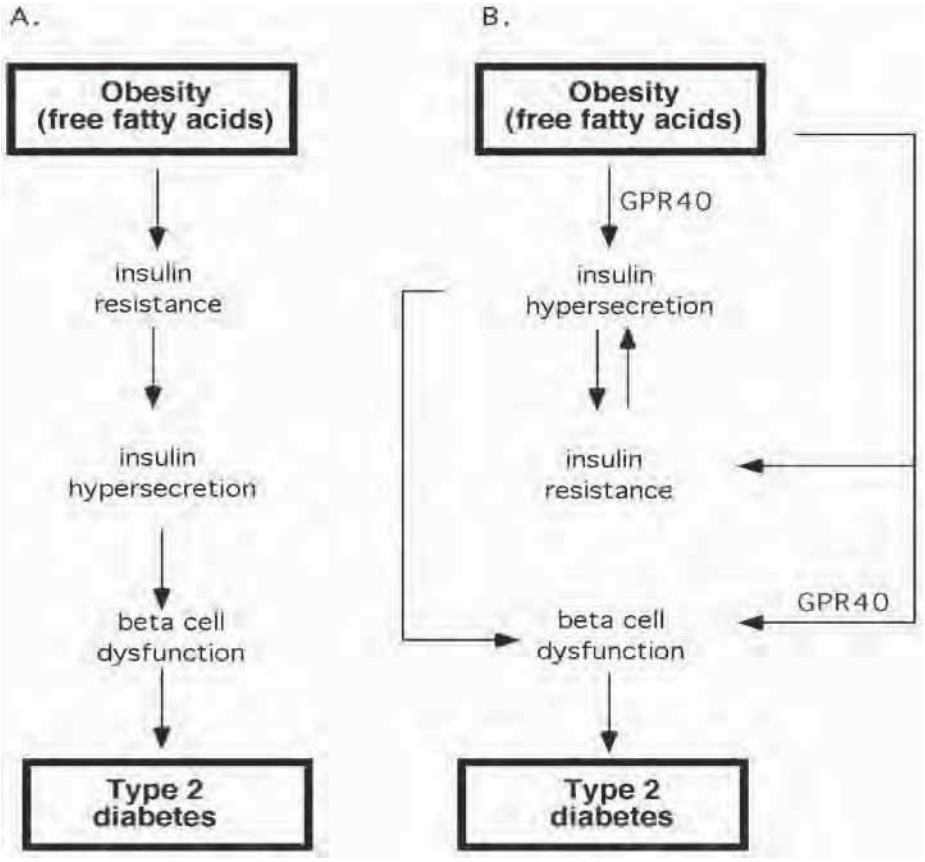

Figure 1: Pathway showing development of diabetes due to obesity.

Moreover, excess adipocytes release inflammatory adipokines (TNF-α, IL-6, complement C3, leptin and MIF), which, along with free fatty acids, provide the pathophysiologic basis for comorbid conditions associated with obesity such as insulin resistance and type 2 diabetes.[19] Along with fatty-acid lipotoxicity, visceral adipokines also contribute to the adipokine inflammatory injury that leads to pancreatic β-cell dysfunction, which, in turn, decreases insulin synthesis and secretion.[20,21] Adipocytes also stimulate fat-associated macrophages that also secrete monocyte chemoattractant protein 1 (MCP-1), macrophage migration inhibiting factor (MMIF), and resistin, all of which decrease insulin sensitivity (i.e. enhance insulin resistance) *(Figure 2)*.[22,23]

Obesity and Diabetes interlinked

A strong relationship between obesity and the onset of diabetes has been reported in a number of studies.[24-27] Research has shown that people carrying more weight particularly around the abdomen are more insulin resistant and find difficult to achieve good diabetes control.[10,28,29] A number of mechanisms have been proposed to link obesity and insulin resistance which includes increased production of adipokines or cytokines including tumor necrosis factor-α, resistin and retinol-binding protein 4.[30] Excess body fat and particularly visceral fat release increased amounts of FFAs in the blood. Elevation of FFAs levels directly affects insulin signaling and causes the liver and skeletal muscles to shift towards greater oxidation of FFAs for energy production and a relative inhibition of enzymes in the glycolytic cascades. As a result the capacity of liver and skeletal muscles cells to absorb and metabolize glucose decreases. Also, the tissues capacity to store glucose as glycogen decreases and the cells accumulate more triglycerides instead of glycogen.

Further, in Indians the body fat percentage is significantly higher than the western counterpart at similar BMI and blood glucose level. It has been hypothesized that excess body fat and low muscle mass may explain the high prevalence of hyperinsulinemia and the high risk of type-2 diabetes in Asian Indians.[31]. The risk of diabetes increases exponentially as BMI increases above about 25 kg/m^2.[32,33] In a large cross-sectional study in middle

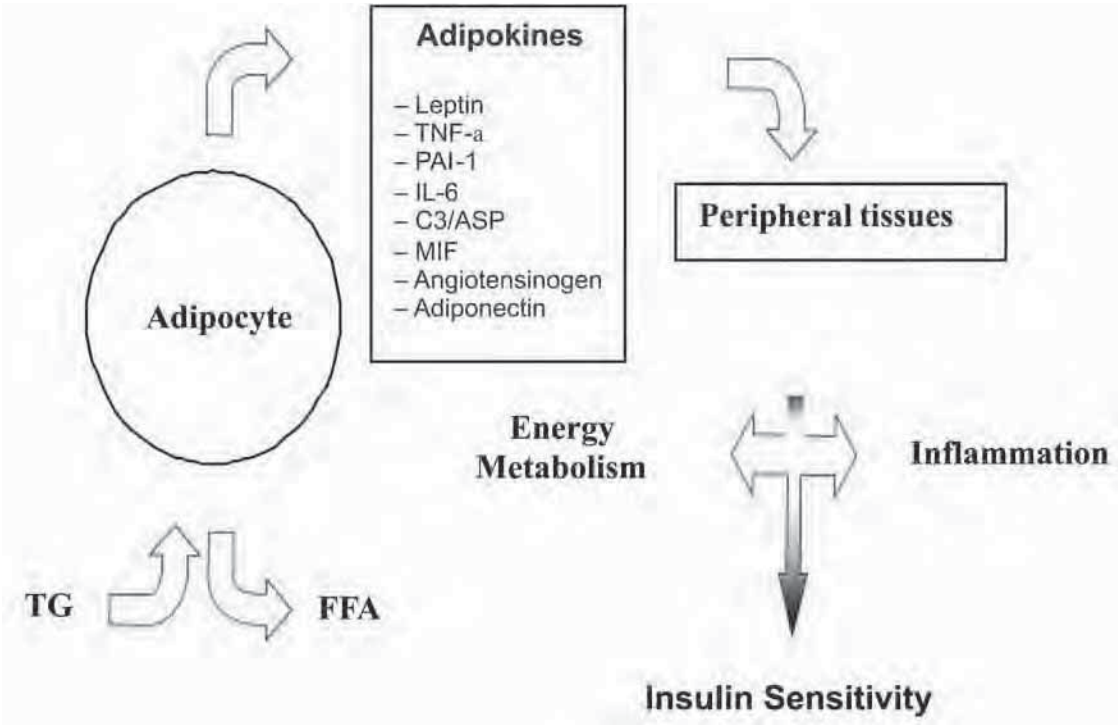

Figure 2: Adipokines regulating insulin sensitivity

aged Indians, a BMI > 23 was found to be associated with increased risk for type 2 diabetes.[34] Visceral fat increases the risk of diabetes by favouring insulin resistance. Patients with diabetes are usually advised to increase their physical activity and reduce weight. Prolonged duration of obesity also has deleterious effects on glucose homeostasis like increased resistance to glucose disposal and decreased secretion of insulin. Resistance to glucose disposal is strongly associated with obesity and results in high fasting and postload serum insulin concentrations. Prolonged duration of obesity could conceivably worsen this resistance.[35] Although excess fat in any region of the body is associated with increased risk of type 2 diabetes, it is generally held that an accumulation of abdominal fat ('central' obesity), as indicated by an increased waist: hip ratio is an independent risk for type 2 diabetes irrespective of the extent of obesity.[36,37] This is mainly attributed to increased intra-abdominal (visceral) adiposity. Excessive deposition of lipid in muscle and liver also enhances the risk of type 2 diabetes through mechanisms of intracellular lipotoxicity.

Genetic Factors Linking Obesity and Diabetes

Obesity as well as diabetes, both are the examples of multi-factorial diseases that arise through the interaction of multiple genetic and environmental factors. There has been evidences present which establish the genetic link between obesity and diabetes. Genome-wide association scans (GWAS) and candidate gene approaches have identified 40 genes associated with type 2 diabetes and a similar number, although largely different, with obesity. Most type 2 diabetes genes appear to be related to beta-cell dysfunction, with many fewer involved in pathways related to insulin resistance independent of obesity.[38-41] A growing understanding of genetics and cellular function of the beta-cell can identify potential mediators predisposing obese individuals to type 2 diabetes and further may provide insights for the development of new therapeutic agents. Although numerous diabetes and obesity associated genes have been identified, the known genes are estimated to predict only 15% of type 2 diabetes and 5% of obesity risk.[42,43] Recent genome-wide studies have shown multiple loci on chromosomes which affect the obesity-related phenotypes.[44] It can be speculated that the susceptibility to type 2 diabetes and obesity might also partly be due to shared genes. By comparing all of the published genome scans for type 2 diabetes and obesity, five overlapping chromosomal regions for both diseases have been identified and by analysing these five

susceptibility loci for type 2 diabetes and obesity, 27 functional candidate genes have been pinpointed that are involved in eating behaviour, metabolism and inflammation. These genes might reveal a molecular link between the two disorders.[45] By comparing the defined obesity-relevant pathways and Non-insulin dependent diabetes mellitus (NIDDM)-relevant pathways, it has been found that obesity-relevant pathways contains a gene set related to the insulin receptor, and coincidentally, there is a NIDDM-relevant gene set containing genes 2-fold up-regulated by insulin. Other than that, all relevant pathways in obesity and NIDDM are literally different.[46]

Co-relation of Adiponectin, Obesity and Diabetes

Adiponectin is a novel fat protein secreted by adipose tissue and abundantly present in the circulation in humans. It has been hypothesized that this protein has a role in the pathogenesis of obesity and type 2 diabetes and that lower plasma levels of adiponectin are predictive of type 2 diabetes and found in patients with diabetes as well as in obese people.[29,47-49] A review done by Hussain et al (2010) has reported that concentrations of adiponectin may be down regulated by weight gain.[19] Diabetes and obesity both are associated with low plasma adiponectin concentrations and hypoadiponectinemia in obese people is in large part attributable to insulin resistance.[50] A previous study in Japanese individuals has shown that the plasma adiponectin concentration is negatively correlated with body mass index (BMI) and therefore found to be lower in obese than in lean subjects,[51] which is also the case in Indian population.[52] However, the mechanism behind this close association between plasma concentration of adiponectin and insulin sensitivity is still unknown. Further experimental studies are required to study the molecular link between plasma levels of adiponectin and the pathogenesis of obesity and diabetes.

Conclusion

Obesity and diabetes which have become major health problems in India as well as globally are closely linked together, obesity serves as a major risk factor for type 2 diabetes and weight management can reduce the risk of diabetes to a larger extent. Urbanization, changing life style & food habits are the main reasons for increasing obesity in India and consequently responsible for increasing incidence of diabetes. Increasing physical exercise and maintaining ideal weight can lower the chances of developing diabetes. Further, reducing FFAs levels should be an important goal in the management of patients with type 2 diabetes mellitus. Preventing obesity (BMI ≥ 30kg/m2) could largely prevent diabetes. More large-scale clinical studies are required to understand the molecular mechanisms behind obesity causing insulin-resistance and the genetic relationship between both the diseases in order to develop better therapeutic remedies.

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ORGANIZATIONAL NEWS

2nd Annual Conference of Association of Diabetes Educators (ADE), 2013, Nagpur

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A Healthy Heart Diet

Niyati Likhite

"A healthy diet and lifestyle are your best weapons to fight cardiovascular disease. It's not as hard as you may think! Remember, it's the overall pattern of your choices that counts which serves as long term benefits to your health and heart"

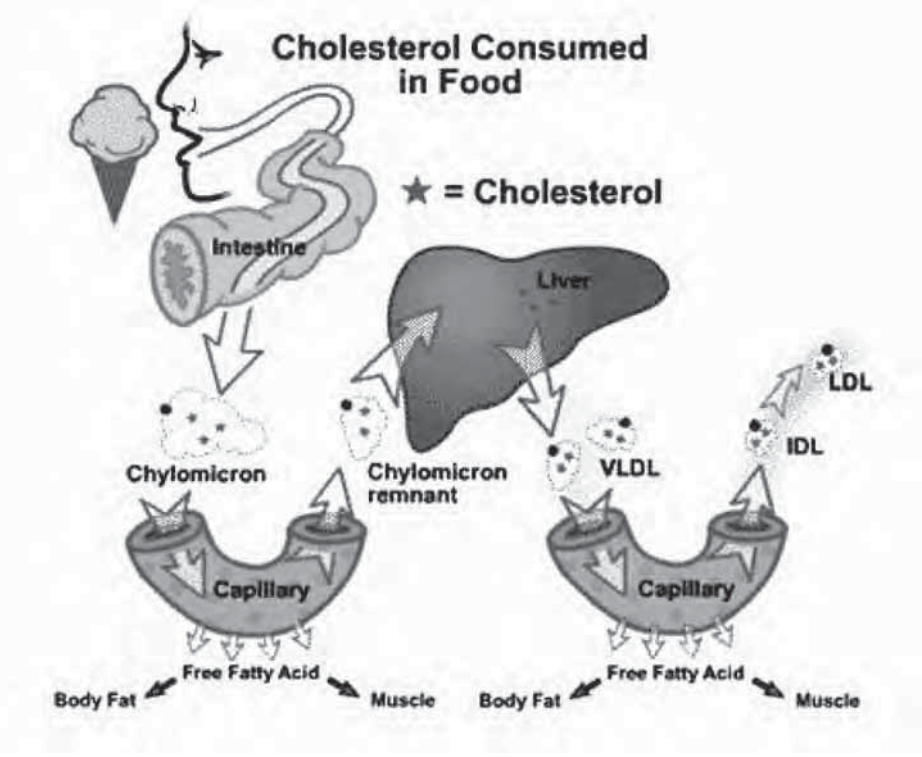
Heart disease is said to be one of the leading causes of death all over the world. It affects people of all ages, but is most frequent in middle age and is most often caused by atherosclerosis(hardening of arteries). It may affect he pericardium, myocardium or endocardium. The blood vessels within the heart, leaving the heart or the heart valves may be diseased. Coronary artery disease (CAD) is highly predictable, preventable and treatable. The prevalence of CAD in urban areas in India is four -fold higher than the overall US prevalence. Those in the high socio-economic group are the first ones to adopt an adverse lifestyle such as intake of high saturated fatty acid diet, sedentary lifestyle and cigarette smoking.

With the exponential increase in the number of people suffering from various heart- related problems, there is an increasing need among people to change their lifestyle and diet patterns. A combination of healthy eating and physical activity can help achieve and maintain an ideal body weight and also reduces the risk of the disease. A healthy diet can also increase your chances of survival after a heart attack by relieving pressure on your heart and circulation.

Role of fat in the development of Atherosclerosis:

Cholesterol and triglycerides are the main forms of fat carried in the blood stream. These fats or lipids come partly from food, partly from the body's own production in the liver. With the help of lipoprotein, digested fat from the liver is carried to various parts of the body by the blood vessels. The cholesterol returns to the liver and repeats its job. The liver places cholesterol into packages called lipoproteins. There are mainly four kinds of lipoprotein packages: Chylomicrons, VLDL (Very low density lipoprotein), LDL (Low density lipoprotein) and HDL (High density lipoprotein).

Chylomicron carries triglycerides. VLDL also transport triglycerides but mainly endogenous triglycerides formed in the liver. The VLDL travels through blood vessels to unload fat throughout the body. The empty VLDL becomes LDL. LDL is main carrier of cholesterol. Some LDL pieces get stuck to the blood vessel walls, narrowing the same. LDL is called bad cholesterol because it causes atherosclerosis. HDL plays a role in the reverse transport of cholesterol from tissues throughout the body back to the liver. It is called as good cholesterol. If too much of fat is



This article is reproduced from Diabetes today, Vol 21, No 1; Jan-Mar, 2011 with permission. Ms Niyati Likhite, Dietician, Diabetes Endocrine Nutrition Management and Research Centre (DENMARC), Mumbai, Email Id: **niyatichitre@gmail.com**

consumed the liver makes extra VLDL to carry the fat. More LDL pieces get stuck if not enough HDL is present to rescue them all. The blood vessel may become blocked. It this happens in heart, a heart attack may result which may be fatal.

Saturated and trans fatty acids are the most dangerous for the heart as they raise the levels of LDL and total cholesterol and lower the HDL levels, thus increasing the risk of heart disease.

Bioactive foods and Nutraceuticals for heart protection:

Foods and nutrients play a vital role in normal functioning of the body. They are helpful in maintaining the health of the individual and in reducing the risk of various diseases. Worldwide acceptance of this fact led to the recognition of a link between "nutrition" and "health" and the concept of "nutraceuticals" was evolved.

Bioactive foods are defined as foods or dietary supplement that demonstrate specific health or medical benefits including the prevention and treatment of disease beyond basic nutritional functions. Bioactive foods are fortified with nutrients to make them more usable within daily recommended allowances (RDA). These nutrients are rich in vitamins, minerals and nutraceuticals or any food or part of a food that provides health or disease prevention benefits with higher nutrition values.

Nutraceuticals are medicinal foods that play a role in maintaining well being, enhancing health, modulating immunity and thereby preventing as well as treating specific diseases. Nutraceuticals affect chronic disease and are known to have a favourable impact on cardiovascular diseases such as heart attack/ischemia, stroke, hypertension, deep vein clots and atherosclerosis. It can have an effect on cardiovascular disease in following ways:

- They tend to reduce circulating levels of LDL(bad) cholesterol which is achieved by modulating cholesterol production in liver, binding cholesterol within the intestines or by increasing the LDL receptors uptake in the liver.
- It reduces the possibility of oxidation by neutralizing radicals with antioxidants.

Now bioactive foods and nutraceuticals have emerged as potential supplements in cardiovascular and cancer preventive natural sources from food. Several nutraceuticals are available in market. The below table shows a sample of available nutraceuticals, their components and their potential human health benefits:

Sr. No.	Nutraceuticals	Components	Health benefits
1	***Carotenoids***		
	Beta-carotene	Carrots, various fruits	Neutralizes free radicals, which may damage cells; bolsters cellular antioxidant defenses
	Lycopene	Tomatoes and processed tomato products	Anti oxidant
2.	***Dietary Fiber***		
	Insoluble fiber	Wheat bran	May contribute to maintenance of a healthy digestive tract, decreases risk of colon cancer
3.	***Fatty Acids***		
	Mono unsaturated fatty acids	Tree nuts	May reduce risk of coronary heart disease
4.	***Flavonoids***		
	Flavonols	Onions, apples, tea, broccoli	Neutralize free radicals, which may damage cells; bolster cellular antioxidant defenses
5.	***Isothiocyanates***		
	Sulforaphane	Cauliflower, broccoli, cabbage, kale, horseradish	May enhance detoxification of undesirable compounds and bolster cellular antioxidant defenses
6.	***Phenols***		
	Caffeic acid, ferulic acid	Apples, pears, citrus fruits, some vegetables	May bolster cellular antioxidant defenses; may contribute to maintenance of vision and heart health

Sr. No.	Nutraceuticals	Components	Health benefits
7.	***Plant Stanols/Sterols***		
	Stanol/sterol esters	Fortified table spreads, stanol ester dietary supplements	May reduce risk of coronary heart disease by reducing LDL cholesterol level
8.	***Polyols***		
	Sugar alcohols (xylitol, sorbitol, mannitol, lactitol)	Some chewing gums and other food applications	May reduce risk of dental caries (cavities)
9.	***Prebiotics/Probiotics***		
	Lactobacilli, bifidobacteria	Yogurt, other dairy and nondairy applications	May improve gastrointestinal health and systemic immunity
10.	***Phytoestrogens***		
	Isoflavones (daidzein, genistein)	Soybeans and soy-based foods	May contribute to maintenance of bone health, healthy brain and immune functions; for women, maintenance of menopausal health
11.	***Soy Protein***		
	Soy protein	Soybeans and soy-based foods	May reduce risk of coronary heart disease
12.	***Sulfides/Thiols***		
	Dithiolthiones	Cruciferous vegetables	May contribute to maintenance of healthy immune function

Nutraceuticals that are found to be beneficial in cardiovascular disease are:

1) **Fatty Acids:** Fatty acids consist of chains of carbon atoms linked together by chemical bonds. The chemical bonds between carbon atoms can be either single or double bonds. Single bonds have more hydrogen molecules around them than double bonds. These chemical bonds determine whether a fatty acid is saturated or unsaturated. Fatty acids also come in different lengths: short chain fatty acids have less than 6 carbons, while long chain fatty acids have 12 or more carbons.

   Saturated Fats are the fats containing saturated fatty acids (single bonds). Foods high in saturated fats include lard, butter, whole milk, cream, eggs, red meat, chocolate, and solid shortenings. Excess intake of saturated fat can raise one's blood cholesterol and increase the risk of developing coronary artery disease.

   Monounsaturated fats and polyunsaturated fats are two types of unsaturated fatty acids. They are derived from vegetables, plants and animal sources. The American Heart Association recommends a diet that provides <10% of calories from Saturated fatty acids (SFA), up to 10% from Poly unsaturated fatty acids (PUFA), and as much as 15% from Mono unsaturated fatty acids (MUFA). The total dietary fat should be 30% of total calories.

   Monounsaturated fats are a healthy alternative to the trans fats and refined polyunsaturated fats found in most processed foods. It can be found in olives, olive oil, nuts, peanut oil, canola oil and avocados. Many studies have shown that, increased consumption of monounsaturated fats (for example eating more nuts) is beneficial in lowering LDL cholesterol (the "bad" cholesterol) while maintain HDL (good) cholesterol levels.

   Polyunsaturated fats are found in safflower, sesame, corn, cottonseed and soybean oils. This type of fat has also been shown to reduce levels of LDL cholesterol, but too much can also lower your HDL cholesterol.

Essential fatty acids:

Omega 3 fatty acids are essential poly unsaturated fatty acids, mainly found in fish. The two main types of omega 3 fatty acids are Docosa Hexanoic Acid (DHA) and Eicosa Pantenoic Acid (EPA) which are mostly found to be associated with triglyceride lowering properties, increasing HDL and thus protecting heart. They are necessary for human health but the body can't make them, you have to get them

through food. Several other studies also suggest that these fatty acids may help lower high blood pressure and may act as anticoagulant to prevent blood from clotting.

Sources of Omega 3 fatty acids

- All fish contain omega 3 fatty acids, but they are more concentrated in fatty fish such as mackerel, salmon, sardines and herring. The American Heart Association recommends eating fish at least 2 times a week.
- Other food sources of omega 3 fatty acid include:
    - o Soybeans and tofu
    - o Some nuts and seeds like walnuts, flax seeds, and pumpkin seeds
    - o Cooking oils such as flax seed oil, canola oil, and soybean oil
    - o Fish oil (American Heart Association recommend at least 1gm daily of DHA and EPA, preferably from dietary sources, but supplementation if required in coronary artery disease. For elevated triglycerides, 2-4 grams daily of DHA and EPA using fish oil supplements)

Omega 6 fatty acids are essential poly unsaturated fatty acids. Foods rich in omega-6 fatty acids include corn, safflower, sunflower, soybean, and cottonseed oil.

Omega-6 and omega-3 PUFA play a important role in heart and brain function and in normal growth and development.

2) Plant sterols and stanols:

Plant sterols are compounds naturally found in the membranes of plant cells. They are the plant versions of cholesterol (which occurs only in animals). These compounds can help lower cholesterol and reduce the risk of heart disease. Plant sterols and stanols are substances naturally found in fruits, vegetables, whole grains, legumes, nuts and seeds.

Although many plant-based foods contain small amounts of plant sterols, but since it would be difficult to get enough plant sterols to achieve any medicinal benefit through the diet, many products now contain added plant sterols. Such foods are known as “functional foods” and include margarines, cheese, orange juice, milk and bread. However these foods are not available in India. Some dietary supplements also contain plant sterols. In India, Benecol (which is a plant sterol) is available as the brand name Colred and can be used as a dietary supplement to lower LDL cholesterol levels.

The National Cholesterol Education Programme recommends that you take in 2 gms of plant sterols and stanols each day. However plant sterols and stanols alone won’t help you in lowering cholesterol levels, you need to make lifestyle changes that includes eating a healthy heart diet, exercising and smoking cessation.

3) Soy Protein:

Soy foods are a source of high-quality protein. Certain plant foods like flax seed and soy beans also contain omega 3 fatty acids which may help reduce the risk of coronary heart disease. Compared to other beans, soybeans have higher fat content but this fat contains heart healthy omega-3. Many studies have shown that soy protein may lower “bad” LDL cholesterol and triglycerides without lowering “good” HDL cholesterol. Researchers aren’t exactly sure how soy protein does this. It may be a combination of the effect of the protein and natural chemicals in soy called isoflavones. Hence, Consumption of soy protein provides health benefits that may help prevent or treat certain chronic diseases.

Many studies have proven the positive effect of soy foods on lowering cholesterol levels, including the harmful LDL cholesterol.The Food & Drug Administration recommends eating 25 grams of soy protein every day as part of a diet low in saturated fat and cholesterol.

Soy protein exerts several anti-atherogenic effects:

- It decreases LDL-cholesterol levels significantly.
- It tends to increase HDL-cholesterol levels, this is rather unique since most dietary interventions such as oat bran intake or decreased saturated fat intake significantly decrease HDL-cholesterol levels
- Soy isoflavones, plant chemicals unique to soybeans, have antioxidant properties which protect LDL from oxidation

How does diet help heart health?

Healthy diet can reduce your risk of heart disease by:

- Lowering blood pressure which is a major risk factor for heart disease
- Increasing good cholesterol (HDL) that transports fat away from arteries and back to liver for processing
- Reducing levels of bad cholesterol (LDL) that can form fatty deposits in the arteries and contribute to heart disease
- Preventing blood clots that can lead to heart attack and stroke
- Controlling blood sugars if you are diabetic.

Dietary Guidelines for Healthy Heart diet:

The goals of a heart healthy diet are to eat foods that help to maintain healthy levels of cholesterol and fatty molecules called lipids. A healthy heart diet is basically low in calories, high in fibre, low fat particularly low saturated fat,low cholesterol, high Mono unsaturated fatty acids(MUFA) and Poly unsaturated fatty acids(PUFA), normal protein, vitamins and minerals.

The American Heart Association(AHA) current dietary and lifestyle guidelines recommend:

- Balance calorie intake and physical activity to achieve or maintain a healthy body weight. (Controlling weight, quitting smoking, and exercising regularly are essential companions of any diet program. Try to get at least 30 minutes, and preferably 60 - 90 minutes, of daily exercise.)
- Eat a diet rich in a variety of vegetables and fruits. Vegetables and fruits that are deeply colored (such as spinach, carrots, peaches, and berries) are especially recommended as they have the highest micronutrient content.
- Choose whole-grain, high-fiber foods. These include fruits, vegetables, and legumes (beans). Good whole grain choices include whole wheat, oats/oatmeal, rye, barley, brown rice, millets, etc
- Eat fish, especially oily fish, at least twice a week (about 8 ounces/week). It is rich in the omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Consumption of these fatty acids is linked to reduced risk of sudden death and death from coronary artery disease.
- Get at least 5 - 10% of daily calories from omega-6 fatty acids, which are found in vegetable oils such as sunflower, safflower, corn, and soybean as well as nuts and seeds.
- Limit daily intake of saturated fat (found mostly in animal products) to less than 7% of total calories, trans fat (found in hydrogenated fats, commercially baked products, and many fast foods) to less than 1% of total calories, and cholesterol (found in eggs, dairy products, meat, poultry, fish, shellfish) to less than 300 mg per day. Choose lean meats and vegetable alternatives (such as soy). Select fat-free and low-fat dairy products. Grill, bake, or broil fish, meat, and skinless poultry.
- Use little or no salt in your foods. Reduce or avoid processed foods that are high in sodium (salt). Reducing salt can lower blood pressure and decrease the risk of heart disease and heart failure.
- Cut down on beverages and foods that contain added sugars (corn syrups, sucrose, glucose, fructose, maltrose, dextrose, concentrated fruit juice, honey).
- If you consume alcohol, do so in moderation. The AHA recommends limiting alcohol to no more than 2 drinks per day for men and 1 drink per day for women.
- People with existing heart disease should consider taking omega-3 fatty acid supplements (850 - 1,000 mg/day of EPA and DHA). For people with high triglyceride levels, higher doses (2 - 4 g/day) may be appropriate. The AHA recommends against taking antioxidant vitamin supplements (C, E, beta-carotene) or folic acid supplements for prevention of heart disease.

Suggested Further Reading:

1. http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyDietGoals/Healthy-Diet-Goals_UCM_310436_SubHomePage.jsp.
2. http://www.heartfoundation.org.au/healthy-eating/Pages/default.aspx.
3. Sharma R, Singh RB. Bioactive Foods and Nutraceutical Supplementation Criteria in Cardiovascular Protection. The Open Nutraceuticals Journal. 2010; 3: 141-153.

Religious Fasts and Diabetes

Priyanka Dhane

People fast for many reasons. Some people like to take a day off from eating after the holidays to give their digestive system a break. Some people fast to lose weight. Many people fast for religious reasons. Any fast that lasts for a long period of time is dangerous, whether you have diabetes or not. If a fast lasts more than a week, you run the risk of your body breaking down muscle to get the amino acids it so desperately needs. Long term fasting can also damage your heart, liver, and kidneys.

The Purpose of Fasting

Fasting is a body cleansing procedure during which food is restricted and only liquids are consumed. Strictly water fasts are the most brutal; herbal teas and juices made from fresh fruits and vegetables are consumed during a more liberal fast.

Juice fasting is the preferred method amongst many doctors and European fasting clinics, perhaps because it is less harsh than other treatment plans.

Today in the Western Hemisphere, many chronic health problems result from bad eating habits. There are a mix of people who are over-nourished, malnourished, or both. We eat chemically altered, high-fat toxic foods that do not provide a sufficient amount of essential vitamins and minerals to our bodies. Clogging of the eliminative systems with excess mucus is thought to sustain congestive diseases. Ineffective digestion and poor nourishment result in vitamin and mineral deficiencies. A well-balanced diet can overcome this health crisis. A diet of raw foods and fluids helps cleanse the body, and fasting takes the cleansing a step further.

What happens to your body during fasting?

The changes that occur in the body during fasting depend on the length of the continuous fast. Usually your body enters into a fasting state eight or so hours after the last meal. Your body will initially use stored sources of glucose and than later in the

fast it will break down body fat to use as the next source of energy. Using your body's fat stores as an energy source can in the long run help to reduce your cholesterol levels and blood pressure as well as your weight .Losing weight, particularly if you are overweight can also lead to better control of diabetes. However, fasting should not be used as a way of losing weight in the long term.

Diabetes and Fasting: Does Type Matter?

Whether you have type 1 or type 2 diabetes, fasting needs to be approached with care. "Fasting should be

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rare if you have diabetes because an individual with type 1 or type 2 on oral medication can experience hypoglycemia (low blood sugar). Risks from low blood sugar include seizure, coma, or even death if left untreated.

On the other hand, depending on the individual, fasting without using insulin can result in high blood sugars or in diabetic ketoacidosis (a serious diabetes complication caused by blood build-up of acids called ketones). Dehydration is another fear if fluids are avoided during the fast.

Therefore, in general a type 1 diabetic should almost never fast .While a type 2 diabetic can fast with some precautions.

Diabetes and Fasting: Does the Reason Make a Difference?

Religious reasons. Some people with diabetes may want to fast for religious observances such as Hindu, Jain fast, or Ramzan. Given the risky nature of fasting with diabetes, this isn't necessarily a good idea. Both [Hinduism and Islam] have guidelines that exempt those people who will be affected with harmful health consequences by fasting. If you're determined to fast, consult with your doctor or diabetes educator to put a plan in place at least a month or two before the actual fast. Medication dosage should be discussed, as well as how often to test your blood sugar and what to do if your blood sugar is too low or too high.

Also, keep in mind that the fasting itself may not be the only issue to plan for, timing of medication is also important. During religious fasts you may be able to eat a meal at various times (pre-dawn or sunset), which can affect the time you need to take insulin or oral medication to control your blood sugars,

A variety of religious fasts are undertaken everywhere in the world and more so in India.

Hindu fast – The common fast observed by Hindus is during the month of Shravan .This fast goes on for a month. During the day a non-cereal, non-pulse diet, primarily consisting of fruit and milk product is consumed. In the evening, a regular meal is eaten. The all day food intake is somewhat lower in calories but the high carbohydrate content of fruits and milk forestalls the possibility of hypoglycemia. Hence, no major alteration in the dose of insulin or oral agents is called for.

Muslim Fast – This is undertaken for a month during Ramzan period. There is no food or water intake during the day; At night there is one large meal after sunset and a small meal in the early hours of morning .In a study conducted by us it was found that the overall calorie intake was lower than usual with a mild weight loss at the end of the month .Usually, the control of diabetes is maintained during this fasting regime. We advise a larger insulin or oral hypoglycemic agent uptake in the evening and smaller dose in the morning.

Jain fast – Jain religion is marked by austerity and hence, there are a large variety of fasts undertaken by the followers of the religion. The total Jain fast consists of only water intake and can often extend up to 1-8 days, and at times up to a month. In

another variation of this basic theme, total fast and a 2 meal plan on alternate days is continued up to a year.

The fasting regimes in all religions allow a restricted diet during fast or soon after. This diet needs to be devised ingeniously, to conform to religious dictates and nutritional balance. Such meals can often be high in fat, calories and carbohydrates, and need to be discussed and planned.

Medical tests or procedures. Sometimes fasting is unavoidable, as in the case of medical or surgical procedures and certain lab tests. “This type of fast is short-term and poses much less risk than a prolonged fast such as Jain fast.”Your health care provider will give specific instructions based on the type of test and how your diabetes is managed. Also, try to schedule your appointment as early as possible in the morning to prevent a longer fast, and bring food with you to eat as soon as the procedure is complete and you are cleared to eat.

Diabetes and Fasting

Fasting can be a challenge for anyone, but it can be especially difficult for someone with diabetes. As every diabetic knows, successful blood sugar management relies on healthy meals eaten at regularly spaced intervals. So what happens when one or more meals need to be skipped for religious reasons or because of a medical or dental procedure? Each individual’s situation is different, so consultation with your physician is crucial.

Fasting with Diabetes: What every diabetic should know

Like most diabetics, you try to deal with your disease proactively. You have regular check-ups, take your medications as prescribed, exercise on a regular basis, and try to stick to a stringent diabetic diet. So what do you do if you want or need to keep fast? Since both healthy and diabetic people may have reasons to fast from time to time, it’s important for diabetics to understand what effects fasting can have on their overall health and well-being.

Once it is established that you will be fasting, and you are a diabetic patient, you should consult with your physician to determine if it is safe for you to fast and how to correctly go about it.

If you are fasting for religious reasons, you will most likely find that it is perfectly fine not to do so, especially if safety issues are involved. Pregnant women with gestational diabetes should not fast because it is unsafe to do so. Other reasons not to fast for religious purposes are if you have recently suffered a heart attack, you have a cold, the flu or some other infection or if you have a difficult time controlling your blood sugar under normal non-fasting conditions. Should your doctor give you the go ahead to fast for religious reasons, make sure he or she understands the nature and duration of your fast and that you understand what medications should or should not be taken during your fasting period. Your doctor will also tell you to check your blood sugar often, and instruct you as to what to do if your blood sugar gets too high or too low. Should your blood sugar begin dropping too low at any time during your fast, you should stop fasting immediately.

Once you have been cleared for fasting, the next big question that should be on your mind is:

How will fasting affect your diabetes medicine?

As a diabetic, you have done your homework, and you know the role that insulin plays in regulating blood sugar. You should also know that if you refrain from eating or drinking for prolonged periods, insulin will cause your blood sugar to get too low. The same rule applies for many diabetic medications that are taken at meal times. Make sure your prescriber goes over each of your meds and clearly notes which ones you should or should not take, either right before or during the duration of your fast. As fasting tends to change the rules of blood sugar

control, your doctor may also alter the dose of your diabetic drugs (the dose will usually be lower) as well as the time of day you should take them.

What things should you watch out for when you fast?

Here are some of the things you need to watch for when fasting. First and foremost, the blood sugar may get too low during a fast. Make sure you check your blood sugar often. As you may already know from experience, low blood sugar can make you feel nauseated, shaky, confused, cold and sweaty. You may also experience a rapid heartbeat that can be fairly scary. That's why before you fast you need to ask your prescriber two critical questions, the first being, what blood sugar number is too low for you? The second and most critical question is, what do you do if your blood sugar gets too low? As to the 1st question, that number can depend on a number of factors, but in general terms, blood glucose numbers between 70 and 80mg/dl are considered red flags for low blood sugar (hypoglycemia). Whenever you fast, always keep a good sugar source with you at all times, such as fruit juice, glucose tablets, hard candies, or crackers, just in case your blood sugar drops too low.

As strange as it may seem, abnormally high blood sugar during fasting can pose a real problem for other types of diabetics. For people with type 1 diabetes, this scenario can be extremely dangerous. If you have type 1 diabetes, rely on your prescriber to tell you what insulinadjustments to make while fasting. Although you still need to take some insulin, the dose will usually be lower.

Finally, it cannot be emphasized enough how important it is that you consult your physician if you have diabetes and plan to fast for any length of time. For those with or without diabetes, mounting evidence shows that fasting can be a cleansing and healthy experience for your body. The key for diabetics is to fast as safely as possible.

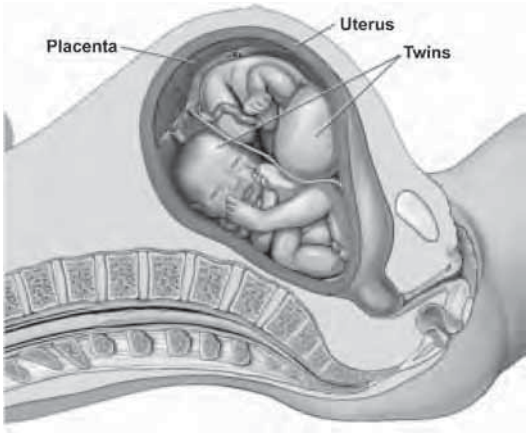
Diabetes and Pregnancy

Niyati Likhite

Diabetes mellitus is a disorder characterized by deficiency or insensitivity to insulin and exposure of organs to chronic hyperglycemia; this is a common medical complication of pregnancy. Diabetes in pregnancy falls into two main categories :

a) Pre-existing Diabetes: Diabetes that is diagnosed prior to pregnancy.

b) Gestational diabetes Mellitus (GDM): Diabetes that is discovered during pregnancy. Gestational diabetes is a result of hormonal changes that occurs during pregnancy. Increased levels of hormones from the placenta interfere with the ability of insulin to control glucose. It is known as insulin resistance. If the pancreas cannot produce enough insulin to overcome this effect of increased hormones, glucose levels will rise resulting in gestational diabetes. It resolves after delivery, but may recur in subsequent



pregnancies and may be associated with increased lifetime risk for developing type2 diabetes. Gestational diabetes does not directly produce diabetes in the baby. However if left untreated, it may cause the baby to produce too much of insulin and gain too much weight, increasing the risk of premature delivery.

Diabetes in pregnancy is associated with acute, as well as chronic maternal and fetal complications. Diabetics may have menstrual problems and difficulty in conceiving. It is also hazardous for the fetus as it increases the risk of fetal loss and major congenital malformations. However in today's world, advanced technologies are available that enable women with multiple diabetic complications to have successful pregnancies.

Screening and diagnosis for GDM:

The American Diabetes Association recommends (ADA,2010):

1) Women who are at high risk should be screened as early as possible after the pregnancy is confirmed.

Risk factors for GDM:

- Severe obesity
- Advanced maternal age at time of conception (>35 years of age),
- History of GDM or delivery of large for gestational age infant,
- Presence of glycosuria
- Diagnosis of polycystic ovary disease (PCOS)
- Strong family history of type 2 diabetes, glucose intolerance or pre- diabetes.

2) Women with high risk who are not found to have diabetes in early pregnancy and those with average risk are should be screened at 24-28 weeks of gestation.

3) Women with low risk do not require screening, that includes:
   - Age <25years
   - Weight normal before pregnancy
   - Member of ethnic group with low prevalence of diabetes
   - No known first degree relative with diabetes
   - No history of abnormal glucose tolerance
   - No history of adverse obstetrical outcome

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Ms Niyati Likhite, Dietician, Diabetes Endocrine Nutrition Management and Research Centre (DENMARC), Mumbai.
Email Id: **niyatichitre@gmail.com**

ADA earlier recommended 2 step procedures for screening:

All the women should be screened for gestational diabetes at their first medical visit after week 20 of pregnancy.

Two-step approach:

- Initial screen using 50g oral glucose load, blood glucose 1 hour later.
- A result of >140mg/dl identifies approximately 80% of women with GDM, a result of >130mg/dl identifies about 90%.
- Women who have >130 mg/dl after 2 hours, an 100 g OGTT or 75g OGTT is done on a separate day.
- A one-step screening procedure, utilizing 75gm of oral glucose load at any time of the day in fasting or fed state is now accented as standard procedure. A 2- hr value of >155mg/dl is indicative of gestational diabetes.

What is OGTT:

It is referred as Oral Glucose Tolerance Test, which measures the body's ability to metabolize glucose or clear the glucose from the blood. This test is used to diagnose diabetes, pre-diabetes and gestational diabetes. For GDM the test is performed at 24-28 weeks of gestation. Before conducting a test, patient should fast for at least 8-12 hours. In this, firstly the fasting blood glucose is drawn and after wards 75 g of glucose load is given to the patient. Then, blood is drawn at various intervals to measure the glucose levels, usually for 2 hours after the glucose load is given. All the readings are noted carefully and based on that the diagnosis is made.

Diagnosis criteria for GDM with 75g or 100 g of glucose load. Patient must exceed 2 of the values for diagnosis of GDM

	mg/dl	mmol/L
100g glucose load		
Fasting	95	5.3
1 hour	180	10.0
2 hour	155	8.6
3 hour	140	7.8
75g glucose load		
Fasting	95	5.3
1 hour	180	10.0
2 hour	155	8.6

* ADA diagnosis and classification, 2010

* 75g glucose load is not as well validated as the 100g OGTT.

The ADA Standards of Medical Care for 2010 for GDM do not support glycosylated hemoglobin as a diagnostic test during pregnancy.

Complications of Gestational Diabetes/Pre existing diabetes:

a) Neonatal complications

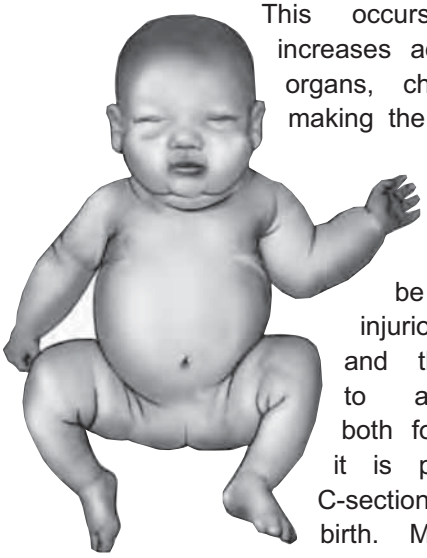
Hypoglycemia: Infants of diabetic mothers are at increased risk of hypoglycemia at 1 to 3 hours after birth. Hypoglycemia is defined as blood glucose level less than 30 mg/dl in any infant regardless of gestational age. If maternal plasma glucose is > 105 mg/dl during last four hours of labor in a diabetic mother, a higher incidence of neonatal hypoglycemia is likely.

Hypocalcemia: In some of the diabetic mothers, babies' serum calcium is < 7 mg/dl which is detectable during 2nd or 3rd day of birth. Respiratory distress and fetal metabolic acidosis may contribute to low levels of calcium in fetus. Hypomagnesemia may coexist and also needs to be treated.

Respiratory Distress Syndrome: It is a syndrome in premature infants caused by immaturity in the lungs. This syndrome is more frequent in infants of diabetic mothers and in the second-born of premature twins.

Hyperbilirubinemia: This is a common abnormality due to increased bilirubin production. It may also result due to immaturity of the liver.

Macrosomia (Large babies): It is a condition where the baby weighs more than 4 kg.



This occurs because insulin increases adipose tissue in the organs, chest and abdomen, making the trunk and shoulders disproportionately larger than the head. In this case, vaginal delivery can be more difficult and injurious for both the baby and the mother. Hence, to avoid complications both for mother and child, it is preferred to choose C-section rather than vaginal birth. Macrosomia can be

avoided by meticulous control of maternal blood glucose.

Obesity: Babies are at increased risk of obesity in late adolescence and early adulthood.

Glucose Intolerance: Babies are at risk of developing glucose intolerance or diabetes in late adolescence or early adulthood.

b) Maternal complications

Hypoglycemia: It may occur in the first trimester of the pregnancy. This is due to physiological adaptation, attempt for strict control of blood glucose and the nausea of early pregnancy.

Diabetic Ketoacidosis (DKA): As pregnancy has some features of starvation state, ketoacidosis is a real hazard. DKA has deleterious effect on the fetus.

Hypertension: This type of hypertension is known as Pregnancy Induced Hypertension (PIH).

Preeclampsia: is generally defined as PIH with protein loss in urine. Preeclampsia occurs three to four times more frequently in women with pregestational diabetes than in nondiabetic women.

Premature labour pain: Giving birth to a baby before 37 weeks of pregnancy.

Caesarean: Problems during pregnancy may also lead to caesarean.

Type 2 diabetes and gestational diabetes in subsequent pregnancies: Mothers are more likely to develop gestational diabetes in future pregnancies and are at higher risk of developing type 2 diabetes later in life. Those with previous history of GDM should be screened for diabetes 6-12 weeks postpartum for diabetes or pre-diabetes and at least every three years.

Management of Diabetes in Pregnancy:

It is very important for diabetic mothers to understand the disease and act accordingly.

- She should be aware about the implication of GDM for her baby and herself.
- Should follow the diet and exercise recommendations.
- It is very important to self monitor your blood glucose levels.
- One should know the self administration of insulin and adjustment of insulin doses.
- Should be able to identify and treat hypoglycemia.
- Should try to reduce stress, anxiety and cope up with denial.

Recommendations for management of Diabetes in Pregnancy:

- Diabetic mothers should receive nutritional counseling by dieticians on regular basis. As per the recommendations by the American Diabetes Association (ADA), Medical Nutrition Therapy (MNT) includes the adequate intake of calories and nutrients to meet the needs of pregnancy and should be consistent with the maternal blood glucose goals as recommended. Non-caloric sweeteners may be used in moderation. (eg SugarFree Natura and Splenda containing sucralose).
- If BMI is >30 kg/m2, 30- 33% of calorie restriction (25kcal/kg actual weight/ day) has been shown to reduce hyperglycemia and plasma triglycerides. Restriction of carbohydrates to 35-40% of calories has been shown to decrease maternal glucose levels and improve maternal and fetal outcomes.
- When MNT fails to maintain self monitored glucose at the following levels:

  Fasting Blood Glucose (FBG) – less than or equal to 105 mg/dl

  1 hour Post Prandial Glucose – less than or equal to 155 mg/dl

  2 hour Post Prandial Glucose – less than or equal to 130 mg/dl

  insulin should be started.
- Human insulin should be used when insulin is prescribed and Self Monitoring of Blood Glucose (SMBG) should be done for adjusting the doses and timing of the insulin. All types of human insulin as well as certain approved synthetic insulin have been shown to be effective in lowering blood glucose and are safe for both mother and fetus during pregnancy.

- Oral glucose lowering agents are not been recommended during pregnancy. However, studies on glibenclamide and glipizide have been encouraging. In one of the studies with GDM it was found that the glycemic control between those receiving insulin (88%) and glibenclamide (82%) was same. The frequency of macrosomia and fetal hypoglycemia were also similar between the 2 groups. The only significant difference involved maternal hypoglycemia, with insulin recipients having a much higher frequency (20% versus 2%).
- Metformin, an oral biguanide, may be an alternative to insulin for women with GDM, who are unable to cope up with the increasing insulin resistance of pregnancy. Metformin crosses the placenta, however there is no evidence of adverse fetal effect. One of the studies conducted in 2008, suggested that metformin is effective in controlling gestational diabetes and is not associated with a higher risk of maternal or neonatal complications as compared to insulin. It was observed that incidence of neonatal hypoglycemia was higher in insulin group than the metformin group. Another study was conducted to compare the use of metformin with that of insulin for treatment of GDM and type 2 diabetes unresponsive to diet therapy. It was found that glycemic control was better with metformin after 1 week of therapy and also throughout gestation.

  Hence, various studies have shown that metformin is clinically effective, cheap and a safe alternative to insulin therapy in pregnant diabetic women.
- Moderate physical exercise has been shown to lower maternal glucose concentrations in women with GDM. Exercise increases the insulin sensitivity of muscle cells and glucose uptake into muscle cells, resulting in lower blood glucose.

Dietary guidelines for Diabetes in Pregnancy:

A healthy diet is very essential for maternal and fetal health:

- Eat small and frequent meals through out the day at regular intervals. Do not skip meals and snacks.
- Eat less carbohydrate at the breakfast than at other meals because in the morning insulin resistance is greatest.

- Try to eat consistent amount of carbohydrate at each meal.
- If you have morning sickness, avoid fatty and fried foods.
- Consume high fibre foods like whole grains, pulses, vegetables and fruits.
- Avoid sugary and sweet foods
- Consume plenty of liquids (2- 2.5 litres/ day)
- Consume at least 3-4 servings of dairy products and calcium-rich foods like nuts and green leafy vegetables to ensure that you are getting recommended amount of calcium(1200mg/day) per day.
- Consume iron - rich foods like green leafy vegetables, seeds like gingelly, garden cress seeds, grains, meat and meat products to ensure 30mg of iron intake per day.
- Consume foods rich in Vitamin A, C and folic acid, to ensure the adequate intake of vitamins and minerals. Sources include dark colored vegetables, fruits, whole grains and legumes.
- Avoid intake of alcoholic beverages, caffeine (more than 300 mg/day).
- Eat variety of foods to get all nutrients required during pregnancy.
- Use artificial sweeteners instead of added sugars. Sucralose is considered safe during pregnancy

Postpartum care for the mother and baby:

The following steps needs to be taken into consideration for prevention of type 2 diabetes in women with previously diagnosed GDM:

1. Get tested for diabetes 6- 12 weeks after the delivery and later on every 2-3 years.
2. Breastfeeding improves glycemic control and should be encouraged in women who had gestational diabetes. Also it lowers the risk for developing type 2 diabetes.
3. Try to reach the pre pregnancy weight within 6-12 months after the delivery. Being overweight is a higher risk for developing type 2 diabetes.
4. Make healthy food choices. Consume healthy foods like vegetables, fruits, whole grains, pulses, low fat milk and milk products, nuts(almonds and walnuts), lean meat and meat products.
5. Keep yourself physically active. Exercise atleast 30-40 minutes a day.

Post partum intervention includes healthy eating, regular physical activity, weight reduction, breast feeding and regular blood sugar monitoring.

Nine months of pregnancy can be a challenge for diabetic women. Hence, they should be educated regarding lifestyle modifications like diet and exercise as it increases sensitivity to insulin and therefore brings down the blood glucose levels. Also self monitoring of the blood glucose should be initiated immediately after diagnosis is made. Ideally 4 times a day, blood glucose should be monitored. Monitoring helps in modifying the meal plan and adjusting the insulin doses. Medications that worsen insulin resistance (e.g., glucocorticoids, nicotinic acid) should be avoided if possible. Patients should be advised to seek medical attention if they develop symptoms of hyperglycemia or hypoglycemia. Education should also include the need for family planning to ensure optimal glycemic regulation from the start of any subsequent pregnancy. Low-dose estrogen-progesterone oral contraceptives may be used in women with prior histories of GDM, as long as no other medical contraindications exist. Offspring of women with GDM should be followed closely for the development of obesity and/or abnormalities of glucose tolerance.

ORGANISATIONAL NEWS

MEMBERSHIP OF ASSOCIATION OF DIABETES EDUCATORS (ADE)

Eligibility Criteria

Ordinary Membership

a) Graduates in Nutrition or Nursing (B. Sc and above), Pharmacist (B-pharm or above) with minimum 3 months of practical experience in Counseling people with diabetes; or 2 years of practical experience in any health care setting. Those applying within 5 years since basic graduation can be admitted as Members at a concessional rate, but they must fulfill other criteria.

b) Medical graduates recognized by Medical Council of India. Only those who have demonstrated abiding interest in diabetes education shall be admitted after due scrutiny by the Executive Council.

Documents to be submitted:

a) Application form duly filled and signed

b) Self attested photocopy of basic degree (Bachelors degree) certificate

c) Self attested photocopy of practical Experience certificate

d) Self attested photocopy of additional degree certificate

Submit your application with attachments (hard copy or soft copy) and cheque or demand draft by courier or through net banking or personally to Ms Sonal Modi at the following address:

103-104 Lady RatanTata Medical Center, M. Karve Road, Mumbai – 400 021

Tel: 91-22-2284 0244 Email id- **ademembers@gmail.com**

Life Membership fee:

Membership fee (Ordinary) – Rs.1000/-

Membership fee (within 5 years of basic graduation) - Rs. 500/-

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Association of Diabetes Educators (ADE)

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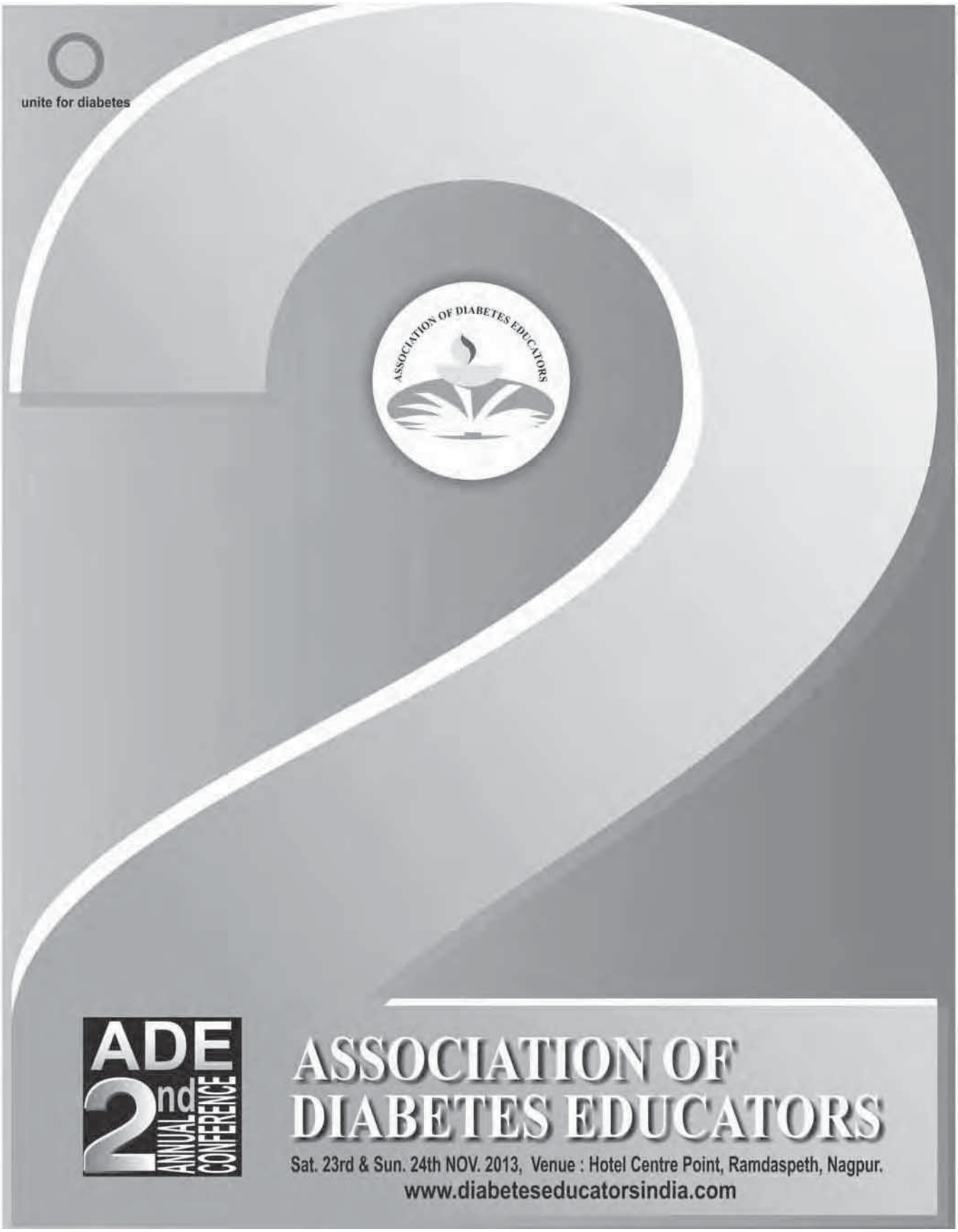
Signature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ADE
2nd
ANNUAL CONFERENCE
ASSOCIATION OF
DIABETES EDUCATORS
Sat. 23rd & Sun. 24th NOV. 2013, Venue : Hotel Centre Point, Ramdaspeth, Nagpur.
www.diabeteseducatorsindia.com

ASSOCIATION OF DIABETES EDUCATORS

2nd ANNUAL CONFERENCE, NAGPUR - Sat. 23rd & Sun. 24th Nov. 2013

ADE 2nd Annual Conference

Dear Colleagues,

On behalf of the organizing committee we wish to inform you that the 2nd Annual National Conference of Diabetes Educators (ADE 2013) will be held in Nagpur.

We are highly privileged and honoured in extending a warm and cordial Invitation to you to attend the conference to be held ***on Sat, 23rd Nov, & Sun, 24th Nov, 2013****, at Hotel Center Point, Ramdaspeth,* ***Nagpur.***

Today, Diabetes Education plays an important and pivotal role in management of diabetes. Keeping this fact in mind we have designed Scientific Sessions which include Basics of Clinical Diabetology, Recent Advances, Workshops, Practical Guidelines, International and National Recommendations, through 10 Scientific Sessions spread over two days, Our program includes Discussions, Role Plays, Live Diabetes education Program and "Hello Diabetes" for people with diabetes.

With a sole motto 'to empower the educators' an array of top brass national faculty will impart their knowledge of the subject. We assure you that you will enjoy the Scientific, Practical and Clinical content of the conference and additionally enjoy freshness and sweetness of 'Oranges' and 'Saoji tadka'.

We look forward to welcoming you to ADE-2013.

Dr. Sunil Gupta
Organising Chairman

Ms. Kavita Gupta
Organising Secretary

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TENTATIVE SCIENTIFIC PROGRAM

Saturday, 23rd Nov 2013

10.00 am : **Registration**

Session-I

11.00 am - 01.00 pm
- **Workshop**
  "Diabetes Counseling Strategies: Breaking Barriers"

Session-II

01.00 pm - 01.30 pm
- Diabetic Dessert Competition (For Registered Delegates Only)

01.30 pm - 02.15 pm : **Lunch**

Session-III

02.15 pm - 04.45 pm
- Diabetes Guidelines for 2013: glycemic targets
- MNT in DM during religious fasts
- Complimentary Medical systems - Is there a place in Diabetes?
- Diabetes Microangiopathy
  - * Diabetic Neuropathy- Foot care & Footwear Counseling
  - * Diabetic Nephropathy: Nephrotoxic drugs and diseases

04.45 pm - 05.15 pm : **Tea**

Session-IV

05.15 pm - 06.45 pm
- Insulin Therapy in emergencies
- Prevention of Hypoglycemia
- Non-weight bearing exercise program

Session-V

06.45 pm - 08.00 pm : Special Updates :
- Diabetes & Sex
- Artificial Sweeteners

Innauguration:

08.00 pm - 08.30 pm
- Inauguration

08.30 pm Onwards
- Dinner

COMPETITIONS
(For Registered Delegates Only)
NO ENTRY FEE

DIABETIC DESSERT
Last Date for registration : 15th Nov. 2013

SKIT / SONG / MIME
ON DIABETES
Solo / Group
Last Date for registration : 10th Nov. 2013

ESSAY WRITING
(English /Hindi / Marathi < 400 Words)
"Barriers in Diabetes Education - How to overcome"
Last Date of submission : 10th Nov. 2013

For More Details Contact :
Ms. Vinita
Mobile : 09765000554
Ph : +(91)712 2428111, 222, 333, 444
E-mail : ade2013nagpur@gmail.com

Sunday, 24th Nov 2013

Session-VI

09.00 am - 10.30 am
- Lesson from- Look Ahead Trial
- Intercurrent illnesses in DM
- Facts about Fat

Session-VII

10.30 am - 11.15 am : Special Updates :-
- CGMS & Insulin Pump
- Carbohydrate Counting

11.15 am - 11.30 am : **Tea**

Session-VIII

11.30 am - 01.30 pm
- Diabesity- Fit Fat Concept & Bariatric Surgery
- Glycemic load: Indian Context
- Diabetes and travel

1.30 pm - 02.30 pm : **Lunch**

Session-IX

02.30 pm - 03.30 pm
- Practical Learning : Skit / Songs / Mimicry

Session-X

03.30 pm - 05.30 pm
- Diabetes Education Program-"Hello Diabetes"

2nd Annual National Conference of Association of Diabetes Educators (ADE)

Secretariat : Sunil's Diabetes Care n' Research Centre Pvt. Ltd. 42, Lendra Park, Ramdaspeth, Nagpur - 440 010
Ph : +(91)712 2428111, 222, 333, 444. E-mail : ade2013nagpur@gmail.com www.dcrcindia.com
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ADE 2nd ANNUAL CONFERENCE
NAGPUR- 23 -24 NOV. 2013

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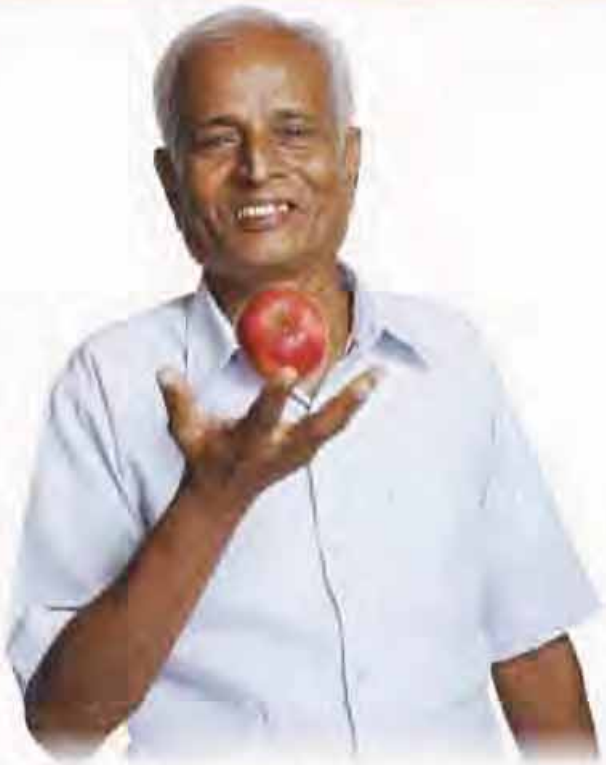

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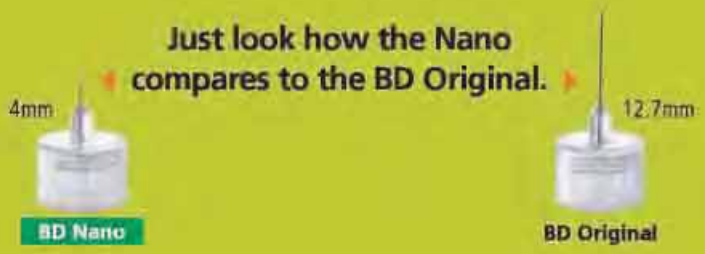

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1,3,5. Hirsch LJ, Gibney MA, Albanese J, et al. Comparative glycemic control, safety and patient ratings for a new 4 mm x 32G insulin pen needle in adults with diabetes. *Curr Med Res Opin.* 2010; 26 (6): 1531–1541.

2,6. Data on File.

4. Tested with adults of BMI 20-49.

7. As of April, 2010.

8. Gibney MA, Arce CH, Byron KJ, Hirsch LJ. Skin and subcutaneous adipose layer thickness in adults with diabetes at sites used for insulin injections: implications for needle length recommendations. *Curr Med Res Opin.* 2010; 26 (6): 1519–1530.

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• Based on AACE, ADA and NICE guidelines year 2012 which recommends DPP4 in monotherapy failures 1. Adapted from Gerich J. DPP-4 inhibitors: What may be the clinical differentiators? diabetes research and clinical practice 90 (2010) 131-140 2. Adapted from Vildagliptin add-on to metformin produces similar efficacy and reduced hypoglycaemic risk compared with glimepiride, with no weight gain: results from a 2-year study D. R. Matthews, S. Dejager, B. Ahren, V. Fonseca, E. Ferrannini, A. Couturier, J. E. Foley & B. Zinman, Diabetes, Obesity and Metabolism 12: 780-789, 2010 3. Adapted from Effects of vildagliptin twice daily vs. sitagliptin once daily on 24-hour acute glucose fluctuations: Raffaele Marfella, Michelangela Barbieri, Rodolfo Grella, Maria Rosaria Rizzo, Giovanni Francesco Nicoletti, Giuseppe Paolisso: Journal of Diabetes and Its Complications 24 (2010) 79-83 4. Adapted from Schweitzer, S. Damager, J. E. Foley, A. Couturier, M. Liguenos-Saytan & W. Kothny - cerebrovascular safety of vildagliptin: meta-analysis of adjudicated events from a large Phase III type 2 diabetes populatio: Diabetes, Obesity and Metabolism 12: 485-494, 2010 5. Adapted from Clinical experience with vildagliptin in the management of type 2 diabetes in a patient population ≥75 years: a pooled analysis from a database of clinical trials A. Schweizer, S. Dejager, J. E. Foley, Q. Shao & W. Kothny: Diabetes, Obesity and Metabolism 13: 55-64, 2011 6. Adapted from Poster Presentation # 840. Presented at: 47th Scientific Sessions of the European Association for the study of Diabetes; September 12-16, 2011; Lisbon, Portugal

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Basic Succinct Statement - GalvusMet®
Presentation: Vildagliptin/Metformin hydrochloride fixed combination: 50 mg/500 mg, 50 mg/850 mg, 50 mg/1,000 mg tablets. **Indications:** ♦GalvusMet® is indicated as an adjunct to diet and exercise to improve glycaemic control in patients with type 2 diabetes mellitus (T2DM) whose diabetes is not adequately controlled on metformin hydrochloride or vildagliptin alone or who are already treated with the combination of vildagliptin and metformin hydrochloride, as separate tablets. **Dosage:** ♦Do not exceed the maximum recommended daily dose of vildagliptin (100 mg). ♦Should be given with meals. ♦Starting dose for patients inadequately controlled on vildagliptin monotherapy: 50 mg/500mg twice daily and gradually titrated after assessing adequacy of therapeutic response. ♦Starting dose for patients inadequately controlled on metformin hydrochloride monotherapy: 50 mg/500mg, 50 mg/850 mg or 50 mg/1,000 mg once or twice daily. ♦ Starting dose for patients switching from combination therapy of vildagliptin plus metformin hydrochloride as separate tablets: 50 mg/500 mg, 50 mg/850 mg or 50 mg/1,000 mg. **Contraindications:** Known hypersensitivity to vildagliptin or metformin hydrochloride or to any of the excipients ♦renal disease or renal dysfunction ♦congestive heart failure ♦acute or chronic metabolic acidosis including diabetic ketoacidosis with or without coma ♦should be temporarily discontinued in patients undergoing radiologic studies involving intravascular administration of iodinated contrast materials. **Precautions/Warnings:** ♦Risk of lactic acidosis. ♦Monitoring of renal function. ♦Caution with concomitant use of medications that may affect renal function or metformin hydrochloride disposition. ♦Should be temporarily discontinued in patients undergoing radiologic studies involving intravascular administration of iodinated contrast materials. ♦Discontinue treatment in case of hypoxemia. ♦Temporary discontinuation in patients undergoing surgical procedure. ♦Excessive alcohol intake to be avoided. ♦Not recommended in patients with hepatic impairment including patients with a pre-treatment ALT or AST >2.5X the upper limit of normal. Liver function tests (LFT) to be performed prior to treatment initiation, at three-month intervals during the first year and periodically thereafter. Withdrawal of therapy with GalvusMet recommended if an increase in AST or ALT of 3X upper limit normal or greater persist. Following withdrawal of treatment with GalvusMet and LFT normalisation, treatment with GalvusMet should not be reinitiated. ♦Risk of decreased vitamin B12 serum levels. ♦Should not be used in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis. ♦Risk of hypoglycaemia. ♦May be temporarily withheld in case of loss of glycaemic control. ♦Should only be used in elderly patients with normal renal function. ♦Not recommended in paediatric patients. **Pregnancy:** Should not be used in pregnancy unless the potential benefit justifies the potential risk to the foetus. **Lactation:** Should not be used during breast-feeding. **Interactions:** ♦Interactions with Vildagliptin: low potential for drug interactions, no clinically relevant interactions with other oral antidiabetics (glibenclamide, pioglitazone, metformin), amlodipine, digoxin, ramipril, simvastatin, valsartan or warfarin were observed after co-administration with vildagliptin. ♦Interactions with metformin hydrochloride: furosemide, nifedipine, cationic drugs, drugs tending to produce hyperglycaemia, alcohol. **Adverse reactions:** ♦Vildagliptin: Rare cases of angioedema. Rare cases of hepatic dysfunction (including hepatitis). ♦Vildagliptin monotherapy - Common: dizziness – Uncommon: headache, constipation, oedema peripheral. ♦Metformin monotherapy – Very common: nausea, vomiting, diarrhoea, abdominal pain, loss of appetite. Common: metallic taste. Very rare: decrease of vitamin B12 absorption, lactic acidosis, liver function test abnormalities, hepatitis, skin reactions such as erythema, pruritus and urticaria. ♦Other effects with combination of Vildagliptin and Metformin - Common: headache, tremor, dizziness. ♦Post-marketing experience: Rare: hepatitis (reversible with drug discontinuation)- Uncommon: pancreatitis, urticaria. **Packs:** Box containing 6 strips of 10 tablets each. **Note:** Before prescribing, consult full prescribing information available from Novartis Healthcare Private Limited, Sandoz House, Dr. Annie Besant Road, Worli, Mumbai- 400 018, Tel: 022 2495 6888.
For the use only of a registered medical practitioner or a hospital or a laboratory only.
India BSS dtd 24 May 11 based on International BSS dtd 29 April 11

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