PRINT: ISSN 0970-9274 ONLINE: 2456-6608
J. Hum. Ecol., 21(2): 139-147 (2007)
DOI: 10.31901/24566608.2007/21.02.11

Dietary influence on TYPE 2 Diabetes (NIDDM)

Analava Mitra*, D. Bhattacharya and S. Roy***

** B.C. Roy Technology Hospital, **Chemical Engineering Department, Indian Institute of Technology, Kharagpur 721 302, West Bengal, India*
E-mail: amitra@adm.iitkgp.ernet.in

KEYWORDS Nature-Nurture Role. Triglyceride. Lipoprotein

ABSTRACT Incidence of diabetes particularly Non-insulin dependent diabetes mellitus is on the rise amongst the entire world population particularly in the residents belonging to younger age groups of developing countries located in Southeast Asia. The causes are many and predominated by nature-nurture role. Improper dietary intake of w-6 and w-3 fatty acids chasing the cholesterol myth of 70's may be an important reason of increased diabetes in India. Coronary risk in Indians is associated with an increased prevalence of Type 2 diabetes mellitus, rise in plasma or tissue levels of triglycerides and low-density lipoprotein (HDL) cholesterol. A review on available literature points out that proper diets and life-style patterns as advised reduce the incidence of diabetes and also check relentless progress of the disease.

INTRODUCTION

According to recent estimates the prevalence of diabetes mellitus is 4% worldwide and that indicates 143 million persons are affected which will increase to 300 million by the year 2025. Of this, developing countries share the disease load of 77%; India is second in the list after Indonesia. As per World Health Organization (WHO) report, in Southeast Asia by 2025 the estimated prevalence will be 79.5% in comparison to 32.7% in the year 2000. Between 1995 and 2025, the number of people with diabetes in India is projected to rise from 19 to 57 million, an increase of 195%, according to study by WHO (King, 1998).

Worldwide, a 122 % rise is projected, from the total of 135 to 300 million. This more than 2-fold global increase will occur because of population ageing and growth, as well as from obesity, unhealthy diets and a sedentary lifestyle. These latter factors are closely associated with urbanization and industrialization. In India currently about 50% of diabetics live in towns and cities, which are likely to see a 3-fold increase in the numbers in the next 30 years, due to migration. Table 1 shows prevalence of diabetes in different countries (US Census Bureau, Population Estimates, 2004: US Census Bureau, International Data Base, 2004)

Address for correspondence: Analava Mitra, B-145 IIT Campus, Indian Institute of Technology, Kharagpur 721302, West Bengal, India
Telephone: 091-0322-279970; *Fax:* 091-03222-282631
E-mail: amitra@adm.iitkgp.ernet.in

In the next 25 years, India will not only have the largest number of people with diabetes in the world but also the greatest increase in the disease burden. In India there appear to be more men than women with diabetes, but in the Westernized countries there are almost twice as many women than men with diabetes.

Another factor with enormous public health implications is the difference in age structure of the diabetic populations. It has been reported that diabetes occurs at a younger age in the developing countries. In the developed world, most people with diabetes are over 65 years of age while in developing countries the majority is in the age group 45-64, a trend that will accentuate. Figures 1-7 shows different statistics showing the spectrum of diabetes in India and some neighboring countries where life-style and socio-cultural patterns are similar to Indian perspective.

FACTORS RESPONSIBLE FOR INCREASED HEART DISEASES AND DIABETES MELLITUS

WHO Expert Committee opined that the undernutrition protects populations against diabetes and as undernutriton is common in India, incidence of diabetes should be less in Indians, but on the contrary, it is more. Indian lifestyle, habits, especially diet, might be invoked to explain the increased susceptibility to glucose intolerance based on Neel's thrifty genotype hypothesis. Since then attempts have been made to assess the role of diet to diabetes in population surveys. Diet may

Table 1: Prevalence of diabetes in different countries

Country/Region	*Extrapolated Prevalence*	*Population Estimated Used*
Type 2 diabetes in North America (Extrapolated Statistics)		
USA	17,273,847	293,655,405[1]
Canada	1,912,227	32,507,874[2]
Type 2 diabetes in Europe (Extrapolated Statistics)		
Austria	480,868	8,174,762[2]
Belgium	608,722	10,348,276[2]
Britain (United Kingdom)3,545,33560,270,708 for UK[2]		
Czech Republic	73,304	1,0246,178[2]
Denmark	318,434	5,413,392[2]
Finland	306,735	5,214,512[2]
France	3,554,365	60,424,213[2]
Greece	626,325	10,647,529[2]
Germany	4,848,506	82,424,609[2]
Iceland	17,292	293,966[2]
Hungary	590,139	10,032,375[2]
Liechtenstein	1,966	33,436[2]
Ireland	233,503	3,969,558[2]
Italy	3,415,145	58,057,477[2]
Luxembourg	27,217	462,690[2]
Monaco	1,898	32,270[2]
Netherlands (Holland)	959,894	16,318,199[2]
Poland	2,272,138	38,626,349[2]
Portugal	619,067	10,524,145[2]
Spain	2,369,457	40,280,780[2]
Sweden	528,611	8,986,400[2]
Switzerland	438,286	7,450,867[2]
United Kingdom	3,545,335	60,270,708[2]
Wales	171,647	2,918,000[2]
Type 2 diabetes in the Balkans (Extrapolated Statistics)		
Albania	208,518	3,544,808[2]
Bosnia and Herzegovina	23,976	407,608[2]
Croatia	264,521	4,496,869[2]
Macedonia	120,004	2,040,085[2]
Serbia and Montenegro	636,817	10,825,900[2]
Type 2 diabetes in Asia (Extrapolated Statistics)		
Bangladesh	8,314,145	141,340,476[2]
Bhutan	128,562	2,185,569[2]
China	76,402,799	1,298,847,624[2]
East Timor	59,955	1,019,252[2]
Hong Kong s.a.r.	403,242	6,855,125[2]
India	62,651,210	1,065,070,607[2]
Indonesia	14,026,643	238,452,952[2]
Japan	7,490,176	127,333,002[2]
Laos	356,948	6,068,117[2]
Macau s.a.r.	26,193	445,286[2]
Malaysia	1,383,675	23,522,482[2]
Mongolia	161,841	2,751,314[2]
Philippines	5,073,040	86,241,697[2]
Papua New Guinea	318,839	5,420,280[2]
Vietnam	4,862,517	82,662,800[2]
Singapore	256,111	4,353,893[2]
Pakistan	9,364,490	159,196,336[2]
North Korea	1,335,150	22,697,553[2]
South Korea	2,837,279	48,233,760[2]
Sri Lanka	1,170,892	19,905,165[2]
Taiwan	1,338,225	22,749,838[2]
Thailand	3,815,618	64,865,523[2]
Type 2 diabetes in Eastern Europe (Extrapolated Statistics)		
Azerbaijan	462,846	7,868,385[2]
Belarus	606,501	10,310,520[2]

Table 1: Contd. ...

Country/Region	*Extrapolated Prevalence*	*Population Estimated Used*
Bulgaria	442,233	7,517,973[2]
Estonia	78,921	1,341,664[2]
Georgia	276,111	4,693,892[2]
Kazakhstan	890,806	15,143,704[2]
Latvia	135,665	2,306,306[2]
Lithuania	212,229	3,607,899[2]
Romania	1,315,032	22,355,551[2]
Russia	8,469,062	143,974,059[2]
Slovakia	319,033	5,423,567[2]
Slovenia	118,321	2,011,473 [2]
Tajikistan	412,444	7,011,556 [2]
Ukraine	2,807,769	47,732,079[2]
Uzbekistan	1,553,553	26,410,416[2]
Type 2 diabetes in Australasia and Southern Pacific (Extrapolated Statistics)		
Australia	1,171,361	19,913,144[2]
New Zealand	234,930	3,993,817[2]
Type 2 diabetes in the Middle East (Extrapolated Statistics)		
Afghanistan	1,677,275	28,513,677[2]
Egypt	4,477,495	76,117,421[2]
Gaza strip	77,940	1,324,991[2]
Iran	3,970,776	67,503,205[2]
Iraq	1,492,628	25,374,691[2]
Israel	364,647	6,199,008[2]
Jordan	330,070	5,611,202[2]
Kuwait	132,796	2,257,549[2]
Lebanon	222,189	3,777,218[2]
Libya	331,269	5,631,585[2]
Saudi Arabia	1,517,408	25,795,938[2]
Syria	1,059,816	18,016,874[2]
Turkey	4,052,583	68,893,918[2]
United Arab Emirates	148,465	2,523,915[2]
West Bank	135,953	2,311,204[2]
Yemen	1,177,933	20,024,867[2]
Type 2 diabetes in South America (Extrapolated Statistics)		
Belize	16,055	272,945[2]
Brazil	10,829,476	184,101,109[2]
Chile	930,820	15,823,957[2]
Colombia	2,488,869	42,310,775[2]
Guatemala	840,035	14,280,596[2]
Mexico	6,174,093	104,959,594[2]
Nicaragua	315,279	5,359,759[2]
Paraguay	364,198	6,191,368[2]
Peru	1,620,253	27,544,305[2]
Puerto Rico	229,291	3,897,960[2]
Venezuela	1,471,610	25,017,387[2]
Type 2 diabetes in Africa (Extrapolated Statistics)		
Angola	645,797	10,978,552[2]
Botswana	96,425	1,639,231[2]
Central African Republic	220,145	3,742,482[2]
Chad	561,090	9,538,544[2]
Congo Brazzaville	176,355	2,998,040[2]
Congo kinshasa	3,430,413	58,317,030[2]
Ethiopia	4,196,268	71,336,571[2]
Ghana	1,221,001	20,757,032[2]
Kenya	1,940,124	32,982,109[2]
Liberia	199,449	3,390,635[2]
Niger	668,266	11,360,538[2]
Nigeria	1,044,138	12,5750,356[2]
Rwanda	484,627	8,238,673[2]

Table 1: Contd. ...

Country/Region	*Extrapolated Prevalence*	*Population Estimated Used*
Senegal	638,361	10,852,147[2]
Sierra leone	346,111	5,883,889[2]
Somalia	488,505	8,304,601[2]
Sudan	2,302,833	39,148,162[2]
South Africa	2,614,615	44,448,470[2]
Swaziland	68,778	1,169,241[2]
Tanzania	2,121,811	36,070,799[2]
Uganda	1,552,368	26,390,258[2]
Zambia	648,569	11,025,690[2]
Zimbabwe	215,991	12,671,860

1. US Census Bureau, Population Estimates, 2004
2. US Census, International Data Base, 2004.

contribute to the development of diabetes in two ways: quantitatively, by supplying calories and if activity is low by resultant obesity and qualitatively by the effects of specific foods. The long term effects of intermittent starvation and the pathological metabolic stress consequent to it on the course of glucose tolerance are not known yet, though some populations in India where incidence incidence of diabetes is on the higher side (tribal belts of Rajasthan and Gujrat), consume very little protein on some days and an alternate starvation-excess cycle exists (Ramachandran et al., 1990).

Interpopulation differences exist in both diet and the socio-cultural factors both within and outside the subcontinent. Available data suggest that diet is the main culprit. NIDDM prevalence rates are higher on the east coast of Andhra Pradesh, particularly Eluru (83) and Tenali, where rice is traditionally consumed compared to urban and rural Hyderabad, mainly wheat eating community. The dietary pattern, eating and methods of cooking vary in different parts of India (Ramaiya et al., 1990; Mitra and Bhattacharya, 2005).

However, with migration, the traditional dietary pattern changes rapidly with an increase in the consumption of "modern" foods coping with the west where changes in dietary adjustments has extended over many generations leading to genetic adjustments. Traditionally, Hindu Indians are pure vegetarians, but with modernization the diet has become more lactovovegetarian. In addition to the use of meat, fish, and poultry with moderate use of eggs, dairy products, and a relatively high intake of vegetables oils Hence the intake is of less saturated fat and cholesterol, and more polyunasaturated fat (Mitra and Bhattachrya, 2005). Some workers believe that that a vegetarian diet reduces the risk of developing diabetes but in a multicentre Indian study diabetes was more prevalent in vegetarians (2.1-2.8%) than in non-vegetarians (<2%). In Tanzania, Hindu Indians, majority of who are vegetarians also had a higher prevalence of diabetes than Muslim Indians who are non-vegetarians. Asian Indians living in America maintaining Indian dietary habits have higher insulin levels, higher plasma glucose levels and lower insulin binding to erythrocytes after a glucose load than Caucasoid Americans, suggesting an increased risk of developing NIDDM, though the risk reduces in 2nd generation of American Indians (Ramaiya et al., 1990). The contribution of diet, therefore, to the increased prevalence of diabetes in Asian Indians is still hazy and more nutritional studies are required to known the specific contribution of diet, independent of obesity to the pathogenesis of NIDDM.

One important cause of the sharp increase in the prevalence of heart disease and diabetes mellitus (DM) in India and other neighboring countries is lipid toxicity of unsaturated fats used as cooking medium according to Raheja (1999). It has been reported that unsaturated fats came into wide use following the cholesterol hypothesis in 1970 implicating saturated fats in *atherogenesis*. The result, ironically, has been an increase in the incidence of heart disease and diabetes in the country.

Linoleic acid, the precursor of w-6 poly-unsaturated fatty acid, promotes oxidation of low-density lipoprotein (LDL) cholesterol and atherogenesis. In contrast, w-3 poly-unsaturated fatty acids reduce the susceptibility of LDL to oxidation. Both w-6 and w-3 are essential fats, obtained only from the diet.

The metabolites of w-6 and w-3 fats modulate a variety of pathophysiological mechanisms including insulin action and secretion, platelet and vascular reactivity, cell-mediated immune and inflammatory responses. While the metabolites of w-6 fats are pro-inflammatory and thrombogenic, those of w-3 fats are cardio protective and immunomodulatory. Consequently, diets high in w-6 fat but poor in w-3 fat (high w-6/w-3) ratio) have become a major risk factor for a variety of immune inflammatory discords which may potentiate the increase incidence of diabetes mellitus and coronary heart disease (CHD). Most high fat diets in today's urban society contain this type of fat intake pattern. One of the reasons for non-prevalence of CHD and DM at a remarkably low level for several centuries in India was mainly

due to the use of mostly ghee in cooking which contain negligible concentrations of w-6 and w-3 fats. The traditional lacto vegetarian diets containing invisible fat provided the full requirement of w-6 fats (Ghafoarunissa, 1996). Deficiency of w-3 fats was avoided by increased consumption of leafy vegetables, some condiments and small quantities of mustard seeds and oil as observed by Raheja (1999).

In chasing the phantom of cholesterol, traditional fats are condemned as highly saturated and atherogenic, and replaced them with refined vegetable oil rich in unsaturated fats but mostly with the absence of w-3 fats. These were promoted as cholesterol lowering, 'heart friendly' fats. Their use in the last 3 decades, instead of lowering the coronary risk, thus resulted in a variety of epidemics, including that of DM, CHD and some cancers.

Today the prevalence of CHD, DM, hypertension or dyslipidaemias in any community correlates with the intake of unsaturated fat in the diet and its w-6/w-3 ratio. Correction of the faulty fat intake pattern improves glycaemic control, corrects dyslipidaemias and prevents DM and CHD as stated by Raheja (1999).

The increased coronary risk in Indians is often not associated with an increase in total cholesterol. Rather, the risk is associated with an increased prevalence of Type 2 diabetes mellitus, rise in plasma or tissue levels of triglycerides and low-density lipoprotein and low high-density lipoprotein (HDL) cholesterol (Mitra and Bhattacharya, 2005). Unsaturated fats induce these risk factors for coronary heart disease and insulin resistance. As being consumed along with, trans-fatty acids (vanaspati) further increase small dense LDL cholesterols, which are regarded as atherogenic, diabetogenic and, obesity inducers.

It thus appears that none of the refined oils with elevated w-6/w-3 ratio that has replaced traditional fats in the Indian kitchen can be called heart friendly. There is considerable evidence to indicate that the present epidemics of DM, CHD and several other disorders in India and elsewhere are the result of the lipid toxicity of unsaturated fats associated with the high fat urban diet as mentioned by Raheja (1999).

ROLE OF FOOD COMPONENTS IN DEVELOPING NIDDM

Carbohydrates: Excessive consumption of carbohydrate will increase the blood cholesterol and triglyceride level. An ordinary person requires 160 to 240 gm of carbohydrates per day. Reducing the daily carbohydrate intake such as starches and excluding sugars such as sucrose, fructose and lactose control this endogenous synthesis of cholesterol.

A person on a low-fat, high-carbohydrate diet utilizes the fatty acid synthetic pathway extensively; this pathway requires citrate to leave the mitochondria to generate malonyl-CoA. The resultant high concentration of malonyl-CoA inhibits the activity of the enzyme that is the gateway to fatty acid oxidation. It is for this reason that extreme low-fat diets do not result in weight loss nearly as fast as one might expect. The burning of excess fat is actually inhibited by the high carbohydrate intake. In persons with low body fat and good lean mass, high carbohydrate intake spares the use of fatty acids and the medium chain fatty acids that are otherwise so quickly used for energy, may accumulate above "normal" in membranes.

One might speculate that a diet low in carbohydrate would be effective in reducing hyperglycaemia in Type 2 diabetic patients. This, however, is not true since low carbohydrate diets decrease proinsulin synthesis and insulin secretion and result in diminished peripheral responsiveness to insulin. A more effective approach is to provide relatively good quantities of complex carbohydrates that are digested and absorbed slowly so that the rate of glucose delivery into the extracellular space is not excessive and rapid, but rather modest and sustained, thereby giving endogenous insulin a diminished task and a longer time to effect glucose disposal (Kahn and Weir, 1998). The blood glucose response to various foods of similar carbohydrate content varies widely. These variations are attributed to the variable quantity and quality of fibre and related products contained in these foods. It was observed that incidence of diabetes increases with the rise in consumption of high fructose corn syrup. By examining the consumption of food macronutrients (fats, proteins and carbohydrates) consumed by the population from 1909 to 1997, researchers were able to correlate, with startling clarity, the rise of diabetes with the consumption of refined sugars and carbohydrates (www.Junkfoodwarning.org/002035,2005).

High fiber diets and guar gum have been used in NIDDM and diabetics in general to decrease

and delay carbohydrate digestion and absorption (*Per-Henrik Groop et al., 1993;* http: // www.bawarchi.com/health/carbohydrate.html). Difficulties with compliance and limited effectiveness have been major problems with these approaches (http: //www.britannica.com/ eb/article?tocId=26157).

Gums act as a useful therapeutic agent in diabetic gastropathy and enteropathy. Soluble fiber is almost fully fermented in the colon into short chain fatty acids that may inhibit liver cholesterol synthesis and increase the body clearance of low-density lipoprotein cholesterol (LDLC). Fiber supplementation of 20 g per day decreased cholesterol Levels (Macrae et al., 1993).

Fats and Oils: Dietary fat is important to ensure the optimum quantity and quality of fat in the total diet. Fat should provide a minimum of 15% of total calories and its quality should be such that it furnishes adequate linoleic acid and linolenic acid (essential fatty acids). As the "essential" fatty acids are mostly supplied by vegetable based oils, a percentage of such oils will also have to be included in the diet.

High intakes of total fat and saturated fatty acids promote cholesterol synthesis, and raise serum very low-density lipoprotein (VLDL) and LDL cholesterol, which increase the risk of arteriosclerosis and thrombosis. Adequate intake of polyunsaturated fatty acids (PUFA) lowers blood cholesterol.

In Indian diet the invisible or fat rich in PUFA is provided from cereals, millets, pulses, spices and vegetables. People in the middle age having one or more risk factors for cardiovascular disease should restrict fat intake to the minimum level of 20 gm / day. In general vegetable fats are better than animal fat with exception of fish fat. Likewise among vegetable sources, coconut oil and hydrogenated oils promote cholesterol synthesis and thereby raise serum cholesterol.

Various types of hydrogenated oils (vanaspati ghee) also have very high percentage of saturated fatty acids depending upon the base and the processing involved. Poly unsaturated fatty acids available in some other edible oils and fats are: Mustard oil 25%, Rice bran oil 35%, Vanaspati 6% (Chow, 1992). Nutritionists are, however, agreed that not more than 30% of the total calories consumed should be derived from fat and saturated fatty acids should not account for more than one forth thereof. A minimum of 50 percent of all fats consumed should be vegetable based to ensure the supply of sufficient "essential" fatty acids to the body. Consumption of large amounts of fat at one time raises cholesterol levels more than if the same quantity and quality of fat is taken in small dozes.

The cholesterol intake should be restricted to 300 mg/day. Foods rich in cholesterol, e.g., butter, egg, meat, organ meat and shellfish should be avoided or restricted.

Most experts now agree that the best oils for cooking are canola or mustard with low erucic acid and olive oil or sesame oil (http://www. canola-council.org/production/thetruth.html). These oils are composed chiefly of monounsaturated oil that is more resistant to the damaging effects of heat compared to highly polyunsaturated oils like corn, safflower and soy (Ornish, 1996).

Several plant-derived oils, as well as fish oils, are being used for medicinal purposes. Evening primrose, black currant and borage oils contain gamma-linolenic acid (GLA), and omega-6 fatty acid that eventually acts as a precursor to some favorable prostaglandin (Lands, 1986). Because GLA can be formed from linoleic acid, it is difficult to determine to what extend the effects are due to GLA vs. linoleic acid. Most sources of GLA are much richer in linoleic acid than GLA. For example, evening primrose contains only 9% GLA, but contains 72% linoleic acid (Chow, 92). In most instances, high linoleic acid containing oils, like safflower and soy oil may provide nearly as much benefit as commercially available GLA preparations, at a fraction of the cost. The only exceptions to this generalization may be in individuals with diabetes and people who cannot form GLA from linoleic acid. GLA supplementation in diabetics has been shown to improve nerve function and prevent diabetic nerve disease. However, rather than simply relying on common vegetable oils, it appears most individuals would be better off supplementing their diet with omega-3 oils particularly as salad oil. Although most of the research on omega-3 oils has featured fish oils rich in eicosapentaenoic acid (EPA), EPA is manufactured in the body from alpha-linolenic acid - the primary fatty acid in flax oil. Flax oil contains more than twice the amount of omega-3 oil compared to fish oil and is also a good source for linoleic acid as well. In addition, flax oil may offer other benefits over fish oil and GLA

products. Flax oil is also much less expensive than these products. Flax oil is very tasty when mixed in yogurt, or cheese. The sulphur in these foods is also needed to assimilate the oil, reports Murray (http: //www.barleans.com).

Vegetable fats are used for cooking by both the vegetarians and non-vegetarians and are a good source of saturated fat (SFA) and both monounsaturated (MUFA) and polyunsaturated fat of omega-6 series. Milk fat is other important source of SFA. Daily food either raw or cooked in oil meets the need of SFA and MUFA particularly when a mixture of mustard oil and ground nut oil or palm oil is used in cooking (Chow, 1992)., A blend of different oils is ideal as it provides adequate requirement of different fatty acids other than Omega -3 (Ng TKW, 1997). Researchers from Harvard School of Public Health examined the long-term relationship between different types of dietary fat and the risk of type 2 diabetes. More than 840,000 women aged 34 through 59 were involved in the study. All were free of diabetes, cancer, and cardiovascular disease at the start of the study. After 14 years, slightly more than 2,500 cases of type 2 diabetes were documented. The researchers concluded that trans-fatty acids were responsible for the increased incidence.

Adults with diabetes can drink alcohol in moderation provided their diabetes is well-controlled and they use proper precautions when drinking. The American Diabetes Association suggests that alcohol intake for adults with well-controlled diabetes should follow the same guidelines as those established for the general population by the United States Department of Agriculture (USDA): 'no more than two drinks per day for men and one drink daily for women. Adults over age 65 should talk with their doctor about the appropriate limits for daily alcohol consumption'. While taking alcohol it is better to know the calorie consumed, to eat fast acting carbohydrate snacks to prevent hypoglycaemia, be accompanied by a friend and to monitor blood glucose level (http: //diabetes.about.com/cs/alcoholdiabetes/a/alcohol_basics.htm). One drink is equivalent to: 1.5 oz of 80-proof distilled spirits (100 calories) or 12 oz of beer (150 calories) or 5 oz of wine (100 calories) [One drink equals 2 fat exchanges; beer is an additional 1 starch exchange.]

Protein: The role of protein in attenuating the progressive loss of renal function that accompanies diabetes is now recognized though there is limited evidence for this in people with diabetes without kidney disease.

People with diabetes have no less or more need for protein than the general public. The American Diabetes Association nutrition guidelines suggest eating between 15 and 20% of calories as protein. The Recommended Daily Allowance (RDA) for protein is 0.8 grams per kilogram of body weight or 12-15% of total daily calorie intake.

People with diabetes have a greater risk of heart disease earlier in life. One of the most important nutrition guidelines to follow is "eat less saturated fat." A quick way to do that is to cut down on animal protein foods — meats, whole milk dairy foods, and high-fat cheeses (such as cheddar, brie, or American). Some studies say that a lower amount of protein in diabetic diets may reduce the chance of getting kidney disease. Other studies say plant protein may prevent or slow down diabetic kidney disease (http: //www.dmc.org/health_info/topics/nutr3310.html).

In the case of people with nephropathy, a diet low in protein, with about 0.6 to 0.8 g of protein per kilogram of body weight, is associated with a better kidney function and decrease in the onset of end stage renal disease. The other interesting issue we should take into account is that a high protein diet can deplete our body from calcium, thus increasing the risk of osteoporosis.

Animal protein contains all the amino acids our body needs to function properly and is called complete protein. Vegetable protein does not provide all the amino acids we need in one food. However, the mixture of amino acids from cereal and grains (rice, oat, wheat, corn) plus the amino acids contained in starchy legumes (beans, soy beans, chickpeas, dry peas, lentils) provides our body with complete protein. That is why the traditional meals in most ancient cultures have been for centuries the mixture of grains and legumes, like beans and corn for Americans, soybeans and rice for Asians, or chickpeas and lentils plus wheat for the protein requirement (http: //diabetes.about.com/od/mealplanning/a/protein.htm).

MINERALS

It is not generally realised that basic mineral and trace elements found in food are just as

essential to health as protective foods, as are the vitamins. There seems to be relationship between manganese, zinc, molybdenum, selenium and chromium in the prevalence of heart disease and diabetes. Chromium is needed to maintain normal blood sugar levels and deficiency of zinc in the system may lead to baldness, loss of appetite and sexual dysfunction. Drinking water that contains calcium and magnesium or other trace elements may be beneficial to lower cholesterol and triglyceride levels.

Beverages: A high intake of coffee and cola will elevate serum cholesterol levels. Caffeine containing beverage should be restricted since it may aggravate irregular heartbeats. A cup of coffee provides 60 mg of caffeine and a cup of tea provide 30 mg of caffeine and daily intake of caffeine should not exceed 100 mg/day.

Antioxidants: Antioxidant prevent oxidized LDL to attract monocytes and to the further formation of artherosclerotic plaques. Dietary intake of antioxidants including flavanoids naturally present in vegetables and fruits and vitamin E is associated with a decline in coronary heart disease and diabetes.

PROBABLE REMEDY

NIDDM (Non-insulin dependent diabetes mellitus) is an alarming global concern in present day scenario considering changes in the pattern of human civilization, industrialization, urbanization, socio-cultural values, ethnic orientation, economic up gradation and various other parameters reflecting psychosomatic spectrum of human life.

Control Through Diet: The major portion of the fat in the diet comes form either refined vegetable oils as seen in the lacto vegetarian Indian diet or in combination with storage animal fats as seen in the western type of diet. All these fats are deficient in w-3 fats and have elevated w-6/w-3 ratios. To avoid this toxicity, it would be prudent to resort to the low fat dietary Pattern. Cooking fat may be limited to ½ kg/ month/person. The diet may be supplemented with a small intake of fish or fish oil or flax oil, which provides w-3 fat. This simple approach can save both of us and our children from the present day epidemics. Vegetables steamed or cooked with very little oil, would ideally be the remaining one fourth of diet.

Since, oil and water does not mix, various emulsifying strategies are adopted by the body, using cholesterol, lecithin, and bile, to form complexes with the lipids initially very low density lipoproteins (VLDL), then low density lipoprotein (LDL) and finally to high density lipoproteins (HDL). The relative quantity of the later in the blood is a fair measure of the efficiency with which fat transfer has occurred, and is thus a positive indicator for the prevention of fat deposition in the blood vessels reports (Brennar, 1981).

People on high fat diets usually eat less fresh vegetables and fruits and thus may lack in the protective factors contributed by these foods. They are at increased risk for colon cancer. To decrease cancer risks plenty of fresh, different colored, seasonal fruits and vegetables are to be consumed. Aim should be for at least 5 servings of vegetables and fruits per day. Plenty of high fiber foods e.g. whole grains, whole fruits and vegetables, can supplement a daily fiber intake of 20-30 grams. Fat intake should be adjusted to 30% or less of total energy consumed. Fast food should be avoided as far as practicable.

It is admitted that the enzymes of foods cooked at temperature higher than 50 ^{o}C ordinarily get destroyed. Under these circumstances, if one fourth of the intakes of food were cooked, temptation of glands would make available digestive chemicals in large quantities.

Malnutrition of protein particularly at growing ages leads to M.R.D.M (malnutrition related diabetes mellitus). The studies on clinic children in Uganda where classical kwashiorkor is the prevailing form of PEM, showed a beautiful parallelism between plasma concentration of albumin and insulin observe Lunn and Whitehead (1973). Milward (1970) suggested that protein is a more important stimulus than carbohydrate. Fasting plasma insulin levels are generally low in severe PEM, rising during recovery and peaking during the phase of rapid growth. The subnormal insulin response to a glucose load as mentioned by James and Coore (1970) or to stimulation with glucagons, as stated by Milner (1971) may persist for many months after recovery. There is impaired glucose tolerance in some studies and that may be alleviated by addition of chromium. The insulin resistance may be a further factor contributing to glucose intolerance. Possible causal factors are the raised concentration of cortisol, growth hormone, and free fatty acids and reduction in binding site affinity rather than their number. As

per Mitra and Bhattacharya (2005) the best indicator to prevent diabetes is to follow ADA guidelines regarding diet (Table 2).

Control through Weight Reduction and Physical Activity: Calorie requirements of a person depend upon frame, metabolic rate, age, climate and the type of work he does. Calorie requirement of a person doing hard manual labor could even be 50 % or 100 % more than that of a person with a white-collar job doing office work only. Brisk walking for about 45 minutes covering 5 km/day is ideal for a diabetic sedentary to reduce and to maintain optimum body weight state Snowdon and Humphreys (1993).

Table 2: Recommended dietary factors for the control of NIDDM (Kahn and Weir, 1998, based on ADA guidelines)

Dietary Factor	*Recommendations*
Calories :	Should be prescribed to achieve and maintain a desirable body weight.
Carbohydrate :	Ideally should comprise 55-60% of the calories, with the form and amount to be determined by individual eating patterns and blood glucose and lipid responses. Unrefined carbohydrates should be substituted for refined carbohydrates to the extent possible modest amounts of sugars may be acceptable as long as metabolic control and desirable body weight are maintained.
Protein :	The recommended dietary allowance of 0.85 g/kg body weight for adults is an appropriate guide for those with diabetes. Some reduction in protein intake from previously high consumption levels may help prevent or delay the onset of the renal complications of diabetes.
Fat/Cholesterol:	Should comprise £ 30% of total calories, and all components should be reduced proportionately. Replacement of saturated with polyunsaturated fat is desirable to reduce cardiovascular risk. Cholesterol should be restricted to £ 300 mg/day to reduce cardiovascular risk.
Alternative Sweeteners:	Both nutritive and non-nutritive sweeteners are acceptable in diabetes management.
Sodium:	Should be restricted to 1000 mg/10000 kcal, not to exceed 3000 mg/day, to minimize symptoms of hypertension. Severe sodium restriction may, however, be harmful for persons whose diabetes is poorly controlled and for those with postural hypotension (low blood pressure and consequent dizziness when first standing up) or fluid imbalance.
Alcohol:	Should be used in moderation and may need to be restricted entirely by persons with diabetes and insulin induced hypoglycemia, neuropathy, poor control of blood sugar blood lipids, or obesity.
Vitamins/minerals:	Should meet recommended levels for good health. Supplements are unnecessary for persons with diabetes except when caloric intake is exceptionally low or the variety of foods consumed is limited. Calcium supplements may be necessary under special circumstances.

In 1895, Bose reported diabetes is prevalent among the richer class in India, whose life-style is indolent in nature as per Indian custom. Exercise is probably a protective factor against the development of diabetes and experimental evidence supports the value of physical exercise. There is increased insulin sensitivity in peripheral tissues, especially in muscle (Kahn and Weir, 1998). Quantitative assessment of physical activity is difficult and existing methods are crude (Snowdon and Humphreys, 1993). The interaction of diet, exercise, and obesity is complex, and it is not possible to isolate and study exercise as a single factor in relation to diabetes prevalence and incidence. In Indian Muslims in Tanzania, the increased prevalence of NIDDM was attributed to physical inactivity and genetic factor. Rural-rural migrants within south India had higher prevalence for diabetes than did indigenous populations, and obesity, sedentary activity, higher socio-economic status, and hypertension were more common in migrants than in the native population. Migration is usually accompanied by higher rates of diabetes. Recent studies in migrant Indians in Fiji (132) have shown an association of NIDDM with sedentary physical activity independent of obesity. The lack of differences in the high prevalences of diabetes in rural and urban Indians in Fiji, suggests that genetic factors were important than environmental factors in this ethnic group (Ramaiya et al., 1990).

The most powerful risk factor for NIDDM was obesity. Previous studies have shown both a strong and a weak relationship between obesity and NIDDM. The two most important aspects are extent and duration. In the western world, two-thirds or more of patients with diabetes are obese as compared with NIDDM subjects in India where obesity is not common. A possible reason is the confounding influence of weight loss with onset of disease or with therapy. Comparison between Indians in Malaysia and South Africa showed that the differences in diabetes prevalence in Indians abroad could not be explained on the basis of adiposity. The high prevalence of diabetes in the Fiji Indian population could not be explained on the basis of BMI. Inter-ethnic differences have to be accounted for. In South Africa, a study with

immigrant Indians showed that there was no correlation between BMI and fasting or 2 h plasma glucose levels over the entire range of BMI in Indians thereby negating the role of obesity as a risk factor for glucose tolerance. The effects on glucose tolerance became significant only after a certain threshold of BMI, i.e. 25 or 27. In rural-urban comparisons of diabetes prevalence also, the high prevalence rates in the urban population cannot be explained on the basis of obesity. Ramachandran et al. (1990) found that only a quarter of the diabetes were obese in an urban quarter of the south India. Studies of migrant Indians in South Africa, Mauritius and Tanzania however have shown a positive association of obesity with diabetes, especially among females. Insulin insensitivity and hyperinsulism with associated impaired tolerance of glucose and triglycerides correlate primarily with fat cell size rather than with either fat cell number or total body fat. These tend to improve with caloric restriction and disappear once weight reduction corrects the hypertrophy. Intra-abdominal fat cells measured by waist-hip ratio have been implicated in the pathogenesis of NIDDM). The waist-hip ratio correlates with glucose intolerance. There is no evidence, however, those differences in diabetes prevalence between Indian subcontinentals and other racial groups. Identical twin studies suggest that the genetic component in NIDDM acts independently of obesity and therefore there may be additional powerful environmental factors in the urban lifestyle operating independently of obesity which contribute to a high degree of diabetes prevalence in urban dwellers and migrants (Ramaiya et al., 1990).

REFERENCES

Bose, R.K.C.: Diabetes mellitus and its prevention. *Indian Med Gaz,*. **30:** 135 (1895).

Brennar, R.R.: Nutritional and hormonal factors influencing saturation of essential fatty acids. *Prog. Lipids Res.,* **20:** 41 (1981).

Chow, Ching Kuang: *Fatty Acids in Foods and Their Health Implications.* Marcel Decker Inc. New York (1992).

Ghafoarunissa: Fats in Indian diets and their Nutritional and health Implications. *Lipids*, **31**: 287-291 (1996).

Hilary, King: WHO Projects 195% growth in the number of Diabetics in India by 2025. *Presidential Address. IX National Congress of Diabetes* held in Mumbai (1998).

James, W.P.T. and Coore, H.G.: Persistent Impairment of Insulin Secretion and Glucose Tolerance after Malnutrition. *American Journal of Clinical Nutrition,* **23:** 386-389 (1970).

Kahn, C.R. and Weir, G.C. (Eds.): *Joslin¢s Diabetes Mellitus.* Asian Edition. B.I. Waverly Pvt. Ltd. New Delhi (1998).

Lunn, P.G. and Whitehead, R.G.: Progressive Changes in Serum Cortisol, Insulin, and Growth Hormone Concentrations and Their Relationship to the Distorted Aminoacid Pattern During the Development of Kwashiorkor. *British Journal of Nutrition,* **41:** 73-84 (1973).

Lands, W.E.M.: *Fish and Human Health.* Academic Press INC. New York (1986).

Macrae, R., Robinson, R.K. and Sadler, M.J.: *Encyclopedia of Food Science, Food Technology and Nutrition.* 2nd volume. Academic Press, London. pp. 925-945, 1362-1387 (1993).

Michael T. Murray, N. D. http: //www.barleans.com.

Mitra, A and Bhattacharya, D.: Effects of overall consumption, dietary patterns,cooking, on patients suffering from Non insulin dependent diabetes mellitus. *J of Interacad*., **9 (4):** 635-642 (2005).

Milner, R.D.G.: Metabolic and hormonal responses to glucose and glucagons in patients with infantile malnutrition. *Pediatric Research,* **5:** 33-39 (1971).

Milward, D.J.: The hormonal control of protein turnover. *Clinical Nutrition*, **9:** 115-126 (1970).

Ng, T.K.W.: Dietary fat and fibre intakes of Malaysian adults: issues and implications when 'western targets' are set as dietary goals. *Mal. J. Nutr.,* **3:** 137-147 (1997).

Ornish, Dean: *Program for Reversing Heart Disease*. Ivy Books. New York (1996).

Per-Henrik, G., Antti, A., Svante, S. and Lief, G.: Long-term effects of guar gum in subjects with non-insulin-dependent diabetes mellitus. *Am. J. Clin. Nutr,* **58:** 513-518 (1993).

Raheja, B. S.: Increase in heart disease blamed on unsaturated cooking fats. *Medical Times,* **29(1):** 2 (1999).

Ramachandran, A., Snehalatha, C., Kapur, A., Vijay, V., Mohan, V., Das, A. K., Rao, P. V., Yajnik, C. S., Prasanna Kumar, K. M. and Nair, Jyotsna D.: High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. *Diabetes Metabolism Review,* **6:** 125-146 (1990).

Ramaiya, K.L., Kodali, V.R. and Alberti, K.G.: Epidemiology of diabetes in Asians of the Indian subcontinent. *Diabetes Metab. Rev.,* **6:** 125–46 (1990).

Snowdon, L. and Humphreys, M. (Eds.): *Fitness Walking.* Orient Paperbacks, New Delhi (1993).

Warshaw, Hope S.**:** *Diabetes Meal Planning Made Easy*. ADA Publication. New York **(**2005).

www.Junkfoodwarning.org/002035,2005

http://www.bawarchi.com/health/carbohydrate.html

http://www.britannica.com/eb/article?tocId=26157

http://www.canola-council.org/production/thetruth.html

http://www.barleans.com)

http://diabetes.about.com/cs/alcoholdiabetes/a/alcohol_basics.htm

http://www.dmc.org/health_info/topics/nutr3310.html

http://diabetes.about.com/od/mealplanning/a/protein.htm