Review of Literature
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Nutrition Transition

India, like other countries in the world is also in a phase of constant demographic and nutrition transition. Since the 1980s there has been remarkable economic growth, incomes have been rising steadily and per capita income has increased across all groups. Consistent with high rates of growth, the proportion of per capita expenditure has increased. Economic growth has also been accompanied by rising urbanization. The degree of urbanization has risen from a mere 17.3% in 1950 to 28% in 2000 and is projected to be as high as 41% (Census of India 2001).

Increased urbanization has seen the rise of middle class and it is predominantly lifestyle preferences of this group that mark a change with the past. Economic growth alters the structure of labour force in urban areas characterized by increased female participation with important consequences on the family diet. Increased economic growth not only brings about divergences in dietary practices between different socio economic groups but also along all the age groups. The process of nutrition transition or diet transformation in India has evolved in two separate stages;

(i) **Income induced diet diversification**: The first phase that occurs with rapid economic growth where, diets diversify but predominantly maintain traditional features.

Economic prosperity enabled the consumers to afford a more varied, balanced diet and demand nutritionally superior food products. In this stage, the demand of food was directed towards traditional foods with positive income elasticities of demand as opposed to foods that display negative elasticities. Consumers typically moved away from rice consumption or consumed higher quality varieties of rice. Increased wheat consumption in the form of bread and other wheat based products such as cakes, cookies, etc. was also observed.
Diet Globalization: As globalization begins to exert its influence, a marked adoption of different diets is observed, diets that no longer conform to traditional local habits.

Globalization is defined as “the flow of information, goods, capital and people across political and economic boundaries”. As growth consolidates, the economy opens up to globalization, households start to adopt food consumption patterns that differ from traditional ones. The new dietary habits reflect global patterns, and are unlike the habits that had developed locally over many generations. Consumers exhibit a strong preference for meat or fish, temperate zone foods such as apples and highly processed convenience foods and drinks which are readily available in supermarkets and fast food outlets. A critical implication of globalization is the severing of the link between diets and local availability of resources and local habits. In this stage, consumers have access to varieties of food that were not previously available to them. Thus, consumers are no longer constrained in their demand to purchase local produce. Diet globalization is catalyzed by the media and internet; the products broaden their appeal by linking up with specific movies or personalities. Thus, they have a particularly huge appeal with the youth (Pingali and Khwaja 2004, Popkin et al 2001).

Economic prosperity has propelled India from a ‘state of famine’ to a ‘state of prosperity’ (Table 2, 3, 4, 5 & 6). These dietary shifts have drastically increased total dietary intake from 1986 Kcal/day in 1974 – 76 to 2403/day Kcal in 1997 – 99 and are projected to cross 2900 Kcal/day by 2030, if dietary practices are not modified (WHO 2002). Nutrition transition itself is marked by a shift from relatively monotonous diets of varying nutritional quality, based on the indigenous staple grain or root, locally grown legumes, vegetables and fruits, and limited food of animal origin (except among prosperous subpopulations), to what can be described as relatively industrialized diets, usually more varied, including more processed food, food of animal origin, increased sugar and fat consumption, more processed drinks and foods, and alcohol (Popkin et al 2001, Shetty 2002). Comparisons of food consumption patterns show a gradual reduction in cereal grain consumption from 1975 to 1995, which did not affect the average energy intake (Table 2).
Table 2: Food consumption patterns in India

<table>
<thead>
<tr>
<th>Product</th>
<th>Averages (cal/cap/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>79-81</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>------------</td>
</tr>
<tr>
<td>Grand Total</td>
<td>2083</td>
</tr>
<tr>
<td>Total Animal Product</td>
<td>120</td>
</tr>
<tr>
<td>Animal Fats</td>
<td>23</td>
</tr>
<tr>
<td>Aquatic Products, Other</td>
<td>0</td>
</tr>
<tr>
<td>Eggs</td>
<td>3</td>
</tr>
<tr>
<td>Fish, Seafood</td>
<td>5</td>
</tr>
<tr>
<td>Meat</td>
<td>16</td>
</tr>
<tr>
<td>Milk- Excluding Butter</td>
<td>71</td>
</tr>
<tr>
<td>Offal’s, Edible</td>
<td>2</td>
</tr>
<tr>
<td>Total Veg Product</td>
<td>1963</td>
</tr>
<tr>
<td>Alcoholic Beverages</td>
<td>5</td>
</tr>
<tr>
<td>Cereals</td>
<td>1368</td>
</tr>
<tr>
<td>Fruits- Excluding Wine</td>
<td>32</td>
</tr>
<tr>
<td>Oil Crops</td>
<td>25</td>
</tr>
<tr>
<td>Pulses</td>
<td>120</td>
</tr>
<tr>
<td>Rice (Milled Equivalent)</td>
<td>670</td>
</tr>
<tr>
<td>Spices</td>
<td>11</td>
</tr>
<tr>
<td>Starchy Roots</td>
<td>41</td>
</tr>
<tr>
<td>Stimulants</td>
<td>1</td>
</tr>
<tr>
<td>Sugar &amp; Sweeteners</td>
<td>193</td>
</tr>
<tr>
<td>Sugar Crops</td>
<td>8</td>
</tr>
<tr>
<td>Tree Nuts</td>
<td>3</td>
</tr>
<tr>
<td>Vegetable Oils</td>
<td>127</td>
</tr>
<tr>
<td>Vegetable</td>
<td>32</td>
</tr>
<tr>
<td>Wheat</td>
<td>390</td>
</tr>
</tbody>
</table>

Source: FAOSTAT, FAO
### Table 3: Global and regional per capita food consumption (kcal/capita/day)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>World</td>
<td>2358</td>
<td>2435</td>
<td>2655</td>
<td>2803</td>
<td>2940</td>
<td>3050</td>
</tr>
<tr>
<td>Developing countries</td>
<td>2054</td>
<td>2152</td>
<td>2450</td>
<td>2681</td>
<td>2850</td>
<td>2980</td>
</tr>
<tr>
<td>Near East and North Africa</td>
<td>2290</td>
<td>2591</td>
<td>2953</td>
<td>3006</td>
<td>3090</td>
<td>3170</td>
</tr>
<tr>
<td>Sub-Saharan Africa*</td>
<td>2058</td>
<td>2079</td>
<td>2057</td>
<td>2195</td>
<td>2360</td>
<td>2540</td>
</tr>
<tr>
<td>Latin America and the Caribbean</td>
<td>2393</td>
<td>2546</td>
<td>2689</td>
<td>2824</td>
<td>2980</td>
<td>3140</td>
</tr>
<tr>
<td>East Asia</td>
<td>1957</td>
<td>2105</td>
<td>2559</td>
<td>2921</td>
<td>3060</td>
<td>3190</td>
</tr>
<tr>
<td>South Asia</td>
<td>2017</td>
<td>1986</td>
<td>2205</td>
<td>2403</td>
<td>2700</td>
<td>2900</td>
</tr>
<tr>
<td>Industrialized countries</td>
<td>2947</td>
<td>3065</td>
<td>3206</td>
<td>3380</td>
<td>3440</td>
<td>3500</td>
</tr>
<tr>
<td>Transition countries</td>
<td>3222</td>
<td>3385</td>
<td>3379</td>
<td>2906</td>
<td>3060</td>
<td>3180</td>
</tr>
</tbody>
</table>

* Excludes South Africa.

### Table 4: Vegetable and animal sources of energy in the diet (kcal/capita/day)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T</td>
<td>V</td>
<td>A</td>
<td>T</td>
</tr>
<tr>
<td>Developing countries</td>
<td>2059</td>
<td>1898</td>
<td>161</td>
<td>2254</td>
</tr>
<tr>
<td>Transition countries</td>
<td>3287</td>
<td>2507</td>
<td>780</td>
<td>3400</td>
</tr>
<tr>
<td>Industrialized countries</td>
<td>3003</td>
<td>2132</td>
<td>871</td>
<td>3112</td>
</tr>
</tbody>
</table>

T - total kcal, V - kcal of vegetable origin, A kcal of animal origin (including fish products).

Source: FAOSTAT, 2003
### Table 5: Per capita consumption of livestock products

<table>
<thead>
<tr>
<th>Region</th>
<th>Meat (kg per year)</th>
<th>Milk (kg per year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>World</td>
<td>24.2</td>
<td>36.4</td>
</tr>
<tr>
<td>Developing countries</td>
<td>10.2</td>
<td>25.5</td>
</tr>
<tr>
<td>Near East and North Africa</td>
<td>11.9</td>
<td>21.2</td>
</tr>
<tr>
<td>Sub-Saharan Africa&lt;sup&gt;a&lt;/sup&gt;</td>
<td>9.9</td>
<td>9.4</td>
</tr>
<tr>
<td>Latin America and the Caribbean</td>
<td>31.7</td>
<td>53.8</td>
</tr>
<tr>
<td>East Asia</td>
<td>8.7</td>
<td>37.7</td>
</tr>
<tr>
<td>South Asia</td>
<td>3.9</td>
<td>5.3</td>
</tr>
<tr>
<td>Industrialized countries</td>
<td>61.5</td>
<td>88.2</td>
</tr>
<tr>
<td>Transition countries</td>
<td>42.5</td>
<td>46.2</td>
</tr>
</tbody>
</table>

<sup>a</sup> Sub-Saharan Africa includes Ethiopia.

### Table 6: Trends in the dietary supply of fat

<table>
<thead>
<tr>
<th>Region</th>
<th>Supply of fat (g per capita per day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>World</td>
<td>53 57 67 73 20</td>
</tr>
<tr>
<td>North Africa</td>
<td>44 58 65 64 20</td>
</tr>
<tr>
<td>Sub-Saharan Africa&lt;sup&gt;a&lt;/sup&gt;</td>
<td>41 43 41 45 4</td>
</tr>
<tr>
<td>North America</td>
<td>117 125 138 143 26</td>
</tr>
<tr>
<td>Latin America and the Caribbean</td>
<td>54 65 73 79 25</td>
</tr>
<tr>
<td>China</td>
<td>24 27 48 79 55</td>
</tr>
<tr>
<td>East and South-East Asia</td>
<td>28 32 44 52 24</td>
</tr>
<tr>
<td>South Asia</td>
<td>29 32 39 45 16</td>
</tr>
<tr>
<td>European Community</td>
<td>117 128 143 148 31</td>
</tr>
<tr>
<td>Eastern Europe</td>
<td>90 111 116 104 14</td>
</tr>
<tr>
<td>Oceania</td>
<td>102 102 113 113 11</td>
</tr>
</tbody>
</table>

<sup>a</sup> Sub-Saharan Africa includes Ethiopia.

**Source:** FAOSTAT, 2003
Typically, there are dramatic increases in overall fat intake, a corresponding reduction in the proportion of energy from starchy staple foods, accompanied by a shift from coarse grains and legumes towards more refined grains (mainly rice and wheat); greater intake of meat, fish, dairy products, and edible oils (Popkin et al 1993; Popkin et al 1998, Shetty 2002). Nutritional transition is also associated with a reduction in fruit and vegetable intake (Popkin et al 2001). However, these surveys revealed disparities between rural and urban populations and between different socioeconomic groups. Food balance data from the FAO shows that India has a fat calorie ratio of over 15%, a total fat intake of 37.8 g per day and 27.5% animal fat to total fat ratio (FAO 1994). Trends based on food balance sheet data show that the per capita supply of animal products has increased from 7.0 g in 1965 to 12.9 g in 1999, thus contributing almost twice the energy content (increased from 104 to 192 kcal/capita/day). It has been computed that 10–15% of the daily energy in the diet comes from the invisible fat component and this level is adequate to meet the essential fatty acid requirements for both linoleic acid and alpha linolenic acid.

Dietary fat intakes, based on household surveys suggest that the visible fat in poor rural diets is largely vegetable based with negligible animal fats. The differences in the dietary fat intake between rural and urban and between lower and higher socio-economic groups are largely due to large differences in the intakes of visible fats, except in the highest income group (Table 7) where much of it is from animal sources, with the invisible fat intake being similar among these groups (Ghafroonisa 1989). Computations also suggest that 25% of all available fat is consumed by the rural population, while 40% of all edible fat available in India is being consumed by 5% of the total population i.e. 20% of the urban population that constitutes the ‘urban-rich’ (Shetty 2002).

**Shifts in Physical Activity Pattern**

Physical activity has declined in the industrialized world as a result of increasing mechanization (Ferro and Martino 1996). Reduced moderate-to-vigorous physical activity by people of all ages and corresponding habitual inactivity accompany the nutritional transition (Popkin 1998).
Table 7: Dietary fat intake by urban and rural socio-economic group in India

<table>
<thead>
<tr>
<th>Income Group</th>
<th>Fat intake (g/day)</th>
<th></th>
<th></th>
<th>Fat as % of energy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Visible</td>
<td>Invisible</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td><strong>Urban</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>46</td>
<td>49.7</td>
<td>95.7</td>
<td>33.1</td>
</tr>
<tr>
<td>Middle</td>
<td>35</td>
<td>36.5</td>
<td>71.5</td>
<td>27.2</td>
</tr>
<tr>
<td>Low</td>
<td>22</td>
<td>29.9</td>
<td>51.9</td>
<td>21.0</td>
</tr>
<tr>
<td>Industrial Labour</td>
<td>23</td>
<td>30.0</td>
<td>53.0</td>
<td>21.3</td>
</tr>
<tr>
<td>Slum dweller</td>
<td>13</td>
<td>24.2</td>
<td>37.2</td>
<td>16.7</td>
</tr>
</tbody>
</table>
| **Rural**           |                    |           |           |                    *
| > 150               | 25                 | 27.4      | 53.4      | 18.5               |
| 90 – 150            | 17                 | 25.6      | 42.6      | 14.8               |
| 60 – 90             | 13                 | 22.8      | 35.8      | 13.3               |
| 30 – 60             | 9                  | 20.3      | 28.3      | 11.0               |
| < 30                | 5                  | 18.0      | 23.0      | 9.5                |
| Average             | 9                  | 25.6      | 34.6      | 13.7               |

* Rural income in rupees per month

Source: Computed from dietary intake data of the National Nutrition Monitoring Bureau, 1987.

Table 8: The proportion of economically able men and women (18-65 yrs) employed in each sector of work.

<table>
<thead>
<tr>
<th>Region</th>
<th>Percent Agriculture</th>
<th>Percent Industry</th>
<th>Percent Service</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High Income</td>
<td>7</td>
<td>4.2</td>
<td>24.3</td>
</tr>
<tr>
<td>Middle Income</td>
<td>64.1</td>
<td>57.2</td>
<td>50.9</td>
</tr>
<tr>
<td>Small Islands</td>
<td>80.5</td>
<td>75.7</td>
<td>70.8</td>
</tr>
<tr>
<td>Upper low income</td>
<td>79.9</td>
<td>73.5</td>
<td>69.0</td>
</tr>
<tr>
<td>Lower low Income</td>
<td>71.4</td>
<td>67.9</td>
<td>63.6</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High Income</td>
<td>9.7</td>
<td>4.7</td>
<td>31.5</td>
</tr>
<tr>
<td>Middle Income</td>
<td>49.7</td>
<td>42.3</td>
<td>36.5</td>
</tr>
<tr>
<td>Small Islands</td>
<td>92.7</td>
<td>91.2</td>
<td>78.8</td>
</tr>
<tr>
<td>Upper low income</td>
<td>84.5</td>
<td>80.1</td>
<td>75.0</td>
</tr>
<tr>
<td>Lower low Income</td>
<td>82.7</td>
<td>79.5</td>
<td>81.0</td>
</tr>
</tbody>
</table>
One of the most inexorable shifts with modernization and industrialization is the reduction of physical activity at work and home for both men and women. The only information from Asia available at national level is on the occupational distribution of men and women in each of our country groupings. As occupations shift from agriculture and manual labour to manufacturing and service sector, levels of energy expended naturally drop (Table 8).

Also, the decline in time dedicated to productive work has been accompanied by a reduction in energy spent at work resulting from increased mechanization of occupational work. Concurrent to this decrease in the energy expenditure in occupational activities, increased urbanization, universal use of motor cars, mechanization of most manual jobs outside the occupational sphere and increasing leisure time have aggravated this trend. Increased leisure time is most often dedicated to sedentary activities like television viewing, thus altering the structure of leisure time and encroaching on time normally allocated to other activities like playing or exercise. However, there is need to obtain data on levels of physical activity and patterns of activity in both rural and urban settings (Shetty 2002).

**Epidemiological Transition**

Epidemiological transition focuses on the complex changes in patterns of health, disease and mortality that result from these demographic and associated economic and sociological changes. These health and mortality transitions entail substitution of NCDs for infectious and communicable diseases as the primary causes of morbidity and mortality (Popkin *et al* 2001). Developing countries like India, currently lagging behind those having completed the epidemiological transition, will demonstrate a decline in infectious disease mortality and a rise in NCDs.

Further, evidence of an epidemiological transition is obvious in India with NCDs contributing increasingly to premature deaths in adults, particularly in the urban areas. A growing set of studies suggests that perinatal and infant nutrition insults affect
preposition to cardiovascular disease (CVD), obesity, hypertension, and adult-onset diabetes, and hence would make these early nutrition-related insults a contributor to diet-related chronic diseases. This factor is of special importance in rapidly developing countries such as those of Asia where high rates of LBW and stunting are accompanied by concurrent rapid shifts in diets, activity patterns, and increased obesity (Popkin et al 2001). The ‘early malnutrition wars’ begin in utero during early embryonic and foetal development when the developing foetus depends entirely on its own genotype and the materno-fetal environment. During these critical periods of human development there is increased demand for energy to meet the metabolic demands of pregnancy, i.e. to support maternal weight gain and ensure the growth of the foetus, placenta and associated maternal tissues (Prentice et al 1994, Shetty and James 1994, FAO 2004).

Reduced energy intake or food restriction, particularly in an undernourished mother, will predispose to her to nutritional stresses, resulting in intrauterine growth restriction as a coping mechanism, with varying outcomes that include the birth of babies who are small for gestational age or of low birth weight, which has early life and/or later life health consequences (Barker 1997, Godfrey and Robinson 1998). The pattern of growth during the critical periods of foetal life is a strong predictor of later susceptibility to type 2 diabetes, hypertension and hyperlipidemia (Barker et al 1993, Goldberg and Prentice 1994, Lucas et al 2001, Robinson 2001, Ericksson 2004).

There is considerable evidence from animal studies and clinical assessments in human subjects to support the view that the prenatal environment triggers developmental changes in the endocrine, organ and physiological characteristics of the foetus that may persist after birth. The list of postnatal traits shown to be influenced by the prenatal environment has expanded to include important functions such as immunity, reproductive function, growth rates and muscle mass (Prentice and Goldberg 2004, Kuzawa 2005).

Chronic hunger and continuous maternal malnutrition during pregnancy thus triggers foetal adaptations that are aimed at survival, probably with ‘energy economy’ at the centre of these survival mechanisms. The avid desire to economize and preserve energy
at all costs under extreme conditions, most probably occurs at the expense of other metabolic needs such as the synthesis of protective proteins of the immune system. Such compromises may in turn increase the risk of infectious and communicable diseases in the neonate and infant and thus contribute markedly to the high rates of perinatal and infant mortality seen in many poor countries. Figure 2 shows a proposed model of interactions that may have a bearing on health outcomes in food insecure environments.

In context of mothers in poor developing countries, the thrifty phenotype and thrifty genotype hypotheses (Hales and Barker 1992) are of particular significance. In the former it is proposed that foetuses subjected to early nutrient restriction become metabolically attuned to these conditions and sustain aspects of a thrifty metabolism for the rest of their life (Wells 2002, Wells and Cole 2002). The implications are that if the conditions of poverty and chronic hunger are replaced by affluence or nutritional abundance in later life, then thrifty phenotypes may be especially vulnerable to diseases of affluence, including type 2 diabetes and CVD (Moore 1998). Furthermore, the ‘veterans’ of the ‘early malnutrition wars’, who are born alive and may be of low birth weight, are more susceptible to infectious illnesses developed through the effects of intrauterine malnutrition on the developing immune system (Prentice and Goldberg 2004).

The thrifty phenotype model suggests that the organism adapts to poor nutrition in early life by programming its insulin metabolism to expect a similarly depleted environment subsequently, and this adaptation appears to operate through insulin resistance rather than insulin secretion (Fowden and Hill 2001). The thrifty phenotype and thrifty genotype hypotheses are best viewed as complementary. The thrifty genotype hypothesis can account for selection over many generations, and hence explain the population differences in susceptibility to type 2 diabetes, whereas the thrifty phenotype hypothesis lends itself to individual adaptations during a single lifespan (Lindsay and Bennett 2001).
Figure 2: A proposed model of interactions between food insecurity, human adaptations and nutritional and health risk in situations of poverty and chronic hunger. IUGR, intrauterine growth restriction; ↓, decreased; ↑ increased.
The offspring may remain well protected as long as the environment is poor, but the programmed trait becomes inappropriate and maladaptive when the environment improves, e.g. with affluence or improved nutrition. Also, substantial evidence is available on the genetic predisposition to the risk of early onset of NCDs following migration and the consequent environmental changes. Rural and urban differences in the prevalence of NCDs within a region or state in India show variances in disease risk due to internal migration and urbanization, increasing the risk of developing chronic diseases (Shetty 2002).

Popkin identified the unique components that characterize the transition that is occurring in the middle and low income countries. It was the shift in diet which occurred within 100 – 200 years in the West, has occurred only in a few decades in the developing countries. The speed of transition and the factors influencing it vary from country to country and also within a country within various sub-groups. It is also an interesting phenomenon that both under-nutrition and over-nutrition is found in the same countries, even in the same households. There may also be some biological differences (eg. larger prevalence of thrifty genotype) between the earlier and newly developing nations that explain the intensity of the shifts in diet and chronic diseases.

**Emergence of Non Communicable Diseases (NCDs)**

Nutritional, Demographic, Epidemiological and Socioeconomic transitions occurring in many developing countries like India have been established as a cause of continuing undernutrition and escalating overnutrition creating a double jeopardy of communicable and non communicable diseases. This double burden poses an apparent insurmountable health and economic challenge in resource constrained populations (Popkin *et al* 2001). Often overall nutrient intake adequacy improves with an increasing variety of foods, but the movement toward more fats, salt, sugars and refined foods quickly moves beyond this more optimal state to one in which diets contributes to rapidly escalating rates of obesity and chronic diseases (Tucker and Buranapin 2001).
The most immediate obvious result of the combination of energy-dense diets with physically inactive lives is a rapid increase in numbers of overweight and obese people (Popkin et al 2001, Shetty 2002). Salt-sensitive hypertension or fat-sensitive cardiovascular disorders that may not have been expressed on a traditional diet may become prevalent (Solomons and Gross 1995). (Table 9)

Obesity is a natural consequence of over nutrition and a sedentary lifestyle. Persistent obesity dysregulates metabolic processes including action of insulin on glucose-lipid-free fatty acid metabolism and severely affects processes controlling blood glucose, blood pressure, and lipids. Thus begins a cluster of conditions; dysglycemia, dyslipidemia, hypertension, and procoagulant state, known as the metabolic syndrome (Misra and Khurana 2008). Data suggests that obesity and metabolic syndrome are immediate precursors of type 2 diabetes and CVD (Misra and Khurana 2008). With a substantially high annual rate of increase of obesity and rapid emergence of metabolic syndrome in most developing countries, shift in the pattern of NCDs (Table 10) is occurring at a faster rate than it did in the industrialized regions of the world half a century ago (WHO 2002).

Global prevalence of chronic diseases is projected to increase substantially over the next two decades in developing countries. 60% of the global burden of chronic diseases is expected to occur in developing countries. CVD is already the leading cause of mortality in many developing countries. In the World Health Report (1999), it was stated that in 1998, 78% of the burden of NCDs and 85% of CVDs arose from the developing low- and middle-income countries. Furthermore, according to projected data, chronic diseases will account for almost three quarters of all deaths worldwide by 2020 and that 71% of deaths due to CVD and 70% of deaths due to diabetes will occur in developing countries (WHO 1998). In fact, there are more patients with CVD in India and China than in all the economically developed countries (WHO 2002). In 2000, the prevalence of diabetes worldwide was 171 million patients which is expected to increase to 366 million by 2030, of which 298 million will be in developing countries. The number of people with diabetes is projected to double from 2000 to 2030, in developing regions including South Asia (Wild et al 2004).
Table 9: The possible effects of dietary intake and body composition on Non communicable diseases.

<table>
<thead>
<tr>
<th>Dietary Factor</th>
<th>Mechanism</th>
<th>Health Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excess energy intake †</td>
<td>Adipose tissue development †,</td>
<td>NIDDM † (a), CHD † (a), hormone-dependent (e.g., breast) or GI (e.g., colorectal) cancers † (a), osteoarthritis † (a), gallbladder disease † (a)</td>
</tr>
<tr>
<td></td>
<td>metabolic changes</td>
<td></td>
</tr>
<tr>
<td>Total fat †</td>
<td>Passive overconsumption, IR †</td>
<td>NIDDM † (b), CHD † (a), prostate cancer † (b), breast cancer † (c), colorectal cancer † (b)</td>
</tr>
<tr>
<td>Animal fat †</td>
<td>Unclear, fat metabolism by products</td>
<td>Colon cancer † (b)</td>
</tr>
<tr>
<td>Saturated fat †</td>
<td>TC †, LDL-C †, TG †, HDL-C †</td>
<td>Arteriosclerosis † (a), CHD † (a), Hypertension † (b), NIDDM † (b)</td>
</tr>
<tr>
<td>Trans-fatty acids †</td>
<td>LDL-C †, HDL-C †, TC †, immune system †</td>
<td>Cancers † (d), CHD † (c)</td>
</tr>
<tr>
<td>Monounsaturated fatty acids †</td>
<td>LDL-C †</td>
<td>Cancers † (c), CHD † (b)</td>
</tr>
<tr>
<td>Polyunsaturated fatty acids †</td>
<td>HDL-C †, some are anti-inflammatory</td>
<td>Cancers † (b), CHD † (b)</td>
</tr>
<tr>
<td>Sodium †</td>
<td>Abnormal renal function†, disturbed electrolyte balance†</td>
<td>Hypertension † (a), Stroke † (a)</td>
</tr>
<tr>
<td>Antioxidants †</td>
<td>Oxidize LDL-C, change functions</td>
<td>CHD † (b)</td>
</tr>
<tr>
<td>Dietary fibre †</td>
<td>TC †, HDLC †, IR †, TG †</td>
<td>CHD † (b), NIDDM † (b), Stroke † (c), colon cancer † (c)</td>
</tr>
<tr>
<td>Fruits and vegetables †</td>
<td>Prevent oxidization LDL-C, fibre†</td>
<td>Stroke † (b), cancers † (a)</td>
</tr>
<tr>
<td>Foetal malnutrition/stunting †</td>
<td>Central adipose tissue †, IR †, metabolic changes</td>
<td>NIDDM † (b), Hypertension † (b), CHD † (b)</td>
</tr>
</tbody>
</table>

Notes: TC (total cholesterol); LDL-C (low-density lipoprotein cholesterol); TG (triglycerides); HDL-C (high density lipoprotein cholesterol); IR (insulin resistance). Category of the relationship between dietary factors and health outcomes: a: well established; b: fairly well established but data not complete; c: still under debate; d: suggestive data to date.

* Epidemiological studies support much of what is noted here but much controversy surrounds this literature; in particular the mechanisms that are presented in the table. In addition, we omitted the effects of reduced physical activity that is most important in increasing obesity, reducing fitness, and increasing insulin resistance.
India has and will continue to have the highest number of patients with diabetes in the world (Sicree et al 2006). The resulting increase in morbidity and mortality due to obesity and consequent chronic NCDs are a matter of great concern. Between 1990 and 2020, mortality from CVD in developing countries is expected to increase by 120% for women and 137% for men, which is expected to be substantially greater than from developed countries (29% and 48%, respectively). A near tripling of CVD mortality in Latin America, Middle East, and Sub-Saharan Africa is expected to occur in next two decades. In India, increase in CVD mortality is expected to reach two million by 2010 (Misra and Khurana 2008).
Health Costs

A new study, based on surveys conducted in 89 countries covering nearly 90% of the world’s population, provides for the first time a global estimate of the scale and distribution of catastrophic health care spending. From the study, it can be concluded that each year an average of 2.3% households experience financial catastrophe due to health care costs, corresponding to over 150 million people worldwide. More than 100 million people are impoverished because they must pay for health care (Figure 3).

Catastrophic health care spending occurs in countries at all levels of development. Nevertheless, the problem is more frequent and more severe in middle-income countries, and most frequent and most severe in low-income countries like India (Xu et al 2007).

Figure 3: Catastrophic health expenditure and impoverishment due to out-of-pocket health expenditure by WHO region (WHO 2008).
Obesity

Body weight stability and the associated regulatory processes depend upon nutrient intake, but are also influenced by compensatory genetic-dependent metabolic and neuro-endocrine mechanisms. The control and maintenance of body composition has been the subject of a number of theories or pathways such as the occurrence of a physiological set point for body weight, glucostatic or glycogen drives for feeding, metabolic/nutrient partitioning approaches, the participation of the nervous system, an adipostat mediated by signals from the adipose tissue, all of which might be under genetic control and explain individual variability (Martinez 2000). It has been hypothesized that the stability of body weight and composition depends upon an axis with three inter-related and self-controlled components: (1) food intake; (2) nutrient turnover and thermogenesis, and (3) body fat stores (Martinez and Fruhbeck 2001). All three elements underlie complex interrelated feedback mechanisms, which are affected by the individual’s genetic background.

As discussed earlier, the aetiology of obesity is multifactorial which includes genetics, environment, nutrition and level of physical activity. Both animal and human studies have established a high degree of association between genes and obesity. The DNA carries out a triple biological function, being the basis of inheritance, individualization and change; that is, this nucleic acid transmits the specific characters of the species, allowing differentiation among individuals. The individual’s genetic information (genotype) has the capacity to codify both cell development and functions throughout life. Intrinsic and extrinsic noticeable characters and features in each person (phenotype) are the result of the interaction of the genotype with environmental influences (Marti et al 2004).

Any inheritable change in the sequence of DNA constitutes of a mutation that can occur in a single nucleotide (SNPs) through inversion, deletion, repetition, insertion or translocation within chromosomal segments. Obesity, as a complex syndrome with a multifactorial origin may be explained in some circumstances by monogenic mutations, but in most cases appears as a polygenic condition, which may be additionally affected
by a myriad of environmental influences (Marti et al 2004). The role of a genetic predisposition in obesity has long been assumed to affect both sides (intake/expenditure) of the energy equation. Genes may determine afferent and efferent signals as well as central mechanisms involved in body weight regulation. Thus, the transferable genetic information involved in short and long-term stable body weight regulation and diet composition maintenance (Marti et al 2004) acts via (1) different peptides and monoamines involved in the regulation of the appetite, (2) variations in energy and nutrient utilization, resting metabolic rate or response to physical activity, and (3) individual differences in adipocyte metabolism. The possible mechanisms through which the genetic susceptibility could be acting include reduced rates of basal metabolism and macronutrients oxidation, alterations of adipogenesis and quantitative and qualitative deviations of food intake. Also, other factors such as the hormonal profile, energy exercise efficiency and thermogenesis could be specifically involved in the genetic processes affecting the energy balance equation (Marti et al 2004).

Although genetic abnormalities make it more difficult to lose weight, the dramatic rise in obesity over the past decades cannot only be attributed to genetics as genes could not have been modified in such a short span of time. Earlier studies estimated that heredity influence accounted for 80% of the tendency to gain weight, but more recent data indicate that only 33% of the BMI is attributable to genetics (Oeser 1997).

The processes of modernization and economic restructuration in both low and high-income countries have influenced nutritional and physical activity patterns that contribute to the increasing rates of obesity. The IOTF “Causal Web” (Figure 4) highlights the complex matrix of societal factors influencing both childhood and adult obesity. The environmental factors exerting an overarching influence over individual behaviour in most societies are the globalisation of markets, economic development, media and advertising (marketing). These influence populations to differing extents, depending on regional and national factors. The cultural filter determines the degree to which each global factor impacts upon an individual society.
Figure 4: Environmental factors influencing Obesity (The IOTF Casual Web).
Figure 5: Food Consumption and Obesity.

**EXOGENOUS FACTORS**
- Food availability (SES)
- Food variety, palatability, preparation
- Religious, socio-cultural belief, Image of food
- Environment (temperature etc)
- Subjective factors (preference)
Food consumption patterns and preferences are one of the major aetiological causes of obesity (Figure 5). The increasing prevalence of obesity in United States has been paralleled by increase in energy intake, especially of highly palatable dietary ingredients like sugar and fats. They account for almost 60% of the daily energy content of typical American diet; sugar accounts for 22% and fats for 37%. Investigations have shown that women prefer high sugar and high fat foods and men prefer high fat and high protein foods (Salbe et al 2004).

Overall, Asians and South Asians could be classified as ‘metabolically obese’, i.e. they have several metabolic derangements but are non-obese by conventional BMI standards. These non-obese people usually have high body fat; abdominal adiposity; ectopic fat deposition; and, specifically in South Asians, thick truncal fat. These body composition characteristics, individually or in combination, contribute to insulin resistance, dyslipidemia, hyperglycemia, and excess pro-coagulant factors seen commonly in South Asians (Misra and Khurana 2008). (Figure 6)

**Figure 6: Lifecycle: Obesity – The proposed links.**
**Classification of Overweight and Obesity**

Body Mass Index (BMI) is commonly used to classify overweight and obesity in adults. The classification of overweight and obesity, according to BMI, is shown in Table 11 as recommended by WHO (2003). The classification is primarily based on the association of mortality with BMI. Data suggests that the proposed cut-offs for defining overweight and obesity are not appropriate for Asian Indians, and that Asian Indians are 'at risk' of developing obesity related co-morbidities at lower levels of BMI. Hence, a different BMI classification (Table 11) is used for South Asians (Misra et al 2009). BMI and body fat content varies with body build and proportion. However, BMI does not distinguish between weight associated with muscle and weight associated with fat.

**Table 11: Classification of obesity based on BMI.**

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (kg/m²)</th>
<th>WHO cut off</th>
<th>BMI (kg/m²)</th>
<th>South Asian cut off</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt; 18.5</td>
<td>&lt; 18.5</td>
<td>&lt; 18.5</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>18.5 – 24.9</td>
<td>18.5 – 22.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>≥ 25.0</td>
<td>23.0 – 24.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre obese</td>
<td>25.0 – 29.9</td>
<td>25.0 – 27.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>30.0 – 34.9</td>
<td>28.0 – 30.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class II</td>
<td>35.0 – 39.9</td>
<td>30.0 – 34.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class III</td>
<td>&gt; 40.0</td>
<td>≥ 35.0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Adipose tissue consists of adipocytes, inflammatory cells, and vascular, connective and neutral tissues. It is distributed throughout the body as large homogenous discrete compartments and as number of cells 'marbling' or adjacent to other tissues. Most adipose tissue (~ 85%) of total adipose tissue mass is located under the skin (subcutaneous fat), and smaller amount (~ 15%) is located within the abdomen (intra-
abdominal fat) in lean and obese individuals. The relative contribution of intra abdominal fat mass to total fat mass is influenced by sex, age, race-ethnicity, physical activity and total adiposity. The relationship of weight, BMI and WC can be conceptualized by using simple geometric relations that consider the body as a cylinder; WC is the cylinder's circumference, height its length and weight its mass. Thus, BMI provides information about the volume and mass, and WC about its shape. WC reflects both intra abdominal and subcutaneous adipose tissue (Klein et al 2007).

Hence, recently Waist Circumference (WC) is used to assess obesity as it is found to be a good indicator of body fatness. High correlation has been found between WC and BMI, visceral fatness and total body fat (Katzamrzyk et al 1999). The importance of WC in predicting cardiometabolic risk factors (elevated blood pressure, dyslipidemia, and hypertriglyceridermia) and adverse outcomes (diabetes, CHD and mortality) have been examined in large epidemiological studies (Figure 7). Specific relative risk varies depending on the population sampled. It has been found to be a stronger predictor of diabetes, CHD and mortality rate, independent of traditional clinical tests such as blood pressure, blood glucose and lipoproteins (Wang et al 2005, Yusuf et al 2005).

The currently recommended cut-offs of WC (>102 cm men and >88 cm women) are not applicable to all the populations due to heterogeneity in the average levels of measurements. Asian Indians appear to have higher morbidity at lower cut-off for WC than do White Caucasians. WC cut offs of ≥ 90 cm in men and ≥ 80 cm in women identified high odds ratio (4.2 & 2.2, respectively) for cardiovascular risk factors (Misra et al 2009).

**Obesity and Hypertension**

Obesity can lead to development of hypertension. Studies have revealed that both systolic and diastolic blood pressure increase with increasing BMI, and that an obese individual is at a higher risk of developing hypertension as compared to their leaner counterparts (Stamler et al 1989).
Figure 7: Proposed mechanisms by which visceral obesity can be linked to metabolic syndrome.

VISCERAL OBESITY = DYSFUNCTIONAL ADIPOSE TISSUE?

A Portal circulation
   Insulin resistant
   Altered FFA metabolism
   Hyperinsulinemia
   Glucose intolerance
   Hypertriglyceridemia
   Etc.

B ↑ IL-6
   ↑ TNF-α
   ↓ Adiponectin
   ↑ Other adipokines
   Release of cytokines
   Altered metabolic profile:
   - Insulin resistant milieu
   - Proinflammatory state
   - Prothrombotic state
   - Prohypertensive state

C Ectopic fat deposition
   Impaired clearance and
   storage of TG in subcutaneous fat
   Lack of or dysfunctional subcutaneous fat

Kodali et al (1997) conducted studies among Indian subjects aged between 30 – 50 yrs, and reported that hypertensives had a significantly higher body weight, body fat, BMI and WHR. The results from large epidemiological studies and intervention trials suggest that the risk of developing hypertension in normotensive women is inversely correlated with changes in body weight. The reason however is unclear. It has been postulated that as obese and overweight individuals are prone to insulin resistance (Misra et al 2009) resulting in higher circulating insulin levels. This in turn enhances renal retention of sodium, resulting in increased blood pressure levels (Brenner et al 1988).

Recent advances in the genetics and neurobiology of obesity have begun to contribute to the understanding of mechanisms of obesity induced hypertension. Leptin, in addition to its effects on appetite and metabolism, has been found to have sympathetic, vascular, and renal actions that can influence blood pressure. However, the effect of obesity on blood pressure may depend critically on the genetic-neurobiological mechanisms underlying the obesity. Further, modifying alleles in the genetic background may critically influence the blood pressure response to obesity (Mark et al 1999).

Weight loss decreases both systolic and diastolic blood pressure in a dose-dependent fashion; therefore, greater weight loss is generally associated with greater improvement in blood pressure. Weight regain results in a steady increase in blood pressure toward baseline.

**Obesity and Dyslipidemia**

Obesity, especially abdominal obesity is known to cause insulin resistance which in turn causes aberrations in the lipid metabolism. Hepatic overproduction of VLDL is the primary and crucial defect in obesity and the insulin-resistant state. The inability to suppress hepatic glucose production, impaired muscle glucose uptake and oxidation, and the inability to suppress release of non-esterified fatty acids (NEFA) from adipose tissue are the most important consequences of insulin resistance in liver, muscle, and adipose tissue respectively. These events together increase NEFA and glucose flux to the liver, an important regulator of hepatic VLDL production. Another key site in the regulation of
VLDL secretion is the rate of apolipoprotein B-100 (ApoB-100) degradation. Lipids and microsomal triglyceride protein, a heterodimeric lipid transfer protein that is required for the assembly of Apo B-containing lipoproteins, are vital in the translocation of Apo B-100. If this translocation fails to occur, then Apo B-100 is degraded. Insulin is also important for the intracellular degradation of freshly translated Apo B-100. Therefore, obese or insulin resistant individuals, there is an inability to suppress Apo B-100 degradation that occurs along with a consequent imbalance between secretion and degradation (Howard et al 2003).

Small, dense LDL concentration and fasting triglyceride levels are positively correlated, because the formation of small, dense LDL depends largely on the metabolism of VLDL particles. In people with obesity and insulin resistance, the increased concentration and delayed clearance of VLDL particles induce an increased exchange between cholesterol esters in LDL and triglycerides in VLDL, mediated by Cholesterol Ester Transfer Protein (CETP). This exchange produces LDL particles enriched in triglycerides that are rapidly lipolyzed by hepatic lipids, leaving smaller, denser LDL particles. The mechanisms that regulate HDL are not understood completely. Most studies of lipoproteins have shown an inverse relationship between VLDL triglycerides and HDL - C. Impaired Triglyceride Rich Lipoprotein (TRL) lipolysis leads to reduced HDL - C concentration by decreasing the transfer of apolipoproteins and phospholipids from TRL to the HDL compartment. In addition, the delayed clearance of TRLs facilitates the CETP - mediated exchange between cholesterol esters in HDL and triglycerides in VLDL. The increased activity of hepatic lipids in obesity and insulin-resistant states produces smaller HDL particles and facilitates HDL clearance. Finally, insulin also seems to stimulate the production of Apo A₁ or hepatic secretion of nascent HDL. Therefore, in people with obesity and insulin resistance, a substantial decrease of HDL particles occurs, especially of the larger HDL₂ (compared with the smaller HDL₃) and HDL containing mostly Apo A₁ (Howard et al 2003).

Central obesity has a significant role to play not only in insulin resistance but also in obesity induced dyslipidemia. It has been suggested that a limited ability of subcutaneous
fat depots to store excess energy results in an “overflow” of chemical energy to Intra Abdominal Adipose Tissue (IAAT) and “ectopic” sites, such as liver and skeletal muscle. Excessive ectopic fat accumulation then causes metabolic dysfunction in those organs. In fact, increased intrahepatic fat is associated with dyslipidemia and hepatic insulin resistance, and increased intramyocellular fat is associated with skeletal muscle insulin resistance paradigm. IAAT is primarily a marker of the magnitude of overflow of fatty acids from subcutaneous depots. Therefore, increased WC could be a discernible marker of a system-wide impairment in energy storage regulation, in which an increase in IAAT reflects a reduced capacity for energy storage in other adipose tissues.

A third hypothesis proposes a direct effect of omental and mesenteric adipose tissue depots on insulin resistance, lipoprotein metabolism, and blood pressure. Metabolic products of omental and mesenteric adipose tissue depots are released into the portal vein, which provides direct delivery to the liver. Lipolysis of omental and mesenteric adipose tissue triacylglycerols releases free fatty acids that can induce hepatic insulin resistance and provide substrate for lipoprotein synthesis and neutral lipid storage in hepatocytes. In addition, specific proteins and hormones produced by omental and mesenteric adipose tissue, such as inflammatory adipokines, angiotensinogen, and cortisol (generated by local activity of 11-hydroxysteroid dehydrogenase), can also contribute to cardiometabolic disease.

**Obesity and Cardiovascular diseases**

Obesity is an important risk factor for coronary heart disease (CHD), ventricular dysfunction, congestive heart failure, stroke, and cardiac arrhythmias. The longitudinal Framingham study strengthened this association and disclosed that the cardiovascular risk increases with increasing levels of obesity, independent of other standard risk factors. Data from large epidemiological studies have shown that weight variability is associated with an increased rate of CVD mortality. Obesity, particularly severe obesity, is associated with abnormalities in cardiac structure and function. The severity of these defects is associated with both the degree and duration of obesity. Obesity is associated
with an increase in total blood volume and cardiac output and a decrease in peripheral vascular resistance. Ventricular filling pressures are elevated which eventually results in increased wall stress, diastolic dysfunction, and left ventricular hypertrophy. Abnormalities of the right heart can also occur and may represent a combination of left heart disease, recurrent pulmonary thromboemboli, and hypoventilation. Weight loss, particularly in persons who are severely obese, can improve cardiac structure and function. Improvements in fractional shortening are associated with decreases in hypertension and left ventricular internal dimension with reduced atrial and left ventricular free and septal wall thickness. Moreover, improvements in left ventricular diastolic filling and ejection fraction also occur. Improvements in left ventricular mass occur in both normotensive and hypertensive patients and are independent of the reduction in blood pressure. Adding exercise to a low calorie diet may produce greater benefits in cardiac structure (Klein et al 2004).

Obesity also contributes to the development of vascular inflammation which raises the markers of inflammation like C-Reactive Protein (CRP). CRP levels have been shown to be associated with percentage of fat, Waist to hip ratio, WC, and triceps skin fold thickness in Asian Indian children (Vikram et al 2003). In Asian Indian adolescents high CRP levels were seen in 13% subjects overall, in ~22% overweight and ~25% in those with excess body fat (Vikram et al 2006). High levels of CRP levels denote future risk for development of CHD and type 2 diabetes (Ridker 2003).

**Obesity and Diabetes Mellitus**

Obesity is a major cause of type 2 diabetes in all affected populations. No simple metabolic defect is likely to explain the cause of obesity-induced diabetes in large numbers of people and the precise links between obesity and diabetes have yet to be unequivocally identified. Clinicians have observed for centuries that fatter people are more likely to have diabetes and overwhelming evidence has accumulated to prove this clinical impression accurate. The association of obesity with diabetes has been observed in comparisons of different populations and within populations (Tataranni 2002).
Prospective studies of pre-diabetic subjects have shown conclusively that obesity and its duration are major risk factors for type 2 diabetes (Everhart 1992).

Circumstantial and experimental evidence indicate that weight gain causes hyperinsulinemia and insulin resistance (Figure 8). Obese individuals develop resistance to the cellular actions of insulin, characterized by an impaired ability of insulin to inhibit glucose output from the liver and to promote glucose uptake in fat and muscle. The association between obesity and insulin resistance is likely a cause-and-effect relationship since human and animal studies indicate that weight loss/gain correlates closely with increasing/decreasing insulin sensitivity, respectively (Qatanani and Lazar 2007).

Classically, FAs secreted from adipocytes have been considered to serve entirely as energy sources for other tissues of the body. It has been hypothesized that obesity-associated insulin resistance could be explained by competition between these increased circulating FAs and glucose for oxidative metabolism in insulin-responsive cells. More recently, glucose uptake rather than intracellular glucose metabolism has been implicated as the rate-limiting step for FA-induced insulin resistance. In this model, FAs and potentially several metabolites including acyl-CoAs, ceramides, and diacylglycerol serve as signalling molecules that activate protein kinases such as Protein Kinase C (PKC), Jun kinase (JNK), and the inhibitor of nuclear factor-κB (NF-κB) kinase-β (IKKβ). These kinases can then impair insulin signalling by increasing the inhibitory serine phosphorylation of insulin receptor substrates (IRS), the key mediators of insulin receptor signalling (Qatanani and Lazar 2007).

Leptin is an adipocyte-secreted hormone whose absence leads to dramatic metabolic derangements. The discovery of leptin ushered in an era of receptivity to the notion that adipose tissue is an endocrine organ, and that increased adipose mass in obesity could lead to pathological changes in adipocyte hormones (adipokines) that regulate insulin sensitivity. Adiponectin is structurally related to complement 1q, is specifically expressed in differentiated adipocytes, and circulates at high levels in the bloodstream. Adiponectin levels are low in obesity, and administration of adiponectin improves insulin resistance in
animal models. In the liver, adiponectin enhances insulin sensitivity, decreases influx of FAs, increases FA oxidation and reduces hepatic glucose output. In muscle, adiponectin stimulates glucose use and FA oxidation probably via activation of the cellular fuel sensor, AMP-activated protein kinase (AMPK) (Qatanani and Lazar 2007).

Resistin was identified in 2001 as an adipocyte-specific secreted protein whose expression is down regulated by antidiabetic drugs targeting the nuclear receptor peroxisome proliferator-activated receptor γ (PPAR γ). Serum resistin is elevated in rodent obesity, and infusion or sustained expression of resistin produces insulin resistance, conversely, mice lacking resistin have improved glucose homeostasis. This effect is mediated at least in part via increased activity of AMPK and decreased expression of gluconeogenic enzymes in the liver. Moreover, resistin has been shown to induce the expression of suppressor of Cytokine Signalling-3 (SOCS-3), a well-known negative regulator of insulin signalling. The role of resistin in humans is less certain. Some studies show increased resistin expression and serum levels in association with obesity and insulin resistance. Recent studies in humans show a consistent association between resistin and inflammation (Qatanani and Lazar 2007).

Cortisol is another endocrine factor produced by adipose tissue. Elevated glucocorticoid levels cause insulin resistance and type 2 diabetes, primarily by opposing the anti-gluconeogenic effects of insulin in the liver. Insulin resistance and type 2 diabetes are associated with a decrease in mitochondrial function that contributes to the ectopic fat accumulation in muscle and fat. It has been found that severe insulin resistance is associated with significantly higher levels of triglycerides in both muscle and liver in the elderly. These changes were accompanied by decreases in both mitochondrial oxidative activity and mitochondrial ATP synthesis, both indicative of a decrease in mitochondrial function. Other studies have revealed similar decreases in mitochondrial activity and increases in intramyocellular fat content in young insulin-resistant offspring of parents with type 2 diabetes, a group that has a strong tendency to develop diabetes later in life (Qatanani and Lazar 2007).
It was suggested that the insulin-resistant subjects accumulate more intramyocellular fat due to a decrease in the number of muscle mitochondria caused by a decrease in the expression of nuclear-encoded genes that regulate mitochondrial biogenesis, such as PPARγ coactivator 1 α (PGC-1α) and PGC-1β. This idea was supported by microarray studies that show that PGC-1-responsive genes are down-regulated in obese Caucasians with impaired glucose tolerance and type 2 diabetes, and PGC-1α and PGC-1β are themselves down-regulated in both obese diabetic and overweight nondiabetic Mexican-Americans.

Finally, activation or induction of PGC-1α has recently been shown to be associated with improved mitochondrial function as well as increased insulin sensitivity in both animals and humans. These data support the idea that insulin resistance in humans might arise from defects in mitochondrial function, which in turn lead to increases in intracellular FA metabolites (fatty acyl-CoA and diacylglycerol) that disrupt insulin signalling in the muscle as well as the liver. The decrease in mitochondrial function associated with obesity and insulin resistance might seem paradoxical given that it is known that functional mitochondria are needed for an FA-induced increase in ROS (Qatanani and Lazar 2007).

It is possible that an increase in ROS due to FA oxidation occurs early during the development of insulin resistance and prior to mitochondrial dysfunction. At a later stage, ROS might lead to a decrease in mitochondrial function that then leads to the accumulation of fat in the muscle and liver, exacerbating the insulin resistance phenotype via the mechanisms mentioned above (Qatanani and Lazar 2007). The concepts of "glucotoxicity" and "lipotoxicity" have been advanced to explain the pathogenesis of type 2 diabetes (Tataranni 2002). Physiologically, glycemia is maintained within a very narrow range by the daily meals. Despite these fluctuations, fasting glycemia is homeostatically controlled, i.e., glucose always returns to the initial level after each meal. As long as insulin resistance and the resulting mild hyperglycemia persists, the pancreas is forced to constantly over-secrete insulin, a condition termed 'allostatic load'.
Figure 8: Endocrine, inflammatory and neuronal pathways linking obesity to insulin resistance.

(A) The obesity-associated increase in FAs can trigger insulin resistance through intracellular metabolites that activate PKC, leading to the activation of serine/threonine kinases that inhibit insulin signalling. (B) Obesity-associated changes in secretion of adipokines that modulate insulin signalling. (C) Obesity-associated inflammatory factors. Obesity is characterized by an increase in the accumulation of ATMs, which increase the adipose tissue production of inflammatory cytokines that inhibit insulin signalling. (D) Endocrine and inflammatory mediators converging on serine/threonine kinases that inhibit insulin signalling. (E) Obesity-associated activation of NF-κB heightens inflammatory responses that exacerbate insulin resistance. (F) SOCS family proteins, induced by adipokines, induce insulin resistance either by interfering with IRS-1 and IRS-2 tyrosine phosphorylation or by targeting IRS-1 and IRS-2 for proteosomal degradation. (G) FAs also trigger insulin resistance by direct activation of TLR4 and the innate immune response. (H) Obesity-related alteration in the central response to hormonal and nutrient signals alters peripheral insulin sensitivity. (Qatanani and Lazar 2007)
Prospective analysis shows that normal glucose tolerant individuals with a high pancreatic allostatic load have an increased risk of developing type 2 diabetes compared to individuals with a low pancreatic allostatic load. Thus, obesity-induced insulin resistance may cause type 2 diabetes by increasing the allostatic load of the pancreas. One of the possible ways that an increased allostatic load can eventually lead to failure of the endocrine pancreas is through the direct detrimental effect of hyperglycemia on the beta cell, which is commonly referred to as glucotoxicity. Mechanisms include decrease in expression of relevant genes (insulin receptor, GLUT2, glucokinase, inward rectifier potassium channel, beta cell de-differentiation and increased apoptosis. Also, it has been suggested that chronic hyperglycemia per se can worsen insulin resistance (Tataranni 2002).

There is strong evidence that acute and chronic increases in fatty acid supply to peripheral tissues may play an important role in impairing glucose uptake and storage in the muscle. A theory has been set forth proposing that the adipose tissue plays a crucial role in buffering the flux of FFA in the postprandial period and that this buffering action becomes progressively impaired as obesity develops, which would in turn expose extra adipose tissues to excessive lipid fluxes. This theory is partially supported by the following observations. Obesity-induced insulin resistance is associated with increased lipid concentrations in insulin-responsive tissues (Tataranni 2002). Normal glucose tolerant people with enlarged subcutaneous abdominal adipocytes and elevated levels of FFA (possible markers of reduced adipose tissue buffering action) are at increased risk of developing type 2 diabetes. People with lipodystrophy (very little or no adipose tissue available to buffer daily lipid fluxes) are also insulin resistant and prone to type 2 diabetes. Experimental evidence indicates that the adipose tissue progressively loses its normal ability to buffer excessive lipid fluxes as obesity develops.

An unequivocal biochemical explanation for a role for FFA in regulating glycogen synthesis is still missing. Some possible mechanisms proposed, are the effects of long chain fatty acids on glycogen synthase, cellular membrane fluidity, translocation of GLUT4 containing vesicles and activation of the hexosamine pathway (Tataranni 2002).
The toxic effect of lipids may extend to the beta cell. FFA serves as an important source of energy for most body tissues, but they have a broader function as signals in a variety of cellular processes. One such role is to enhance the responsiveness of the pancreas to a variety of insulin secretagogues. However, numerous studies indicate that chronic exposure of the pancreas to elevated FFA concentrations has deleterious effect on the beta cells. Possible mechanisms underlying the lipotoxic effect of FFA on the beta cell include overproduction of NO, interleukin-1B, and ceramide, the latter likely responsible for the accelerated apoptosis observed in fat-laden beta cells (Tataranni 2002).

**Diet Related Non Communicable diseases (NCDs)**

**Hypertension**

Hypertension is a common public health problem, and if it goes untreated it may lead to many degenerative diseases. It is often called the ‘silent killer’ because hypertensives may go undetected for a long time as it may be asymptomatic unless something adverse occurs. A general definition of hypertension is a systolic blood pressure of 140 mmHg or higher or a diastolic blood pressure of 90 mmHg or higher or both. The types of hypertension are

**Primary/Essential/Idiopathic Hypertension.**

In this type the exact cause of the disease is unknown and is a long term progressive disorder. The predisposing factors of this kind of hypertension are:

- a) Genetic, b) Stress, c) Obesity, d) High sympathetic tone,
- b) Increased salt intake (>6g/day), f) Alcoholism, g) Smoking, h) Diabetes

**Secondary Hypertension**

Secondary complication is when hypertension occurs due to some other disease or abnormality. The causes could be Renal, Endocrinal, Neurological, Drugs, Corticosteroids, etc. 90 – 94% of the hypertensives have primary or essential
hypertension and only 6 – 8% of the subjects have secondary hypertension. Table 12 gives the classification of Blood Pressure.

**Table 12: Classification of Blood Pressure.**

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Pre-hypertension</td>
<td>120 – 139</td>
<td>80 – 89</td>
</tr>
<tr>
<td>Hypertension stage I</td>
<td>140 – 159</td>
<td>90 – 99</td>
</tr>
<tr>
<td>Hypertension stage II</td>
<td>≥ 160</td>
<td>≥ 100</td>
</tr>
</tbody>
</table>

**Pathophysiology of Hypertension**

Blood pressure is a function of cardiac output multiplied by the peripheral resistance (the resistance in the blood vessels to the flow of blood). The diameter of the blood vessel markedly affects the blood flow. When the diameter is decreased (as in atherosclerosis) the blood pressure increases. Conversely, when the diameter is increased (as with vasodilator therapy), resistance decreases and blood pressure is lowered. Many systems maintain the Homeostatic control of blood pressure. The major regulators are the sympathetic nervous system (for short term control) and the kidney (for long term control).

In response to fall in blood pressure, the sympathetic nervous system secretes a vasoconstrictor (norepinephrine) which acts on small arteries and arterioles to increase peripheral resistance and raise blood pressure (Figure 9). The kidney, on the other hand regulates blood pressure by controlling the extracellular volume and secreting rennin, which activates the Rennin - Angiotensin system (Figure 10). When the regulatory mechanisms falter, hypertension develops (Figure 11). The plausible causes of hypertension are hyperactive sympathetic nervous system, a stimulated rennin – angiotensin system, low potassium diet, etc.
Figure 9: Sympathetic control of blood pressure (Short term).

- Fall in blood pressure
  - SNS secretes norepinephrine (vasoconstrictor)
    - Acts on small arteries and arterioles
      - Peripheral Resistance
        - Rise in blood pressure

Figure 10: Renal control of blood pressure (Long term).

- Decreased arterial pressure
  - Renin (Kidney)
    - Renin Substrate
      - Angiotensin I
        - Angiotensin II
          - Angiotensinase (Inactivated)
            - Renal Retention (Na & Water)
              - Vasoconstriction
                - Increased Arterial Pressure
Figure 11: Pathogenesis of Hypertension.

Figure 12: Consequences of hypertension.

CHD = coronary heart disease
CHF = congestive heart failure
LVH = left ventricular hypertrophy

All these cause vasoconstriction, which results in ischemia or arterial changes. There are many neurohormonal and intrarenal causes of abnormal blood pressure. In most cases of hypertension, peripheral resistance increases. Resistance forces the left ventricle of the heart to increase its effort in pumping blood through the system. With time, left ventricular hypertrophy and eventually congestive heart failure can develop. Uncontrolled or untreated hypertension affects various organs of the body. Hypertension may lead to haemorrhages and cause stroke, CHD, retinopathy, peripheral vascular disease and proteinuria leading to renal failure (Figure 12).

In a study conducted to assess the hazard of increasing SBP in individuals aged above 65 yrs and of Asian and Caucasian origins, it was found that both the ethnic groups had increasing hazard ratio with increasing SBP. Another interesting observation was that the hazard ratio was almost double in Asians as compared to Caucasians (Perkovic et al 2007). Earlier it was believed that only hypertensives are at a risk of developing stroke and CHD, but recent review of literature has revealed that there is an increased risk in the so called normotensive individuals too as SBP and DBP increases (MacMohan et al 1990). The risk of developing adverse consequences of hypertension is now known as being continuous and constant, steadily rising with increase in blood pressure as indicated by Figure 13.

Figure 13: Relative Risk of Stroke with increasing DBP.

![Figure 13: Relative Risk of Stroke with increasing DBP.](image-url)
Figure 14: RCT of drug trials and its effect of blood pressure reduction on Stroke and CHD.

Meta-analysis of 147 RCT; Law, M R et al. BMJ 2009
The percent occurrence of stroke and coronary events is known to reduce substantially when blood pressure both SBP and DBP is controlled (Law et al. 2009) employing different techniques (Figure 14). Thus, we can conclude from these observations that reduction of blood pressure even in normotensive individuals by diet (like DASH diet) or exercise will not only prevent hypertension but also prevent occurrence of stroke and CHD. It has been estimated that a 2% decrease in DBP would avert 9% deaths due to stroke and IHD, and 7% reduction in SBP would bring down mortality by 11% by 2020. In all, 22% (i.e. 2 million) deaths can be averted in Asia and China by using combined approaches.

**Coronary Heart Disease**

Coronary heart disease (CHD) also known as coronary artery disease (CAD) or coronary atherosclerosis represents the single largest cause of major illnesses, disability and death in developed as well as developing countries. Coronary artery disease, also known as ischemic heart disease (IHD) is a ‘silent killer’, wherein 50% of the population that succumb to the disease have had no previous symptoms (silent ischemia). The secondary manifestations of coronary artery disease include angina pectoris, myocardial infarction, cardiac arrhythmias and sudden cardiac death.

The word “Atherosclerosis” is derived from the Greek word ‘Athero’ (gruel) and ‘sclerosis’ (hardening). It is the term used to describe the degeneration of arteries resulting in thickening and hardening of arterial walls. The anatomical changes in atherosclerosis are characterized by extensive fatty degeneration (atheromatoid foci) and by thickening of the arterial wall (sclerosis). To distinguish the disease, atherosclerosis is divided into stages namely early, advanced and complicated stage.

- **Early lesion**: It is the stage that describes the appearance of fatty streaks, gelatinous elevations and microthrombi in the intimal layer of the artery.
- **Advanced lesion**: This stage is characterized by the occurrence of fibrosis in the arteries and the presence of atherosclerotic plaques (atheromas).
• **Complicated lesion**: It is the stage where the plaques that are mechanically unstable may rupture. There might be ulceration, calcification and haemorrhage, which may further give rise to apoplexy, gangrene, aneurysm and infarction.

Fatty degeneration, connective tissue proliferation, necrosis and calcification are often combined in varying degrees of intensity. Atherosclerosis of the coronary arteries with or without subsequent thrombosis results in stenosis of the vascular lumen, progressing ultimately to complete occlusion (Figure 15). The consequences of this obstruction of flow in the coronary arteries are coronary insufficiency and coronary infarction (Small 1980).

**Figure 15: Cross section of an atherosclerotic artery.**

**Cytogenesis of cardiovascular disease**

Three primary factors which contribute to the impairment of tissue perfusion are enhanced vasoconstrictor responses, increased interaction of circulating blood cells and
structural changes in the arterial intima (Kathirvelu 2002). Endothelium, due to its
strategic location between the circulating blood and vascular smooth muscle cells is a
primary target and a mediator of cardiovascular disease. Functional integrity of the
endothelium is crucial for maintenance of blood flow and antithrombotic capacity
because the endothelium releases humoral factors that control relaxation and contraction,
thrombogenesis and fibrinolysis and platelet activation and inhibition. An imbalance
between endothelium derived contracting and relaxing factors results in endothelial
dysfunction. It often precedes structural and vascular alterations, which indicates a
protective role of a functionally intact endothelium.

Atherosclerosis is caused by functional and structural changes in the blood vessel wall.
These changes include abnormal vasoconstriction, enhanced interaction of blood cells
with the wall, activation of coagulation mechanism and migration and proliferation of
vascular smooth muscle cells. These vascular abnormalities have an important role in the
pathogenesis of angina, myocardial infarction, stroke, etc. Endothelial injury, either
physical trauma or more subtle cellular damage is an important initial event in the
development of atherosclerosis (Figure 16). Some of the proposed mechanisms are:

- Enhancing activities of coagulation factors (V and XII) or altering the anti-
thrombotic function of endothelium by depressing the level of anti-thrombotic
factor (Protein C) or the mechanism of their action Thromodulin or heparin
sulfate (Welch and Losculzo, 1998).

- Promotion of leukocyte recruitment (Poddar, 2001).

- Combination with LDL and producing aggregates that are taken by vascular
macrophages (Welch and Losculzo, 1998).


- Elevated blood viscosity and mean arterial pressure (Nappo, 1999).

- Marked platelet accumulation (Welch and Losculzo, 1998)
Figure 16: Cytogenesis of coronary heart disease- Endothelial dysfunction.

 PRIMARY RISK FACTORS
(e.g. hypertension, diabetes mellitus, atherogenic diet, emotional stress)

REACTION OF THE CELLS OF THE VASCULAR WALL
(Action of proliferation and metabolism in the mural cells)

ENDOTHELIAL
- Activated Endoplasmic Reticulum
- Intensified Cell Proliferation
- Increased permeability
- Villi, Gaps, Cell necrosis
- Deficiency of anti-thrombotic Factor

ELEVATED INFLUX
- Lipoproteins, Vascular Blood cell, Cell debris
- Reaction of Mural Cells (Persistence of pathological mural process)

INTIMAL EDEMA

MEDIAL
- Intensified cell
- Proliferation
- Increased synthesis of Proteoglycans, Collagen, Glycoprotein, Fatty acids, Cholesterol

Deposition of mesenchymal material,
Thickening of Wall
Deformation of structure
Expansion of sub-endothelial zone, Impaired flow of lymph vascular wall
Elevated lipid content

Thrombocyte Adhesion
Thrombocyte Aggregation

THROMBOSIS
LIPIDOSIS
FIBROSIS
Damage due to any of the causes physical or chemical, leads to endothelial dysfunction of the blood vessels, triggering aggregation of thrombocytes at the site of injury. Thrombotic factors then infiltrate the wall and induce migration and proliferation of smooth muscle cells along with the formation of fibres. The principal event in atherosclerosis is the proliferation of smooth muscle cells in the tunica intima which leads to elevated influx and production of proteoglycans, glycosaminoglycans, free fatty acids, etc. These cells increase the synthesis of collagen, especially collagen I (Libby 1995) and elastin fibres thus indicating the actual process of vascular sclerosis. Apart from platelet factors that are responsible for cell proliferation, it is also promoted by LDL. There is a constant perfusion of fluid in the region of the intima directed from the center to the periphery.

When the circulating levels of LDL-C in blood are very high, small amounts of LDL-C that build up in the wall get oxidized. Oxidized LDL-C injures the endothelium and causes the surface endothelium to express a special kind of molecular glue called ‘Endothelial leukocyte adhesion molecules’ (ELAMS). These cause certain kinds of white blood cells to adhere to the endothelium (Porro et al 2001). Along with the monocytes and T-lymphocytes, macrophages play a significant role in atherogenesis. The presence of macrophages reflects an inflammatory process, which is also characterized by the presence of activated T-lymphocytes at the site of plaque rupture.

These lymphocytes can release various cytokines that activate macrophages and promote smooth muscle cells (Libby 1995). The endothelium has luminal surface areas to which the monocytes can adhere. Two of these areas are intra-cellular adhesion molecule (ICAM) and vascular cell adhesion molecule (VCAM). Once in the sub-endothelial space the monocytes are capable of absorbing and degrading considerable quantities of plasma lipoproteins. The cholesterol portion of LDL-C is esterified to cholesterol oleate, which is either deposited in the cell or expelled into the extra-cellular fluid. Many a receptor systems mediate the specific uptake of lipoprotein cholesterol giving rise to cholesterol deposition in the cell and formation of ‘foam cells’. Special significance is given to the receptors of VLDL-C, malondialdehyde modified LDL-C and dextran sulfate complexed
Experimental observations suggest that by absorptive endocytosis GAGs in the interstitial tissues of the arterial wall may complex with LDL-C and specifically react with the macrophages to form 'foam cells' and aggravate the condition.

Acute coronary syndromes are due to an acute or sub acute primary reduction of myocardial oxygen supply provoked by disruption of such a plaque associated with thrombosis, vasoconstriction and micro-embolisation. Coronary atherosclerosis is not a continuous process but a disease with phases of stability and instability. Sudden unpredictable changes in symptoms appear to be related to plaque disruption. Plaques prone to rupture have a large lipid core, low smooth muscle density, high macrophage density, thin fibrous cap – disorganized collagen and high tissue concentration (Fuster et al, 1995). A macrophage infiltration has been demonstrated consistently in pathological studies as the reason for unstable plaques. The proportion of macrophages is 6-9 times higher in ruptured plaques than stable plaques (Moreno et al, 1994). Plaque disruption may result from various combinations of the following:

- **Active rupture** is related to the secretion of proteolytic enzymes by macrophages, which may weaken the fibrous cap.

- **Passive plaque disruption** is related to physical forces occurring at the weakest point in the fibrous cap, at the junction of the plaque and the adjacent normal wall.

Plaque erosion has also been described as one of the underlying mechanisms of acute coronary syndromes (Burke and Tang 1997). A recent study showed 40% prevalence of plaque erosion in sudden coronary death, 25% in myocardial infarction with a higher prevalence in women than in men. For plaque rupture these figures were 37% in women and 18% in men (Arbustini et al 1999). When erosion occurs, thrombus adheres to the surface of the plaque whereas when the plaque ruptures, thrombus involves the deeper layers of the plaque, down to the lipid core. However, when positive remodelling does not accommodate this latter situation, it may contribute to growth and rapid progression of plaque (Bertrand et al 2000). Thrombosis is induced at the site of plaque rupture or erosion. It may lead to rapid changes in stenosis severity, and may result in subtotal or total vessel occlusion. The lipid rich core, which is exposed after plaque rupture, is highly thrombogenic and has a greater concentration of tissue factor than the other components.
of the plaque (Toschi et al 1997). There is also a strong correlation between tissue factor activity and presence of macrophages (Moreno et al 1994). Thrombosis at the site of plaque rupture may fragment into small pieces, which may migrate downstream and may occlude arterioles and capillaries. These platelet emboli may cause small areas of necrosis (small infarcts) in the absence of occlusion of epicardial coronary artery. The platelet rich thrombus can also release vasoconstrictive substances such as serotonin and thromboxane A$_2$ (Willerson et al 1997) that may induce vasoconstriction at the site of plaque rupture or in the microcirculation. This causes episodes of acute transmural ischemia that is provoked by localized coronary vasospasms which severely constricts or occludes one or more large epicardial coronary vessels characterized by transient angina.

A review on the UK based South Asian states that traditional risk factors of CVD such as hypertension, hypercholesterolemia and cigarette smoking alone cannot account for the increased mortality rates observed in South Asians, since the prevalence of factors are no higher that those for Caucasians (Lovegrove 2007). Although the prevalence of generalised obesity is lower in most South Asians than in the general population, higher levels of central obesity have been observed in this ethnic group, with 36% of Indian men, 37% of Pakistani and 32% of Bangladeshi considered centrally obese compared with 33% of men in the general population. This finding is even more apparent in Asian women with 30% of Indian, 39% of Pakistani and 50% of Bangladeshi women considered centrally obese compared with only 30% of women from the general population (Department of Health, 2005). In addition to the anthropometric differences between the ethnic groups, numerous studies have reported higher levels of plasma TAG, insulin resistance, C-reactive protein, plasminogen activator inhibitor-1 and lipoprotein (a) and lower levels of HDL-cholesterol for ethnic groups living in the UK who originate from the Indian subcontinent when compared with white Caucasians. These measures are all characteristics of the metabolic syndrome that may contribute to increased CVD mortality rates observed among South Asians (Lovegrove 2007).

Differences in the clustering of metabolic parameters have been shown in different ethnic groups in developing countries. The variable clustering of the different components in the
metabolic syndrome in different populations suggest varying risk clusters associated with the metabolic syndrome (Misra and Khurana 2008). Asian Indian population has been found to be prone to insulin resistance (Vikram et al 2006). The connection between insulin resistance, hyperinsulinemia and CHD has been established by several transverse, prospective, and experimental studies (Fontbonne et al 1989, Hargreaves et al 1992). Recent data from India showed that one fourth to one third of urban population of India has the metabolic syndrome placing them at a higher risk of developing cardiovascular anomalies. Furthermore, the prevalence is 1.5–2 times higher in women compared with men (Misra et al 2006). The MRFIT cohort studied the combined effect of blood pressure and cholesterol on CVD risk. The trial revealed as the cholesterol and blood pressure levels increased, so did the risk of CVD. The increase in risk was found to be continuous and additive (Stamler et al 1993).

**Diabetes Mellitus**

Diabetes Mellitus is a complex metabolic disorder that involves numerous biochemical abnormalities, a heterogeneous clinical picture and a polygenic heredity component. The existence of the disorder has been known since 600 BC when ‘Susrutha’ an Indian physician described it as an illness characterized by excessive thirst and excessive urination which was also sweet to taste. The term describes a group of metabolic disorders characterised by hyperglycemia resulting from aberrations in insulin secretion, insulin action or both (ADA 2002). The aetiological classification of the disease reflects the defect or the process that may lead to the identification of diabetes at any stage of the disease, even at normoglycemia. The following is the classification given by WHO

1. **Type 1 Diabetes** (Figure 17)
   
   β – cell destruction leading to absolute insulin deficiency.

2. **Type 2 Diabetes** (Figure 18)
   
   Ranges from insulin resistance with relative insulin deficiency, to predominantly a secretory defect with insulin resistance.
3. Other Specific types

Like Genetic defects of β cell function, Genetic defects of insulin action, Endocrinopathies, Infections, Drug/Chemical induced, Immune Mediated, etc.

4. Gestational diabetes Mellitus (GDM)

Clinical expression of type 2 diabetes involves interaction of a variety of factors (Figure 19) which include genetic, environmental, and hormonal factors (Defronzo et al 1992). Insulin resistance is defined as a state of reduced responsiveness to normal circulating levels of insulin and has been recognised as a characteristic of type 2 diabetes (Saltiel 2000). Since insulin resistance precedes the occurrence of the disease, it has been considered a good predictor for development of diabetes (Haffner et al 2000). The commonly associated risk factors of insulin resistance are obesity especially abdominal obesity, sedentary lifestyle and lack of physical activity (Bharadwaj et al 2008).

Individuals ‘at risk’ of type 2 diabetes show various forms of pancreatic β cell dysfunction. Both insulin secretion and insulin sensitivity are affected. These aberrations result in increased blood glucose levels termed as hyperglycemia. An increased glycemic load can eventually lead to failure of β cells of the pancreas, this is through the direct detrimental effect of hyperglycemia on the β cells, which is commonly referred to as ‘glucotoxicity’. Mechanisms include decrease in expression of relevant genes (insulin receptor, GLUT2, glucokinase, inward rectifier potassium channel), beta cell de-differentiation and increased apoptosis. Also, it has been suggested that chronic hyperglycemia per se can worsen insulin resistance (Tataranni 2002).

Insulin is a potent anabolic hormone and is essential for maintenance of body glucose homeostasis. It regulates body glucose levels at multiple sites by reducing hepatic glucose output (via decreased gluconeogenesis and glucogenolysis) and increasing the rate of glucose uptake primarily into the muscle and adipose tissues. Insulin also profoundly influences the lipid metabolism by increasing synthesis of lipids in the liver and fat cells, and attenuating fatty acid release from triglycerides in fat and muscles (Virakamani et al 1999, Pessin and Salteil 2000).
Figure 17: Type 1 Diabetes.

Type 1 Diabetes Mellitus
(Absolute Insulin deficiency)

Symptoms: Hyperglycemia, Polyuria, Polydipsia, Significant Weight Loss, Electrolyte Disturbances

Ketoacidosis
Macrovascular diseases
(Coronary artery diseases, Peripheral vascular disease, Cerebrovascular disease)

Retinopathy
Nephropathy
Neuropathy

Medical Management

Nutritional Management

Idiopathic

Circulating Auto-antibodies

Immune mediated (Autoimmunity)
Viral infection, toxins, etc.

Figure 18: Type 2 Diabetes.

Type 2 Diabetes Mellitus

Symptoms: Hyperglycemia, Polyuria, Polydipsia, Significant Weight Loss, Electrolyte Disturbances

1. Abnormal pattern of insulin secretion and action
2. Decreased cellular uptake of glucose and increased PP glucose
3. Increased release of glucose by liver (gluconeogenesis)

Medical Management

Nutritional Management

• Diagnosis
• Medication
(Sulfonylureas, biguanides, insulin, etc.)
• Regular Blood Glucose monitoring
(glycated hemoglobin, microalbuminuria, etc.)

• Lifestyle strategies
(food & physical activity)
• Nutrition Health Education
• Energy restriction - Weight loss
• Regular Blood Glucose monitoring -
Required adjustments in diet & drugs
Insulin action is initiated through the binding and activation of its cell surface receptor, which consists of two α subunits and two β subunits that are disulfide into a \(\alpha_2\beta_2\) heterotetrameric complex. Following insulin binding, the receptor undergoes autophosphorylation on multiple tyrosine residues, resulting in activation of receptor kinase and tyrosine phosphorylation of family insulin receptor substrate (IRS) proteins. The IRS proteins bind differently to the various downstream signalling proteins transmitting the signal from the receptor to the final cellular effect (Virakamani et al. 1999, Pessin and Salteil 2000). A defect anywhere along this pathway can lead to the development of type 2 diabetes characterized by deterioration of glucose tolerance (Weyer et al. 2001).

The relative roles of peripheral insulin sensitivity and diminished β cell dysfunction in the early pathogenesis of the disorder is however controversial. It has been suggested that reduced insulin sensitivity precedes development of type 2 diabetes. The pathogenesis of type 2 diabetes is therefore a consequence of a dynamic relationship between insulin resistance, resultant hyperinsulinemia and impaired insulin secretion (Saad et al. 1991, Gerich 1998, Haffner et al. 2000, Weyer et al. 2000). Figure 20 gives a comprehensive picture of the role of insulin in the pathogenesis of the disorder and also development of common symptoms. These basic defects in the early stages of the disorder coupled with insulin deficiency in the later stages together with the reciprocal changes in Glucagon affects the carbohydrate, fat and protein metabolism (Cavaghan et al. 2000, Weyer et al. 2000).

Aberrations in insulin activity results in reduced activity of glycolytic enzymes like glucokinase (liver), hexokinase (muscle and adipose tissue), phosphofructokinase, and pyruvate kinase (Cavaghan et al. 2000). There is a reduction in the insulin mediated uptake of glucose from muscles and other tissues and an increase in glucose production from the liver resulting in post prandial hyperglycemia followed by fasting hyperglycemia. There is a manifold rise in the activity of phosphorenoisopyruvate carboxykinase (hepatic gluconeogenic enzyme) leading to increased gluconeogenesis. Muscle protein is broken down to release amino acids and there is an increased FFA production from the adipose tissues.
Figure 19: Pathophysiology of Type 2 Diabetes

Defronzo et al 1992
There is an elevated influx of FFA to the liver, which continues gluconeogenesis, causing an increased synthesis of triglycerides, while accumulating fatty acids (Anderson 1988). Thus, lipid abnormalities are fairly common among diabetics (Mani and Mani 1988, Georg and Ludvik 2000).

**Hypertension and Diabetes**

The pathogenesis of hypertension that accompanies diabetics with or without renal disease has features similar to non diabetic patients with essential hypertension. However, it is observed with twice the frequency in diabetic population. Hypertension is
more prevalent in obese NIDDM patients. Hyperinsulinemia and insulin resistance have a key role in the precipitation of peripheral vascular resistance and hypertension. The proposed mechanism involves; increased renal sodium and water reabsorption, increased blood pressure sensitivity to dietary salt, augmentation of the pressure and aldosterone to angiotensin II, change in trans-membrane electrolyte transport, stimulation of sympathetic nervous activity, reduced synthesis of vasodialatory prostaglandins and increased secretion of endothilin. Good glycemic control and tight control of blood pressure is vital in preventing or postponing micro and macroangiopathy in various target organs.

**Dyslipidemia and Diabetes**

Insulin resistance and hyperglycemia are known to affect each and every lipid and lipoprotein fraction (Georg and Ludvik 2000). Insulin normally inhibits hormone-sensitive lipase in adipose tissue; insulin resistance therefore causes unrestrained lipolysis leading to increased influx of fatty acids, which results in hepatic triglyceride synthesis. The United Kingdom Prospective Diabetes Study (UKPDS) has shown that hypertriglyceridemia is present in diabetics at the time of diagnosis and has been noted in the prediabetic phase as well.

On the other hand, endothelial insulin dependent lipoprotein lipases reduced resulting in diminished triglyceride clearance from triglyceride rich lipoproteins. Large population based studies have reported a 50 - 100% contribution of type 2 diabetes to the elevations in plasma VLDL and total triglyceride (Mani and Mani 1988, Wolffensteinntael and Drzewoski 1999, Georg and Ludvik 2000). The direct effect of insulin on hepatic VLDL triglyceride synthesis appears to depend on the duration of exposure. Chronic hyperinsulinemia increases VLDL triglyceride secretion from the liver (Wiggins and Gibbons 1992). As a consequence of triglyceride enrichment of VLDL, there is an increased transfer of triglyceride to LDL with the help of CETP. Triglyceride rich LDL particles are substrates for hepatic lipase activity leading to the formation of small dense
LDL. Smaller and denser LDL\textsubscript{3} particle have a greater atherogenic potential (Fuller et al 1979).

In a multivariate analysis, male diabetics had lower HDL- C levels than the corresponding non – diabetic subjects after adjusting for other variables (UKPDS). HDL particles show enrichment in triglyceride, increased cholesterol to protein ratio and a selective reduction of Apo A\textsubscript{1}. There is also increased glycosylation of Apo I and Apo II, which appears to accelerate HDL catabolism. There is rapid clearance before they have circulated long enough to acquire sufficient cholesterol to become HDL\textsubscript{2}. Glycosylation of HDL – C probably also impairs its ability to promote cholesterol efflux from cells in vitro. The low HDL persists despite achievement of glycemic control.

**Diabetes and its complications**

To understand the essence of aetiopathogenic mechanisms, which are at the root of diabetic complications development was an essential challenge to modern medical science and practice. Diabetic micro and macroangiopathy are now considered to be polyaetiological multifactorial diseases where persistent hyperglycemia plays the leading role (Bayens 1991, Brownlee 1994, Cosentino and Luscher 1998, King et al 1994). On the other hand, it contributes to the origin of oxidative stress. Along with the others, endogenous and exogenous factors play an important role in pathogenesis of secondary complications of diabetes. Hence, diabetics are exposed to continuously increasing oxidative stress caused by the prolonged hyperglycemia and conditioned by different pathophysiological processes (Figure 21). In state of chronic hyperglycemia, non-enzymatic glycosylation of proteins sets in. The combining of glucose with plasma proteins is completed during the Mailard reaction. In glycosylation it reacts by lateral groups (lying under and over the peptide connection plane), to amino acids’ remains, as well as by amino acids’ remains in C- and N end of the protein molecule until formation of Schiff bases. They are unstable and a few days later they are transformed into stable ketoamine called Amadori product (Figure 21).
Figure 21: Schematic representation: hyperglycemia, oxidative stress and vascular dysfunction

AGEs – Advanced Glycosylation End products; TXA2 – Thromboxane A2; TNF-α - Tumour Necrotising Factor – α; NO – Nitric Oxide
(Goycheva 2006)
The brief period of half life of most cellular and plasma proteins does not provide the possibility for Amadori products to transform further. By contrast, proteins with long half life, a part of Amadori products, undergo partial oxidative degradation and carboxymethyllysine. The rest are included into series of intermediate and subsequent Maillard reactions until formation of pigmented, fluorescent and containing cross-links "advanced Maillard products" also called "advanced glycosylation end products" (AGEP). They can be determined as a class of heterogeneous compounds of monosaccharides and proteins, obtained by consecutive reactions of dehydration, condensation, fragmentation, oxidation and cyclization (Brownlee 1994, Koya and King 1998, Ruderman et al 1992, Goycheva 2006). This produces a combination of glucose with plasma protein free radicals. Together with the transformed proteins they contribute to the intensification of oxidative stress and vessel injury.

Reactions with proteins, glycosylation glucose and auto-oxidation all occur simultaneously. Reactive metabolites of oxygen and ketoaldehydes are formed. The latter can interact with still unchanged proteins and with Amadori products. In this way the consequences of glucose auto-oxidation are intensified. Glucose auto-oxidation products can attach to specific receptors from the surface of endothelial cells and change their properties. For instance, their combining with nuclear factor kappa-B (NF-κB) stimulates the synthesis of atherogenic circulating adhesive molecules and inflammatory cytokines (e.g. Tumor necrotizing factor α, TNF-α). They regulate cellular growth, proliferation and migration and have a very important role for early formation of atherosclerotic lesions.

Hyperglycemia in non-insulin dependent cells activate aldose reductases enzyme, which leads to metabolization of glucose into sorbitol and fructose. It reduces the proportion of NADFN/NADF⁺ and increases the proportion of NADN/NAD⁺. The trouble in the oxidation of NADN in the respiratory chain is indicated as "hyperglycemic pseudo hypoxia" and leads to increase in the quantity of reactive oxygen species (ROS) in the cells. Increased formation of ROS is reinforced hypoxia related to vessel complications in diabetes mellitus, which has already occurred. Reinforced formation of ROS in the
conditions of pseudo and real hypoxia could be connected with activation of protein kinase C – a key enzyme in transmission of signals. The inclusion of sorbitol way increases de novo diacylglycerols synthesis which is a cause for activation of protein kinase C in endothelial cells. Protein kinase C phosphorylates and thus activates phospholipase A. It releases arachidonic acid from membrane phospholipids as at the same time the quantity of superoxide radicals and prostanoides is increased (King et al 1994).

Clinical complications accompanying protein kinase C activation are expressed by increasing endothelium permeability, vasodilator imbalances, disturbed vascular stream and intensified synthesis of basal membrane proteins (Koya and King 1998). The three basic consequences of hyperglycemia are the formation of “final products of advanced glycosylation”, glucose autooxidation and sorbitol increase. All these contribute to the rise and reinforcement of oxidative stress in diabetes mellitus. The results of clinical studies published recently (DCCT, UKPDS) confirm the hypothesis of “glucotoxicity” and long-lasting hyperglycemia or postprandial glucose variations. These two factors, in conjunction with the oxidative stress caused by them, are at the root of the greater part of vascular complications in diabetes mellitus (Baynes 1991).

**Basic functions of endothelium**

Endothelium separates not only the vessel wall from the blood stream, but also possesses its own metabolism. Endothelial cells synthesize factors, which can stimulate them reversibly and maintain their activity for a long time. One of the most important endothelium functions is providing suitable vessel tone. It is achieved by balancing synthesis and release of vasodilators and constrictors. The basic factor stimulating the elimination of vasodilators such as nitric oxide, prostacyclin (PGI₂) and hyperpolarizing factor of endothelial origin (EDNF) is the endothelium irritation by the blood stream. The well-known vasoconstrictors are endothelin – 1, prostaglandin F₂α (PGF₂α) and thromboxane A₂ (TXA₂) (derived from arachidonic acid). Superoxide anion and angiotensin II constrict blood vessels. In the endothelium, oxidation of plasma lipids,
synthesis of angiotensin II, breakdown of catecholamines and kinins circulating in the blood are accomplished. Endothelium also regulates the proliferation of flat-muscular cells of the vessels and the adhesion of leucocytes (granulocytes, monocytes) and thrombocytes. Besides, it modulates vessel permeability and influences inflammatory processes. Endothelium possesses antithrombotic and fibrinolytic properties (Haller 1997).

**Endothelial dysfunction**

In some pathological conditions and diseases like diabetes mellitus, the above mentioned properties of endothelium change. By the term “endothelial dysfunction” means the disturbed balance between vasodilators and vasoconstrictors, thrombotic and fibrinolytic mediators, as well as growth retaining and stimulating substances. The increased vascular tone is responsible for the increased vessel permeability. The loss of antithrombotic and fibrinolytic properties of endothelial cells causes local thrombosis. The imbalance between prostacyclin (PGI$_2$) and thromboxane A$_2$ (TXA$_2$) leads to accelerated aggregation of thrombocytes (Haller 1997).

Endothelial dysfunction is manifested by increased expression of adhesion molecules (intracellular adhesive molecules ICAM-1, VCAM-1, E-selectin) on the cells surface. It intensifies the interaction between them, following which blood-forming elements adhesion and extra-vassal migration of leucocytes occurs. The activation of endothelial cells increases the proteins secretion of intercellular matrix. These endothelial disturbances can vary depending on the type of lesion and its localization. A contemporary clinical observation shows that the earliest vessel changes include increased vasodilatation and corresponding increase in blood flow. Consentino *et al* (1998) established that prolonged action of high glucose concentrations intensifies synthesis of nitric oxide and superoxide anions in human aorta. These two free radicals interact and form peroxynitrate.

Hyperglycemia leads to increased inactivation of nitric oxide on one hand and secretion of powerful vasoconstrictors on the other. Release of arachidonic acid from membrane
phospholipids is stimulated – synthesis of vasoconstrictors prostaglandins – PGF$_2$α and TXA$_2$ is reinforced resulting in endothelial dysfunction. These observations confirm the inactivation of nitric oxide in presence of superoxide anions (Guigliano et al 1996). Intensified formation of prostaglandin H$_2$ (PGH$_2$), TXA$_2$ stimulates thrombocytes aggregation. Thus, supraphysiological levels of plasma glucose alter the endothelium’s anticoagulant properties, antithrombin 3, synthesis of heparin sulphate and release of tissue plasminogenic activator decreases. The followed activation of coagulation cascade increases the quantity of endothelial activators – thrombin and fibrin (Stehouwer et al 1997). Oxidative stress acts upon endothelium expression of growth factors from its adjacent cells. The transforming growth factor β (TGF-β) secreted by mesangealial cells and growth factor of vessel endothelium (VEGF) stimulate its cells proliferation and suppress their ability of regeneration after damage.

Hyperglycemia effects are reinforced by ROS formed in the course of non-enzymatic glycosylation of proteins and glucose auto-oxidation occurring simultaneously (Brownlee 1994). “Final products of advanced glycosylation” activate endothelial cells by attaching to specific receptors on their surface. In these conditions endothelial permeability increases, accumulation of adhesive molecules deepens and synthesis of interleukins (IL-1, IL-6) and tumour necrotizing factor α (TNF-α) is reinforced (Frostegard et al 1991). Previously, it was considered that osmotic pressure of sorbitol was the cause for vessel damage in diabetes. Currently, it is known that nitric oxide in norm activates guanylyclase, which acts as a second mediator and activates protein kinase C. It phosphorylates and activates phospholipase A$_2$. Secretion of arachidonic acid from membrane phospholipids and its metabolism to vasoactive prostanoids is intensified. In conditions of hyperglycemia, however, because of the intensified endothelial transformation of glucose into sorbitol and fructose, a decrease of NADFN quantity is observed and nitric oxide obtained from arginine is compromised. The examined clinical disturbances are expressed by increase in endothelium permeability, vasodilator troubles, damaged vessel stream and reinforced protein synthesis of basal membrane (King et al 1994).
Typically, basement membranes are cell anchored polymers of laminins stabilized by type IV collagen networks, fibronectin, vitronectin and several proteoglycans that form the extracellular structure of tissues and it is influenced by ambient glucose concentration. The link between hyperglycemia and tissue damage is due to the formation and accumulation of AGE products in the tissues. An increase in ECM proteins mainly type IV collagen, laminin and fibronectin, reduces elasticity and alters the filtration properties of the basement membrane (Feener and King 1997).

In vitro and vivo studies have demonstrated that AGEP can induce irreversible cross links in the long living matrix proteins like collagen, leading to abnormal structure of collagen matrix (Brownlee 1988). The structural alterations in the ECM proteins are further enhanced by cross-links with plasma proteins. Reactive AGEP can also directly alter tertiary/quaternary structure of these molecules thereby altering their biological activity (Brownlee et al 1984). Cross-linked collagen is less soluble and less susceptible to proteolytic turnover as compared to native collagen and therefore responsible for basement membrane thickening (Fenner and King 1997). Increased collagen cross-linking and thickening of the glomerular basement membrane, skin, tendon and lung connective tissues of diabetics has been reported. These modifications have been implicated in diabetic microangiopathy (Fenner and King 1997, Wautier and Guillausseau 2001).

Vascular disorders are the major long term complications of diabetes that result in morbidity, mortality and reduced quality of life (Shamoon 1996). These vascular disorders can broadly be classified as microvascular (retinopathy, nephropathy, neuropathy), and macrovascular complications (cardiovascular, coronary, peripheral, cerebrovascular diseases).

**Retinopathy**

Retina is characterized by high contents of lipids and increased consumption of oxygen. This makes it particularly susceptible to the influence of reactive oxygen types formed in
conditions of hyperglycemia. It is established that markers of oxidative stress (malondialdehyde, sulfhydryl protein) in sub retinal liquid in patients with proliferative retinopathy are changed significantly in comparison with healthy controls and patients without retinopathy. The first visible lesions are small in diameter and are micro aneurysms arising from the terminal capillaries of the retina. They may either leak or become thrombosed. The consequences of increased vascular permeability are retinal haemorrhages, retinal oedema, either diffused or localized. Retinal haemorrhages may either appear flame shaped due to its location in the nerve fibre layer or dot blot due to its occurrence in the deeper layers of the retina. Absorption of leaking fluid presumably occurs through uptake from the adjacent capillaries (Non –Proliferative Retinopathy).

Chronic localized oedema leads to the deposition of hard exudates which are composed of lipoproteins and lipid filled macrophages usually seen at the junction of the healthy and oedematous retina. The consequence of retinal capillary non perfusion is retinal ischemia in turn causing retinal hypoxia. Effects of retinal hypoxia are arteriovenous shunts, intraretinal microvascular abnormalities and retinal neovascularization (Proliferative Retinopathy). Subsequent fibro-proliferative changes result in retinal traction and detachment and eventually loss of vision. Patients with diabetes are also at a higher risk for other ophthalmic diseases such as cataracts (Neely et al 1998). The development of retinopathy is duration dependent. The risk of retinopathy is ~ 78% with diabetes duration of 15 yrs or more. It has been reported that about one third of the patients develop macular oedema and one sixth develop proliferative disease (Sulochana et al 2001).

Nephropathy

Classic lesions of nodular glomerular sclerosis are associated with proteinuria and hypertension. 50% of those who have had diabetes for over 20 years develop diabetic neuropathy. Final products of advanced glycosylation take a principal position in pathogenesis of diabetic nephropathy. Initially observed kidney hyper perfusion and hyper filtration are connected with activation of phospholipase A₂ by protein kinase C.
Advanced diabetes however, leads to kidney vasoconstriction and increased deposition of extracellular matrix, contributing to systemic hypertension and nephrosclerosis. Normal renal function in diabetics deteriorates in a series of stages ultimately resulting in End Stage Renal Disease (ESRD). The initial stage is asymptomatic and characterized by hyperfunction and hypertrophy of the kidney tissue, glomerular mesangial expansion, glomerulosclerosis and basement membrane thickening. Albuminuria occurs in 7 to 15 years after onset of nephropathy. This is the third stage known as incipient nephropathy where the Glomerular Filtration Rate (GFR) is slightly elevated. A normal or slightly decreased GFR and proteinuria reflects progression to overt nephropathy (Stage 4). The rate of progression is higher in smokers and hypertensives. This stage is irreversible and can only be slowed down by timely intervention. Stage 5 is associated with continuing decline in GFR and increasing blood pressure (Nathan 1993, Nelson et al 1996, Marks and Raskin 1998). Approximately 50 – 70% patients who reach this point eventually progress to ESRD within 10 to 18 years (Nathan 1993, Marks and Raskin 1998).

Neuropathy

Neuropathy is the most frequent symptomatic complication of diabetes and potentially the most devastating. Most neurological complications with diabetes involve the peripheral and autonomic nervous systems. The consequences can extend to several physiological systems. The reported prevalence varies from 5 % to as high as 60 – 100% (Thomas and Tomlinson 1993). Diabetic neuropathy can be classified as Symmetric/Asymmetric, Diffuse/ Focal, Progressive/ Reversible. The pathogenesis is multifactorial and works in unison to cause peripheral nerve degeneration. The mechanisms in play are hyperglycemia, local nerve ischemia and neurotrophic factor deficiency. The diminished neural perfusion and capillary occlusion due probably to thrombosis, oedema of endothelial cells or proliferations connect neuropathy with micro vascular diseases. It is considered that ROS damages neural fibres. ROS are also induced by nerve ischemia. Neuropathic factors are involved in the development, maintenance and regeneration of responsive elements of the nervous system. Deficiency of these factors may decrease the resistance of the cell to injury from oxidative stress.
The most common symptoms include paraesthesia and numbness. Some diabetics also experience burning dysesthesia, depression and anorexia. Autonomic neuropathy can affect gastric or intestinal motility, erectile dysfunction, bladder function, cardiac function and vascular tone. Subclinical changes can often be detected within 5 to 10 years after the onset of diabetes. In a long term follow up of diabetics, electrophysiologic abnormalities in lower limbs increased from 8% at baseline to 42% after 10 years (Partaten et al 1995, Boulton and Malik 1998). Gastroparesis may alter the absorption of meals and glycemic control. Diabetic diarrhoea and incontinence are rare. Impotence is the most common clinical manifestation of autonomic neuropathy, affecting more that 50% of male diabetics (Partanen et al 1995, Boulton and Malik 1998).

**Cardiopathy**

Cardiovascular mortality is twice as high in men and four times as high in women with diabetes as compared to their non diabetic counterpart. Similarly, the relative risk of myocardial infarction is 50% greater in diabetic men and 150% higher in diabetic women. Also, diabetic men have sudden death 50% more and diabetic women 300% more as compared to age matched nondiabetics (Kannel and McGee 1997). Increased prevalence of cardiovascular anomalies in diabetes is due to presence of multiple risk factors. Hyperinsulinemia is associated with increased blood pressure and low HDL – C levels. Both these factors increase the probability of developing cardiovascular aberrations. Patients with impaired glucose tolerance and type 2 diabetes are prone to be hypertensive and have an erroneous lipid profile. Dyslipidemia among them is characterized by lower HDL – C and higher LDL- C (especially small and dense LDL) and triglycerides all of these are considered to be independent risk factors for development of CVD. The level of chronic glycemia as indicated by HbA1c levels may also be an independent risk factor for CVD (Nathan 1993, Garber 1998, Kowalska et al 2001). The Framingham and the Strong Heart study have identified an association between diabetes and increased left ventricular mass, wall thickness, arterial stiffness and reduced ventricular systolic chamber, myocardial infarction and endothelial dysfunction (Devereux et al 2000).
**Glycemic Control – Why and How?**

The acute and chronic complications of diabetes mellitus occur because of poor short term and long term glycemic control. There is now a consensus that these complications are attributable to the accompanying condition of obesity, hypertension and dyslipidemia. In a series of studies spanning over two decades Mani et al (1987 – 2003) have effectively established the importance of glycemic control using different lifestyle counselling (Mani et al 1989, Deshmukh et al 2000) and food based approaches (Rai and Mani 1997, Rai and Mani 1997, Parikh and Mani 2001, Mani et al 2000, Mani et al 1994, Mani et al 1997). Current strategies for optimal care of patients include vigorous and persistent efforts to achieve the following goals;

a) Maintenance of body weight,

b) Maintaining or improving tolerance,

c) Maintaining euglycemia,

d) Maintaining normolipidemia,

e) Ensure adequate intake of nutrients,

f) Allow ample physical activities,

g) Prevention/treatment of complications of diabetes.


Attainment of these goals and optimizing diabetes care requires multi-disciplinary efforts aimed at correcting lifestyle and inducing appropriate health behaviour. Intervention program outcomes depend on the length of follow up after completion of intervention. Intensive lifestyle intervention and a follow up period ≥6 months tend to demonstrate great effectiveness (Mani et al 1989, Deshmukh et al 2000). Mani et al have demonstrated the intake of food with added ingredients like combinations of cereals, pulses, green leafy vegetables, flax seeds, oyster mushroom and spirulina reduce the Glycemic Index of foods. Intake of low GI foods was found to improve glycemic and lipemic responses of diabetic patients.
**Health Promotion**

Health Promotion is the art and science of helping people discover the synergies between their core passions and optimal health, enhancing their motivation to strive for health promotion, and supporting them in changing their lifestyle to move toward a state of optimal health. Optimal health is a dynamic balance of physical, emotional, social, spiritual, and intellectual health. Lifestyle change can be facilitated through a combination of learning experiences that enhance awareness, increase motivation, and build skills and, most important, through the creation of opportunities that open access to environments that make positive health practices the easiest choice (O’Donnell 2009).

The basis for the recent interest can be traced to the confluence of a number of disparate forces. Anderson considered health promotion under the following headings:

1. Growing emphasis on positive health and improved quality of life.
2. People’s greater desire to exercise control over their lives, associated with trends of consumerism.
3. The limited effectiveness of traditional strategies often associated with health education.
4. Recognition that many health problems are related to individual lifestyles.
5. Growing evidence of weak relationship between health care and health care status, especially the poor return on increasing costly resources invested in health.
6. The community development and communications movements, which promote grassroots, in contrast to top-down.
7. The influence of the self care and women’s movements which require a shift of power to individuals and communities.
8. The pressure brought to bear on social programs and high technology medical care by deterioration in economic conditions around the world.
9. Better social, behavioral and educational research on health issues.
The Ottawa Charter (1986) identifies three basic strategies for health promotion. These are advocacy for health to create the essential conditions for health indicated above; enabling all people to achieve their full health potential; and mediating between the different interests in society in the pursuit of health. These strategies are supported by five priority action areas as outlined in the Ottawa Charter for health promotion:

- Build healthy public policy
- Create supportive environments for health
- Strengthen community action for health
- Develop personal skills
- Re-orient health services

The Jakarta Declaration on Leading Health Promotion into the 21st Century (1997) confirmed that these strategies and action areas are relevant for all countries. Furthermore, there is clear evidence that:

- Comprehensive approaches to health development are the most effective. Those that use combinations of the five strategies are more effective than single-track approaches.
- Settings for health offer practical opportunities for the implementation of comprehensive strategies.
- Participation is essential to sustain efforts. People have to be at the centre of health promotion action and decision-making processes for them to be effective.
- Health literacy/health learning fosters participation. Access to education and information is essential to achieving effective participation and the empowerment of people and communities.

For health promotion in the 21st century, the Jakarta Declaration identifies five priorities:

- Promote social responsibility for health
- Increase investments for health development
- Expand partnerships for health promotion
- Increase community capacity and empower the individual
- Secure an infrastructure for health promotion
Planning Health Promotion Program

Planning is a series of decisions, from general and strategic decisions to specific operational details, based on the gathering and analysis of a wide range of information. Planning encompasses a broad field involving a number of different approaches. These include strategic planning, program planning and operational planning. The planning model (Figure 22) is based on 6 key steps.

Step 1: Pre-planning & Project Management
Step 2: Situational Assessment
Step 3: Identify Goals, Populations of Interest and Objectives
Step 4: Identify Strategies, Activities and Resources
Step 5: Develop Indicators
Step 6: Review the Program Plan

Step 1: Pre-planning and project management - Managing the Planning Process
In planning a health promotion project, the planner must manage a number of elements, including meaningful participation of key stakeholders, time, money and other resources, data-gathering and interpretation and decision-making. When these elements are managed well, project outcomes may be greater than expected. If not managed well, problems are likely to occur.

Guidelines for managing pre-planning and project management
To relate these overall project management elements to the key steps involved in the planning process, let’s consider each in turn.

Participation
From the outset, the planner must identify the key stakeholders (these can include the project team, funders, politicians, community partners, and the community of interest themselves). Then the planner must consider roles (who will be informed, make decisions, provide information, or provide hands-on support). A process for participation must be developed as well.
It is important to focus on the process of developing a health promotion project, not only on its end result. This includes:

- Working with people, rather than for them
- Involving stakeholders in project design
- Ensuring participatory evaluation strategies;
- Using techniques of participatory or action research.

**Time**

The time required for each step of the process depends on a number of circumstances and multiple variations exist. In health promotion, because participation is so important, time...
for each step is often long. There are many trade-offs and the ideal level of desired participation can sometimes be in tension with political and organizational considerations, cost, and other deadlines.

**Money and Other Resources**
Planning involves creating an inventory of available resources. This includes allocated budgets, use of staff time, equipment, and space. Other resources to be considered include expertise, contributions in kind from volunteers and partners. Foregoing other opportunities with the organization, partners, and the community at large are also costs. It is essential to be aware of these costs and resources, and keep reviewing this inventory.

**Data Gathering**
Obtaining the information (data) is needed to guide planning efforts. Approaches to data-gathering can depend on:
- Observing health as more than the absence of disease
- Being clear about the role of theory and examining all determinants of health when assessing needs and designing the project/program
- Observing positive directions and capacities of individuals and communities rather than focusing only on problems and deficits
- Looking for ways to collect positive data in all steps

**Role of Theory**
The model or the theory used to collect and interpret data, makes a difference in planning. If a biomedical approach is used, the concern is about the processes of disease and the factors which are physical in nature and usually amenable to medical intervention. For e.g. in heart disease, the focus would be on screening for hypercholesterolemia, high blood pressure, etc.

If behavioural approach is used, then the behaviours of individuals and how those can lead to disease and disability are studied. The strategies for intervention for heart health might include education about the benefits of a low fat diet, a communication campaign on physical activity, programs to encourage people to quit smoking and lobbying the food
industry to provide low fat alternatives. Data would be collected about levels of smoking, physical activity, consumption of fat in the diet, and the presence of stress in the workplace. Goals and objectives would be set in terms of these outcomes as well as the blood cholesterol and pressure measures from the biomedical model.

If a socio-environmental approach is used, the concern is the conditions in the psychosocial, socioeconomic, and physical environments which create conditions for ill-health or wellness. These factors include such determinants of health as housing, peace and security, belonging to a community, adequate income, food, clean air, water and soil, safe working conditions, etc. Health promotion strategies in this model include political advocacy, community development, healthy public policies, and creating supportive environments in addition to developing personal skills. Each model looks for a different kind of information depending on the topic or population of interest and suggests a range of different kinds of strategies. Each model adds its contribution to the other, so that most health promotion programming is a mixture of all three approaches and related-data collection.

**Decision Making**

Decisions have to be made at every step of the planning process. It is important to be aware of who has to be involved in decisions related to each step, who needs to be consulted and who needs to be kept informed. Part of managing the project is to manage the flow of information and options so that decisions are timely and supported. The first key decisions have to do with whether to proceed with the planning for the program, with what timelines, resources and political realities.

**Step 2 : How to conduct a situational assessment**

This step involves gathering of the following information.

**A Gather the perspectives of key stakeholders**

- List individuals and organizations with an interest in this type of project or area of concern
- Describe the views of stakeholders, intended project (Supporters and opposers)
B Examine the Literature & Previous Experience

- Identify previous experience revelations
- Examine the literature for research about projects, communities, and issues related to your priority issue.
- Examine previous evaluation findings of similar projects.
- Review the literature regarding similar types of projects and recommendations for designs
- Needs data

C Collect Health-Related Data about priority Issue

- Demographic data
- Morbidity and mortality rates
- Health behaviour and practices (if available)
- Health status data (including social, economic and environmental indicators)

D Review Existing Mandates

As part of situational assessment, it is both necessary to review existing mandates, to ensure that the proposed project fits well with these.

- The mandate of respective organizations
- Other legislation and regulations
- Policies and guidelines
- Professional standards and ethical guideline.

E Assess Vision

In addition to examining existing mandates, it is also important to look at

- The vision
- The vision of others involved in the planning process
- The vision of the organization
- Desired directions by managers, politicians, community leaders
- Relevant strategic plans
F Complete a PEEST analysis
Identify the political, economic, environmental, social and technological factors that could potentially affect the project (PEEST analysis).

G Identify Information Gaps
Observe the information in hand. If any gaps are present, particularly related to an issue addressed by the project. Identify where to obtain additional information. Based on all of the information collected, identify the factors which would help the project (enabling factors), factors which act as barriers or constraints (limiting factors) and what it is going to take to proceed with planning this project.

Step 3: Setting Goals and Objectives

Setting goals and objectives is critical to developing an evaluation plan and they are important for understanding the theory of the choice to design your program a certain way. In addition, concise, well-written objectives are critical for evaluating the impact of the program.

A goal statement summarizes the ultimate direction or desired achievement of a program. Most health promotion programs have a single goal, although more complex programs may have several goals. Goals provide the framework for program planning; as a result, it is important that they reflect reality in terms of the intended populations of interest. Well stated outcome-oriented goals can provide a set of clear end points around which many strategies or activities can be organized. As the situation changes, the strategies may change but the goals are rarely affected. Clear goals are essential for setting out an evaluation process.

Description of identifying population(s) of interest
Identifying population of interest (key groups) that require special attention to reach the goal. Being clear about this population of interest is an important part of setting clear and specific goals and objectives.
Importance of identifying population(s) of interest

Identifying population of interest is important because theories about what works are different for different populations of interest and can lead to more appropriate strategies.

Objectives

An objective is a brief statement specifying the desired impact, or effect, of a health promotion program (i.e., how much, by whom, when).

Characteristics of good program objectives include specificity, credibility, measurability, continuity, compatibility and freedom from data constraints. The SMART acronym is an easy way to remember the key features of well-crafted program objectives; that is, good objectives are:

S pecific (clear and precise)
M easurable (amenable to evaluation)
A ppropriate (i.e., realistic)
R easonable (i.e., realistic)
T imed (specific time frame provided for achievement of objective)

Step 4: Developing strategies, activities and resources

In this step, the task is to identify the activities that will achieve the objectives. This process involves determining strategies, activities and resources by

A Brainstorm Potential Strategies: Identify project strategies by brainstorming a list of possible health promotion strategies for each of the objectives developed in the previous section. Use the literature to help identify the most effective strategies (if known). Selection strategies that are most appropriate given budget, time, population needs, staff skills, effectiveness etc.

B Select the Best Strategies and Identify Specific Activities: For each objective, create a list of the major strategies, the specific activities for each strategy, who will implement the actions, and related potential indicator.
C Review Current Activities: Review the program activities currently offered in this area and identify those activities which are to be continued, those which should be dropped, those that need to be changed and those which are new. This will help everyone involved reassign priorities among existing and new program activities.

D Assess Resources
- Review the resources (financial and human) required to implement the plan.
- Review the resources currently and examine the gaps between what is needed and what is there.
- Explore ways of obtaining the required resources from other organizations
- Which parts of the plan are going to be retained and which parts will be on hold until new resources are found.

Step 5: Developing indicators

Step 5 involves developing measurable indicators associated with each objective and strategy. There can be more than one indicator linked to an objective or strategy and often these indicators are measures of parts of goals and objectives that cannot be directly measured. This step is important because it indicates a real commitment to achieving results and measuring this achievement. It is a critical step towards developing an evaluation plan for the program like participation etc. Depending on long term or short term objectives the indicators too can be long or short termed.

Step 6: Review the program plan

This step involves summarizing the plan in a logic model and reviewing it for the logical relationships between goals, population(s) of interest, objectives, strategies and activities. This review of the plan also considers the overall context for the plan and the resources required to implement it. Program logic models clarify objectives, show linkages between elements and clarify appropriate measures—this forms a kind of “logic check”. It is an important step in preparing for evaluation. Reviewing the whole plan also gives the
planning team an opportunity to examine the connections with other planning activities and take a realistic look at feasibility.

**Step 7 and 8: Implementation and Results**

The program is implemented as per the plan and constantly evaluated. Decision making and evaluation is a continuous part of the process. Once implemented the results obtained must be studied in the light of objectives and goals set to evaluate the program.

**Evaluation**

Evaluation is the systematic examination and assessment of features of a program or other intervention in order to produce knowledge that different stakeholders can use for a variety of processes. The process includes

1. Clarifying the initiatives mandate; aims, objectives, initiatives, procedures and structures.
2. Identifying the issues and questions of concern.
3. Obtaining the required information on measurement methods, evaluator and data collected.
4. Collecting of data using methods and procedures agreed on.
5. Analyzing and evaluating data.
6. Making recommendations to the stake holders and clarifying short and long term implications of the findings. Identifying costs and benefits of implementations.
7. Disseminating the findings agencies and stake holders and funding agencies and other stake holders.
OTHER PLANNING MODELS

There are a number of planning models that are also very useful to health promotion practitioners.

- Strategic Planning Process (Bryson, 1995)
- Precede-Proceed (Green and Kreuter, 1999)

Strategic Planning Process (Bryson)

Bryson's model focuses specifically on planning in the public sector. It is especially useful for developing mission statements. It states that the gap between the goals and objectives of public sector programs and the results observed in the population cannot be directly attributed to those programs.

The Precede-Proceed Model (Green and Kreuter)

This model is valuable to health promotion planning because it provides a format for identifying factors related to health problems, behaviours and program implementation (Figure 23). Three categories of factors that contribute to health behaviour are described in this model. They include:

*Predisposing factors (P)* - Forces that motivate an individual or group to take action such as knowledge, beliefs, attitudes, values, cultural norms, etc. The key consideration in understanding predisposing factors is the extent to which behaviour can be predicted.

*Enabling factors (E)* - Include both new personal skills and available resources needed to perform a behaviour. The key consideration for these factors and health behaviour is the extent to which their absence will prevent an action from happening.

*Reinforcing factors (R)* - Provide an incentive for health behaviours and outcomes to be maintained. To understand their importance, we must know the extent to which their absence would mean a loss of support for current actions of an individual or group.
An understanding of these three factors allows us to identify priorities and provides a basis for where to focus efforts. This is a behaviourally-oriented model which does not put much emphasis on the socio-environmental conditions for health outside of their relationship to creating behaviour change. The model also tends to be problem-oriented rather than oriented towards creating positive health outcomes. The model is useful in that it can be adapted so that each category of factors includes socio-environmental conditions and an emphasis on looking for positive factors (strengths and assets).

**Needs/Impact-Based Planning Model (Metro Toronto DHC)**

The Needs/Impact-Based Planning Model (Figure 24) is a systematic approach to health promotion planning developed by Metro Toronto District Health Council. The model sets priorities based on identified needs, potential strategies to address these needs, and the feasibility of the potential strategies.

**Strengths of the model include:**

- It considers values, ethics and other factors influencing decision making.
- It provides a logical and systematic way to make planning and resource allocation decisions.
- The model was developed and applied in Ontario and is recommended for use by the Ontario Ministry of Health.
- The model includes Determinants of Health as indicators of health need.
- Evaluation is inherent to the model.

**Limitations of the model include:**

- It requires hardware and software packages to implement.
- Bringing the necessary stakeholders across the health continuum can be challenging.
- The utility of the method will be influenced by the size of the information. Collection system needed to support the method and the need for qualitative and quantitative research where sufficient information does not exist.
PHASE 1
SOCIAL DIAGNOSIS

PHASE 2
EPIDEMIOLOGICAL DIAGNOSIS

PHASE 3
BEHAVIOURAL & ENVIRONMENTAL DIAGNOSIS

PHASE 4
EDUCATIONAL & ORGANIZATIONAL DIAGNOSIS

PHASE 5
ADMINISTRATIVE AND POLICY DIAGNOSIS

PHASE 6
IMPLEMENTATION

PHASE 7
PROCESS EVALUATION

PHASE 8
IMPACT EVALUATION

PHASE 9
OUTCOME EVALUATION

Figure 23: Precede-Proceed Model (Green and Kreuter, 1999)
2. Set targets for implementation
3. Identify barriers to implementation

**Figure 24: Needs/Impact-Based Planning Model (Metro Toronto District Health Council, 1996)**

- **DETERMINE THE NEEDS**
  - Identify issues with greatest impact on health using defined Health Status Scores
  - RESEARCH

- **EVALUATE IMPACT**
  - Evaluate strategies
  - Action & Advocacy
    - 1. Implement
    - 2. Advocate for implementation where appropriate
  - DETERMINE PRIORITIES
    - 1. Rank the identified health issues on the basis of the evaluation
    - 2. Set targets for implementation
    - 3. Identify barriers to implementation

- **POTENTIAL AND CURRENT STRATEGIES**
  - 1. Identify potentially feasible interventions
  - 2. Estimate impact on identified issues
  - RESEARCH

- **EVALUATE STRATEGIES**
  - Determine efficacy, effectiveness, efficiency, availability and appropriateness
  - RESEARCH
Practice

The cardinal principle of Health Promotion is ‘empowerment’. It is about ensuring that individuals and communities are able to assume the power they are entitled to. Thus, the primary criterion for determining whether a particular initiative would be considered health promoting, is determined by the extent to which the process enables the target audience. The absence of empowering activities signals that the intervention does not fall into the rubric of health promotion. Attempts to encourage public participation are critical to the process of health promotion.

Health practitioners have applied the label of health promotion to the following seven different types of activities;

1. Preventive services (immunization, hypertension survey, tobacco control, etc.)
2. Preventive health education (efforts to influence lifestyle and to increase use of preventive services)
3. Positive health protection (Fluoridation of water)
4. Health education for preventive health protection (lobbying for seat belt legislation)
5. Positive health education (influencing positive health behaviour on positive health grounds, including encouraging productive use of leisure time and helping people develop health-related life skills)
6. Positive health protection (implementation of workplace antismoking policies and provision of leisure facilities)
7. Health education for positive health protection (obtaining support for positive health promotion measure)

Settings

Over the past decade ‘Settings based Health Promotion’ has become a central feature of efforts to promote health. The approach is built on the profound belief in its value and
deployed range of novel theoretical resources from organisational sociology and psychology (Whitelaw et al 2001). The conceptual development, practical use, formal evaluation and reporting of setting based activity appears to have been largely constructive and positive in nature. A range of WHO initiatives like the Ottawa Charter (1996), Jakarta Charter (1995) have identified social and cultural environments as central features and accepting the need for a 'conceptual framework', settings work is sought to draw upon theoretical bases that had been traditionally under-represented in health promotion.

The re-orientation points towards a theoretical shift in emphasis from individual health problems and topic based 'risk factors' to the nature of 'the system' and 'the organization' as a relatively complex phenomena. A number of key factors are accepted as central to such an activity which include personal competencies, reshape environments, build partnerships and bring about sustainable change through participation and developing empowerment. There is also a need to develop new skills and a shift in the potential of traditional medical expert to a 'change facilitator'. Review of literature reveals that this approach has delivered many successful outcomes including;

i. Development of Health Promotion Policies and dedicated health promotion budgets
ii. Improvements in structural and Pschycosocial environments
iii. Frequent and better partnerships
iv. Development of discrete health promotion/education projects
v. Changes in various individual attributes, behaviours and functioning
vi. Economic benefits

There are although there are significant differences in many aspects of this base, for example at the descriptive level the scale of levels can differ from nation approaches to highly localized communities, the subsequent location of work (Schools, colleges, communities, workplace), and diversity of outcomes based on these. The respective emphasis of each component of activity vary ranging from broad policy to environmentally oriented work through individualistic participation.
A proposed ambitious comprehensive model (Table 13) uses the notion of the setting as an entity above the individuals in it and seeks to bring about direct and relatively significant changes in setting structure and culture with an assumption that individuals are relatively powerful to precipitate change to any significant level. The emphasis tends to be more on broad setting policies and strategies, with the focus on the direct action on senior staff.

The difficulties or barriers in translating theory to practical and tangible activities in the settings are

1. 'Competing' forces within the settings
2. Internal pressure to produce deliverable outputs that are identifiable as health promotion
3. Problems associated with status of health promoters as credible agents of change
4. Limitation is providing sufficient support for change (financial, time, training, expertise/consultancy and other resources.

To conclude the working group (WHO) has evolved a few principles which stated that health promotion is an evolving field and can make a major contribution to practice, but it suffers from a shortage of evidence and on the effectiveness of initiatives. It involves a wide range of approaches and models, offering legitimate roles for both qualitative and quantitative methodologies.

It employs a wide range of social science disciplines and approaches and builds on a range of planning models. Lastly, it requires theory and other conceptualizations to be effective and offers many potential roles to evaluators and researchers. As suggested by the discussion, a generic model of a health promotion program is given in Figure 25. The planning of health promotion program should reflect the conceptual framework that underlies the understanding of how one might influence these.
Table 13: Settings based Health Promotion.

<table>
<thead>
<tr>
<th>Type</th>
<th>Core perspective/Analysis of Problem-solution</th>
<th>Relationship between the health promotion and the setting</th>
<th>Practical focus of activity</th>
<th>Indicative Contributory disciplines</th>
<th>Indicators</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passive model</td>
<td>The problem and solution Rest within the behaviour and actions of individuals</td>
<td>Setting is passive; only provides access to participants and medium for intervention; health promotion occurs in setting independent of settings features</td>
<td>Mass media and communication, individual education</td>
<td>Educational/traditional psychological focus</td>
<td>Traditional individual indicators (e.g. knowledge, attitude, behaviour)</td>
</tr>
<tr>
<td>Active model</td>
<td>The problem lies within the behaviour of individuals, some of the solution lies in the setting</td>
<td>Setting provides ‘active’ and comprehensive resources to fulfil health promotion goals; health promotion utilizes setting resources</td>
<td>Mass media and communication individual education plus complimentary work on policy development and structural change around the specific topic area</td>
<td>Primary educational/traditional psychological focus; complimentary focus on political and organizational science, policy studies</td>
<td>As above as end outcomes plus process assessment of wider setting contributions</td>
</tr>
<tr>
<td>Vehicle model</td>
<td>The problem lies within the setting, the solution in learning from individually based projects</td>
<td>Health promotion initiatives provide an appropriate means for highlighting the need for broader setting development health promotion seen as a vehicle for setting change</td>
<td>Principle focus on developing policies and bringing about structural change using feeder activity from mass media and communication, individual education strengthening collective. Facilitating and community action</td>
<td>Primary political and organizational science, policy studies; complimentary focus on psychological focus educational/traditional</td>
<td>A mix of project and contextual indicators (interest particularly in the interaction and association between discrete projects and broader development)</td>
</tr>
<tr>
<td>Organic model</td>
<td>The problem lies within the setting, the solution in the actions of individuals</td>
<td>Organic setting processes involving communication and participation are inherently linked to health and are thus ‘health promoting’</td>
<td>Facilitating and strengthening collective community action</td>
<td>Sociology, anthropology, principles of community development</td>
<td>Organic setting indicators eg. levels of communication and participation; degree of staff development, etc.) Over-arching setting ‘development’ indicators impact (e.g. policy and environmental)</td>
</tr>
<tr>
<td>Comprehensive/Structural model</td>
<td>The problem and the solution lie in the setting</td>
<td>Broad setting structures and cultures inherently linked to health and are thus ‘health promoting’, health promotion as central component of comprehensive setting development</td>
<td>Focus on developing policies and bringing about structural change</td>
<td>Political and organizational science, policy studies</td>
<td></td>
</tr>
</tbody>
</table>
Dietary Interventions

Past few decades have witnessed nutrition coming to the fore as a major modifiable determinant of chronic degenerative diseases (WHO 2003). The macronutrients of diet are carbohydrates, protein and fats. Imbalances of these macronutrients are one of the principle reasons for precipitation of NCDs. Excessive energy intake is one of the primary factors for development of obesity (Desai and Mani 2002). Asian and South Asian diets are prone to develop insulin resistance and metabolic syndrome. Indian diets especially are low in MUFA, n 3 PUFA, and fibre and high in SFA (Misra et al 2008). It has been suggested that foods with a high glycemic index are detrimental to health. Several epidemiologic studies found that diets rich in whole grains may protect against cardiovascular disease, stroke, type 2 diabetes, and certain cancers (Pereira et al 2002).

Low glycemic index diets have been shown to lower urinary C-peptide excretion in healthy subjects, improve glycemic control in diabetic subjects, and reduce serum lipids in hyperlipidemic subjects. Furthermore, consumption of low glycemic index diets has been associated with higher HDL-cholesterol concentrations and, in large cohort studies, with decreased risk of developing diabetes and cardiovascular disease. Case-control studies have also shown positive associations between dietary glycemic index and the risk of colon and breast cancers. Despite inconsistencies in the data, sufficient positive findings have emerged to suggest that the dietary glycemic index is of potential importance in the treatment and prevention of chronic diseases (Jenkins et al 2002).

The protective effects of whole grains may depend on the presence or interaction of several biologically active constituents, including dietary fibre, vitamin E, magnesium, folate, other nutrients and non-nutrients. Dietary fibre has been shown to decrease glucose, insulin, and serum lipid concentrations in both diabetic and nondiabetic persons. Increased intakes of whole grains may reduce disease risk by means of favourable effects on metabolic risk factors. Fasting insulin was 10% lower during consumption of the whole-grain than during consumption of the refined-grain diet (Pereira et al 2002).
Figure 25: Planning model for an ideal Health Promotion Program (HPP)

THE FOUNDATION OF HEALTH PROMOTION

(Or) Charter for health promotion

THE VALUES

POSITIVE HEALTH, HOLISTIC HEALTH, SOCIAL JUSTICE, EQUITY, PARTICIPATION

MECHANISM

EMPOWERMENT ENHANCING CAPABILITIES OF INDIVIDUALS AND COMMUNITIES TO EXERCISE CONTROL OVER THE DETERMINANTS OF HEALTH

DETERMINANTS OF HEALTH

INCOME EQUITY/INEQUITY, SOCIAL STATUS, SOCIAL SUPPORT NETWORKS, EDUCATION, EMPLOYMENT AND WORKING CONDITIONS, PHYSICAL ENVIRONMENT, BIOLOGY AND GENETICS, PERSONAL HEALTH PRACTICES AND COPING SKILLS, HEALTHY/UNHEALTHY CHILD DEVELOPMENT, HEALTH SERVICES

MICRO LEVEL OBJECTIVES

Enhanced individual capacities

MESO LEVEL OBJECTIVES

Enhanced community capacities

MACRO LEVEL OBJECTIVES

Supportive institutional and societal environments

OUTCOMES

More equitable access to health care

Increased health promoting activities

Building healthy Public Policy

Reorienting Health services

Enhanced community capacity

Increased community capacity and participation

Developing Personal skills

Strengthening Community action

Creating supportive environments

INSTRUMENTAL OBJECTIVES, PROCESSES AND OUTCOMES

EVALUATION LOOP

INSTRUMENTAL OBJECTIVES, PROCESSES AND OUTCOMES

Individuals, communities and societies achieve/approach their potentials

Reduced health and social costs

Improved Health & Wellbeing

Reduced mortality and morbidity

Risk reduction, awareness, skills, decision making, behaviour

Enhanced organization capacity

Enhanced community capacity and participation

INTER-SECTORAL COLLABORATION

RESEARCH EVALUATION

Knowledge development & dissemination

Health education & communication

Organizational development

Community development

Policy development

Advocacy

Programmes marketing & material, modifies organization structure & climate, coordination of community efforts, increased resources and capacities, laws, regulations and policy statements, public dialogue on decision making, coordination of policies & activities in sectors that affect health.
The type of fat in the diet influences a range of pathophysiological processes involved in CVD, including lipoprotein metabolism, endothelial dysfunction, plaque structure, vascular reactivity, blood pressure, insulin sensitivity and adipose tissue metabolism and topography (Lovegrove 2007). n-3 fatty acids, antioxidant vitamins (especially vitamins E and C), folic acid, and L-arginine appear to have beneficial effects on vascular endothelial function, either by decreasing endothelial activation or by improving endothelium-dependent vasodilation in patients at high risk of cardiovascular disease as well as in healthy subjects (Brown and Hu 2001).

A link has been established between SFA and elevated LDL-cholesterol. There have been major advances in the understanding of how dietary fats influence CVD. The principal lipid abnormality in metabolic syndrome, elevated plasma TAG, is an independent risk factor for CVD. In addition to the direct and potentially adverse effects of TAG-rich lipoproteins on the artery wall, raised plasma TAG also increases the atherogenicity of other lipoproteins, reducing levels of the cardioprotective HDL and increasing the proportion of small, dense LDL, all of which are associated with CVD risk and metabolic syndrome. These lipid abnormalities can be corrected by a dietary-induced reduction of plasma TAG and most notably by long-chain n-3 PUFA (Lovegrove 2007).

Some Dietary Interventions

The INTER 99 randomized controlled trial studied the effect of group based counselling to individual lifestyle counselling on the changes in dietary intake. After one year follow up the intervention group reported an increase in unsaturated/saturated fat ratio. There was an increase in fruit and vegetable consumption. Offering group based counselling apart from individual counselling resulted in significant improvements and maintenance in dietary intake after a five year follow up (Toft et al 2008).

A review of telephone based interventions carried out to improve dietary fat, fruit and vegetable consumption revealed that the intervention was moderately effective. The studies were found to have a positive outcome in terms of increase in fruit/vegetable
intake and improvements in dietary fat intake. The effect was more pronounced among women participants and the individuals at high risk for developing cancer (Vanwormer et al 2006).

A randomized controlled trial was conducted for patients at increased cardiovascular risk using a web based approach. The objective of the trial was to provide nutrition counselling and social support to the 'at risk' group. No favourable effects of web based nutrition counselling were observed in the anthropometry, blood pressure and serum cholesterol. This was attributed to the low uptake of the intervention. The researches suggested improvements in reach and frequency of the site use would improve effectiveness of the intervention (Verheijden et al 2004).

Physical Activity Interventions

The role of physical activity in the primary and secondary prevention of NCDs has been a subject of interest for researchers since the 1950s. There have been numerous long-term prospective follow-up studies (mainly involving men but more recently women also) that have assessed the relative risk of death from any cause and from specific diseases (e.g., cardiovascular disease) associated with physical inactivity. Both men and women who reported increased levels of physical activity and fitness were found to have 20 -30% reductions in relative risk of death (Warburton et al 2006). Regular physical activity is associated with enhanced health and reduced risk of all-cause mortality. Beyond the effects on mortality, physical activity has many health benefits, including reduced risk of CVD, ischemic stroke, non-insulin dependent diabetes, colon cancers, osteoporosis, depression, and fall-related injuries (Warburton et al 2006, Khan et al 2002).

Routine physical activity has been shown to improve body composition (e.g. through reduced abdominal adiposity and improved weight control), improved lipid lipoprotein profile (e.g., through reduced triglyceride levels, increase HDL -C levels and decrease LDL – C and LDL/HDL ratio), improve glucose homeostasis and insulin sensitivity, reduce blood pressure, improve autonomic tone, reduce systemic inflammation, decrease
blood coagulation, improve coronary blood flow, augment cardiac function and enhance endothelial function. Chronic inflammation, as indicated by elevated circulating levels of inflammatory mediators such as C-reactive protein, has been shown to be strongly associated with most of the chronic diseases whose prevention has benefited from exercise (Warburton et al 2006).

Physically inactive middle-aged women (engaging in less than 1 hour of exercise per week) experienced a 52% increase in all-cause mortality, a doubling of cardiovascular related mortality and a 29% increase in cancer-related mortality compared with physically active women. These relative risks are similar to those for hypertension, hypercholesterolemia and obesity, and they approach those associated with moderate cigarette smoking. Moreover, it appears that people who are fit yet have other risk factors for cardiovascular disease (Figure 26) may be at lower risk of premature death than people who are sedentary with no risk factors for cardiovascular disease.

An increase in physical fitness will reduce the risk of premature death, and a decrease in physical fitness will increase the risk. The effect appears to be graded such that even small improvements in physical fitness are associated with a significant reduction in risk. In one study, participants with the highest levels of physical fitness at baseline and who maintained or improved their physical fitness over a prolonged period had the lowest risk of premature death. Modest enhancements in physical fitness in previously sedentary people have been associated with large improvements in health status. In another study, people who went from unfit to fit over a 5-year period had a reduction of 44% in the relative risk of death compared with people who remained unfit (Warburton et al 2006).

**Exercise and body metabolism**

Endurance exercise improves skeletal muscle insulin sensitivity. Notable points in skeletal muscle insulin signal modulation via this type of exercise include increases in GLUT4 protein concentrations and increased activities of both glycogen synthase and hexokinase, the enzyme that phosphorylates glucose.
Some studies have shown that placing sedentary adults on an endurance exercise program improves insulin sensitivity while increasing Intra Myocellular Triglyceride (IMTG) concentrations. The effect of exercise is whole-body mediated, but in these studies, the improved insulin sensitivity in the presence of increased IMTG concentrations is most likely the result of more efficient lipid turnover in that the muscle is becoming more adept at lipid uptake, transport, utilization, and oxidation. Studies have also shown improvements in mitochondrial biogenesis and electron transport chain activity in older persons after 12 wk of endurance training. A similar observation was made in obese, although their IMTG concentrations remained relatively unchanged. Therefore, the capacity for lipid oxidation is increased, yet given the IMTG increase noted in some of these studies, greater FFA delivery and uptake must also be occurring.

The increase in lipid uptake most likely represents an adaptation by the muscle to the increased metabolic demands that arise from strenuous physical exertion. This, coupled
with increased FFA delivery to the exercising muscles, an expected physiological response, would help to explain increased IMTG concentrations. The improvements in insulin sensitivity despite the increase in IMTG are likely related to reductions in deleterious lipid metabolites from a greater lipid flux. Obese subjects were exposed to endurance training, which yielded reductions in both intramyocellular DAG and ceramide content. Reductions in lipid metabolite concentrations may partly explain the improvements in GLUT4 translocation and activities of hexokinase and glycogen synthase. There is also some evidence suggesting that endurance training reduces susceptibility of skeletal muscle to lipid peroxidation. This may lead to further improvements in mitochondrial function. Lastly, the anti-inflammatory effects of exercise are well known, and studies have shown that exercise reduces TNF-α concentrations, which may in part explain the increases in GLUT4 expression (Corcoran et al 2007).

In addition to endurance exercise, resistance training should also be regarded as an essential component in an individual's daily lifestyle. From a physiological point of view, it is well recognized that endurance exercise increases capillary density, improves blood flow to the muscles and skeletal muscle mitochondrial biogenesis, and enhances translational stability of key proteins involved in insulin signal transduction. However, endurance exercise does not substantially affect skeletal muscle hypertrophy and strength compared with resistance training. Because resistance training increases skeletal muscle mass, it can augment whole-body glucose disposal capacity.

Furthermore, studies have shown that even a single resistance exercise training session can improve insulin sensitivity for up to 24 hours after cessation of exercise and that these benefits are possibly attributed in part to reductions in IMTG stores. At first, this may seem contradictory to studies that have shown increases in IMTG from endurance exercise, which imply a discrepancy dependent on exercise type. However, it is important to distinguish between a single training session and multiple training sessions. Many studies examining a single endurance bout have also shown reductions in IMTG concentrations.
It is widely agreed that to really achieve any substantial long-lasting benefit from physical exercise, the activity must be consistently repeated throughout one's life. A single training session of either endurance or resistance exercise will undoubtedly lead to reduced IMTG concentrations, because these lipids have been shown to be a major fuel source in both exercise types, depending on the intensity of the exercise. The enhanced lipid turnover seen with endurance exercise is a consequential adaptation to the metabolic demands of the body. Studies on the metabolic demands of resistance training are few (Corcoran et al 2007).

There is incontrovertible evidence that regular physical activity contributes to the primary and secondary prevention of several chronic diseases and is associated with a reduced risk of premature death. There appears to be a graded linear relation between the volume of physical activity and health status, such that the most physically active people are at the lowest risk. However, the greatest improvements in health status are seen when people who are least fit become physically active (Warburton et al 2006). Thus, it has been suggested that health promotion programs should target people of all ages, since the risk of chronic disease starts in childhood and increases with age.

It has also been suggested that the following three intervention logic frameworks (Figure 27) should be used to increase physical activity using informational, behavioural and social, and environmental and policy approaches.

- Informational approaches to change knowledge and attitudes about the benefits of and opportunities for physical activity within a community;
- Behavioural and social approaches to teach people the behavioural management skills necessary both for successful adoption and maintenance of behaviour change and for creating social environments that facilitate and enhance behavioural change
- Environmental and policy approaches to change the structure of physical and organizational environments to provide safe, attractive, and convenient places for physical activity.
Some physical activity interventions

ProActive trial was an intensive theory based program with an objective to increase physical activity among adults at risk of type 2 diabetes by face to face and telephonic interventions for a year. Short term benefits were reported in terms of cognitive behaviour like enjoying exercise, social interaction etc. (Hardeman et al 2009).

A review to study the effectiveness of interventions (controlled trials) to promote physical activity among children and adolescents revealed some evidence for potentially effective strategies to improve level of physical activity among children and adolescents. Interventions were found to be effective in increasing 2.6 minutes of activity/day resulting in an increase of 283 minutes/week. However, such interventions require very high quality evaluation techniques (Sluijs et al 2007).

Another systematic review studied interventions to promote physical activity in European children that used a multi-component approach through school, family and community settings. The results revealed short term benefits in the form of increased physical activity due to school setting interventions, support from peers and parents and direct environmental changes (Meester et al 2009).

A study was conducted among previously sedentary women who were randomly assigned to one of three groups to promote physical activity. The groups were Choose to Move (a self-help printed booklet), Jumpstart (a motivationally tailored, print based intervention) and Wellness (women's health materials). Face-to-face contact at months 3 and 12 occurred within participants' communities in local libraries. Results suggest that print-based programs for physical activity may be efficacious short-term, but more research is needed to find approaches that are effective long-term. It is possible to deliver print-based programs through existing community infrastructures. However these approaches need further evaluation to examine maintenance effects (Napolitano et al 2006).

A randomized controlled trial was used to assess the impact of a 12-week graduated pedometer-based walking intervention on daily step-counts, self-reported physical activity...
activity and health outcomes in a community setting. Significant increases were found in the intervention group for step-counts, time spent in leisure walking and positive affect. Significant decreases were found in this group for time spent in weekday, weekend and total sitting with no corresponding changes in the control group. No significant changes in any other health outcomes were found in either group. In comparison with the control group at week 12, the intervention group reported a significantly greater number of minutes spent in leisure time, occupational and total walking, and significantly fewer minutes in time spent in weekend and total sitting (Baker et al 2005). European pedometer based “10,000 Steps Ghent” study was a whole community based intervention was effective in increasing step counts by an average of 896 steps/day in a subsample of adults (Cocker et al 2009).

A cross-sectional study was undertaken in urban, age and SES matched physically active and inactive male volunteers. The study suggested that physical activity alone could not maintain BMI and body fat percent, but it can reduce the risk of overweight and high body fat percent in the population (Kesavachandran et al 2009).

A review of computer tailored physical activity behaviour change interventions targeting adults revealed that ten out of the sixteen studies had successful outcomes in terms of increase in physical activity or weight reduction outcomes (Neville et al 2009).

**Lifestyle interventions (Diet and Physical activity)**

Research has shown that lifestyle interventions combining intensive dietary modification and physical activity can reduce chronic diseases. But a range of factors can negatively influence the successful adoption and maintenance of positive lifestyle changes. Many programs demonstrate short term weight loss (3-6 months) but only a few shown weight loss at 12 months or beyond. Sustained weight loss and modifications of risk factors have been achieved by prescribing diet and physical activity under the supervision of dieticians, counsellors, physical trainers, psychologists and behaviourologists (Pettman et al 2008).
Figure 27: Physical activity interventions – How to plan

Interventions

Modifiable Determinants

Information-based Determinants
(Providing information)

Behavioral and Social Determinants
(Behavioral management skills or social support)

Environmental and Policy Determinants
(Facilities and other resources)

Physical Activity Behavior

Intermediate Outcomes

Aerobic Capacity

Other Physiologic Measures
(Aerobic Capacity, Muscular Strength and Endurance, Ligament and Tendon Strength, Flexibility, Heart Rate, Blood Pressure, Capillary Density, Stroke Volume, Cardiac Arrhythmias)

Body Composition
(Increase or Maintain Muscle Mass, Reduce or Maintain Body Fat)

Skill-Based Fitness
(Speed, Agility, Balance, Coordination)

Metabolic Fitness
(Bone Density, Lipid Profiles, Insulin Levels, Coagulation, Immunologic Function)

Mood
(Symptoms of Depression or Anxiety)

Health Outcomes

Measures of Mortality, Morbidity and Quality of Life

Primary and/or Secondary Prevention Established

Ischemic Heart Disease
Hypertension
Type II Diabetes
Colon Cancer
Falls with Fractures
Health-related quality of life

Measures of Mortality, Morbidity and Quality of Life

Primary and/or Secondary Prevention Suggested

Osteoporosis
Depression
Ischemic Stroke
Peripheral Atherosclerotic Disease
Cholelithiasis
Common Colds

Legend

Relationships for Which Evidence May Be Sought

Relationships for Which Evidence Will NOT Be Sought

Khan et al. 2002
Overweight and obese adults with metabolic syndrome were enrolled and randomly allocated to control or intervention group. Both groups received Australian dietary and physical activity guidelines. The intervention group was given a 16 week intervention on behaviour management, dietary habits, exercise sessions and peer group support. The group based lifestyle intervention program achieved improvements in body composition and cardiometabolic and physical fitness. The study also suggested that a continued follow up may be required for long term maintenance in individuals attempting lifestyle behaviour change (Pettman et al 2008).

A worksite intervention to reduce the cardiovascular risk factors in the industrial population employed multiple components of intervention like posters, banners and handouts in the local dialect. These were designed based on the scientific theories of behaviour change and recent literature on interventions. The five year intervention study was well accepted and showed improvement in terms of self reported behaviour change like increase in physical activity, fruit and vegetable consumption, as well as reduction in salt and tobacco consumption. The intervention site also reported a reduced cardiovascular risk in terms of Framingham CVD risk scores as the intervention group displayed lower weight, WC, SBP, DBP, TC and TG levels and increased HDL – C levels. The control group on the other hand displayed a deterioration of metabolic profile and self reported behaviour change (Prabhakaran et al 2009).

ACORN study helped individuals pursue healthy behaviours through a practice based website. In a nonrandomized 9-month intervention, 6 family practices (4 interventions, 2 controls) encouraged adults with unhealthy behaviours to visit the website. Intervention patients reported greater net improvements at 1 month, although the differences approached significance only for physical activity and readiness to change dietary fat intake. Patients expressed satisfaction with the website but wished it provided more detailed information and greater interactivity with clinicians. The results also revealed referring patients to a well designed website that offers access to the world’s best information is an appealing alternative to offering handouts or impromptu advice. Interactive websites can facilitate behaviour change and can interface with electronic health records. Determining whether referral to an informative website improves health outcomes is a methodological challenge, but the larger
question is whether information alone is sufficient to promote behaviour change (Woolf et al 2006).

In another lifestyle intervention a low intensity program was designed to improve fruit and vegetable intake and was integrated with an ongoing physical activity program. The intervention consisted of brief social cognitive theory based messages delivered in the form of newsletters. The intervention was successful in improving fruit and vegetable consumption in adults. Thus, inexpensive techniques like these can be used to improve health behaviours (Doerksen and Estabrooks 2007).

A Model Health Promotion Program

The Singapore Health Promotion Program can be studied as a model program as it covers a wide spectrum of population, including multiple settings like school, workplace, tough antismoking policies since the 1970’s, etc. A comprehensive mix of strategies was adopted, comprising public education, legislation, tobacco taxation and provision of smoking cessation services. The aim was to alert the public of the dangers of smoking, to make smoking expensive, and remove the pressure to smoke. Results revealed that more creative interventions are required to counter the innovative methods used by companies to market their product.

School set up: In order to tackle the trend of increasing obesity levels, the Ministry of Health, introduced the Trim and Fit (T.A.F.) programme for students in 1992 to reduce obesity and improve physical fitness among school children in primary schools up to pre-university level. Major strategies employed in the T.A.F. programme include the control of food and drinks sold in school canteens, exercise programmes for overweight and obese students, parental involvement and support, and collaboration with other related agencies. Over a ten-year period since launch of the programme obesity in school children has reduced from 14% to 10%. A recent initiative, the C.H.E.R.I.S.H. (Championing Efforts Resulting in Improved Health) Award, recognizes schools that have taken a more creative and proactive approach in
nurturing physical and mental health of students and staff to help them adopt healthy lifestyles.

**Workplace set up:** The National Workplace Health Promotion Programme develops a framework to encourage more organizations to conduct and sustain workplace health promotion programmes that achieve desired health outcomes. This framework utilizes health facilitators, who are employees of an organization, as the primary movers within their companies to encourage and facilitate the adoption of healthy lifestyles among the employees. Training courses and education initiatives are conducted to equip facilitators and management staff with knowledge and skills to conduct and promote a healthy lifestyle at their workplaces. Several initiatives have been undertaken to encourage the adoption of workplace health promotion like the H.E.A.L.T.H award, Workplace Health Promotion Grant, etc.

**Community set up:** Every year, a month-long National Healthy Lifestyle Campaign is organized to reinforce the importance of a healthy lifestyle. The Great Singapore Workout (1993), a compilation of easy, low-impact aerobic steps for both young and old, and a new workout, the “Work Fit” (2001) was specially designed mimicking moves of common sports for easy recall. The public education nutrition programme uses many channels to educate the public on healthier food choices, provide a supportive environment for healthier eating, and ensure adequate supply of healthier food. In 1998, Nutrition Labelling was launched to encourage the food industry to display nutritional information panels. The Healthier Choice Symbol on packaged food was devised for easy consumer recognition and restaurants, food courts and hawker centres have been encouraged to offer customers with more vegetables, less fat and oil options through the “ASK for Healthier Food” Programme. The health promotion efforts of the National Healthy Lifestyle Campaign have contributed to the increased level of reported exercise activity in the population and reduction in smoking prevalence (Toh et al 2002).
Pioneering Departmental Health promotion programs

Health promotion in a school set up

The prevalence of overweight and obesity among school children in Urban Vadodara was mapped in 4 schools. The overall prevalence of overweight and obesity was 12.2% among boys and 13.7% among girls. The risk factors contributing to obesogenic environment identified in this group of adolescents were heredity, socio economic status, erratic eating patterns and inadequate physical activity. Two of these schools were enrolled and Nutrition Health Education (NHE) was imparted to the students using oral interactions powerpoint presentations, Brochures, Posters, Skits and Discussions. NHE was imparted on balanced diet, nutrients, good eating practices, physical activity and its importance, obesity its causes, prevention and management. Games like maze, grid, crossword puzzle and jumbled words were used as an evaluatory tool to assess the retention of knowledge.

Educational information imparted by NHE was well received and a modest increase in knowledge was indicated. After intervention, a significant drop in the fat intake in the OW/ OB groups and also in the frequency of sedentary activity performed. However, no appreciable shift was seen in anthropometric parameters. The observations also indicated that the children were receptive to the innovative NHE techniques and the few positive changes observed is helpful and a step towards the prevention of obesity and also that the school setting is an appropriate place to promote physical activity and healthy eating practices.

Health Promotion Program in an industrial set up

Entire population of an industry was enrolled and the prevalence of NCDs was mapped among them. The prevalence of overweight, obesity, diabetes, hypertension and CHD was found to be 33%, 8%, 8%, 6% and 1% respectively. Increased prevalence of NCDs could be attributed to increased prevalence risk factors like excessive energy intake especially in the
form of fats (saturated and trans fat), tobacco chewing, smoking, central obesity, overweight, physical inactivity, dyslipidemia.

A one day program was organized to impart NHE to the employees and their spouses where, lectures were given on Diet in different stages in life, information about the kind of diet consumption in their canteen and dietary modification in obesity, hypertension and diabetes. Open discussions were organized to clarify any doubts. Educational materials were provided to the families in English and the local dialect (Gujarati). The programs elicited a good response and the employees made queries about the lectures were given. People were inclined to switch to a healthy diet and introduce exercise in the routine. Modifications in diet using the low calorie, high fibre recipes were provided on demand (Desai and Mani 2002).