South Asian diets and insulin resistance

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A role of dietary nutrients in relation to insulin resistance has been suggested but conclusive evidence in human beings is lacking. Asian Indians and South Asians are prone to develop insulin resistance and the metabolic syndrome. In the present paper, data pertaining to nutrient intake, insulin resistance and cardiovascular risk factors in Asian Indians and South Asians have been reviewed. In these populations, several dietary imbalances have been reported: low intake of MUFA, n-3 PUFA and fibre, and high intake of fats, saturated fats, carbohydrates and trans-fatty acids (mostly related to the widespread use of Vanaspati, a hydrogenated oil). Some data suggest that these nutrient imbalances are associated with insulin resistance, dyslipidaemia and subclinical inflammation in South Asians. Specifically, in children and young individuals, a high intake of n-6 PUFA is correlated with fasting hyperinsulinaemia, and in adults, high-carbohydrate meal consumption was reported to cause hyperinsulinaemia, postprandial hyperglycaemia and hypertriacylglycerolaemia. Dietary supplementation with n-3 PUFA leads to an improved lipid profile but not insulin sensitivity. Inadequate maternal nutrition in pregnancy, low birth weight and childhood ‘catch-up’ obesity may be important for the development of the metabolic syndrome and diabetes. Even in rural populations, who usually consume traditional frugal diets, there is an increasing prevalence of cardiovascular risk factors and the metabolic syndrome due to changes in diets and lifestyle. Nationwide community intervention programmes aimed at creating awareness about the consequences of unhealthy food choices and replacing them by healthy food choices are urgently needed in urban and rural populations in India, other countries in South Asia and in migrant South Asians.

Insulin resistance: Type 2 diabetes mellitus: Dyslipidaemia: Dietary fats: Dietary carbohydrates: Asian Indians: South Asians

In most individuals who develop T2DM, insulin resistance is generally present for many years before the occurrence of hyperglycaemia.

It is apparent that diet and physical activity significantly influence insulin resistance, the metabolic syndrome, dyslipidaemia and T2DM. The rapidly increasing prevalence of these disorders in Asian Indians has been largely linked to rapid changes in lifestyle and dietary patterns1–3. Unfortunately, the data regarding the relationship of dietary nutrients with insulin resistance are scarce in Asian Indians. Although some studies have reported an influence of dietary nutrients on insulin resistance and cardiovascular risk factors in South Asians and Asian Indians, no review on this subject is available.

The present study aims to review the influence of dietary nutrients on insulin resistance and the metabolic syndrome in Asian Indians and South Asians. The literature search has been done by using the terms ‘insulin resistance, hyperinsulinaemia, the metabolic syndrome, diabetes, dyslipidaemia, and nutrition, diet, fat, carbohydrates, whole grain, fibre, micronutrients, and South Asians and Asian Indians’ in the medical search database PubMed (National Library of Medicine, Bethesda, MD, USA) from 1966 to April 2008. A manual search of the relevant quoted references was also carried out from the retrieved articles. Databases of research institutions in India (Indian Council of Medical Research, New Delhi, and National Institute of Nutrition, Hyderabad) were also searched from respective websites; however, mostly peer-reviewed studies have been referenced. In the present review ‘Asian Indians’ refers to individuals originating from India, and ‘South Asians’ refers to individuals originating from various countries (India, Pakistan, Bangladesh, Nepal and Sri Lanka).

Abbreviations: T2DM, type 2 diabetes mellitus; TFA, trans-fatty acids.
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in the Indian subcontinent. Other terms ‘Indian Asians’, ‘Indo-Asian’ and ‘Indo-Sikhs’ used are reproduced as in the original studies, and broadly signify individuals from India and neighbouring countries.

Dietary fats

Since the 1930s, several studies have implicated high dietary fat intake with the development of obesity and hyperglycaemia8–10. A high dietary intake of fat has been reported in Asian Indians4–6,10. Fat consumption ranged from 13 to 59 g/d in different regions and states in India. Further, individuals in rural areas in India consume lower (17 %) energy intake from dietary fat as compared with urban residents (22 %)11. Yagalla et al.10 have reported a higher average energy intake from total fat (32 %) in migrant Asian Indians in the USA. Further, vegetarian migrant Asian Indians had higher values of BMI than non-vegetarians due to an increased intake of high-fat dairy products. Those migrant Asian Indians who consume high amounts of dietary fats have high levels of serum TAG and low levels of HDL-cholesterol10. It is important to note that migrant Asian Indians have higher BMI and more risk factors for CVD than Asian Indians in India, possibly contributed by changes in lifestyle and consumption of excess energy and fats12. Finally, a high intake of dietary fats significantly correlated (correlation coefficient 0·67) with cardiovascular mortality in different regions of India13. While a wide variety of fat-enriched food items are consumed by South Asians, the fatty acid profile of some commonly cooked dietary items by South Asians is shown in Table 1.

Dietary saturated fatty acids

Various investigators have shown that the intake of SFA was a significant independent predictor of fasting and postprandial insulin concentrations14–16 and that hyperinsulinaemia is decreased by lowering SFA intake14. Thus, the overall intake of dietary SFA is positively related to insulin resistance, and there is evidence that replacing SFA with MUFA or PUFA in dietary fat may prevent metabolic deterioration.

Yagalla et al.10 have reported an average energy intake of 8 % from SFA in migrant Asian Indians in the USA, while the mean SFA intake was 6·5 % in adult city dwellers belonging to the low socio-economic stratum living in India9. Further, we have recently reported a high mean intake of SFA (9·4 % of total energy intake) in urban Asian Indian adolescents and young adults17. In general, there is a rural-to-urban variation in consumption patterns of SFA, with rural Asian Indians having a low, and urban individuals having a high consumption of SFA.

Interestingly, we have shown that intake of SFA is an independent correlate of high C-reactive protein levels, a marker for subclinical inflammation, in urban-based adolescents and young adult Asian Indians18. Based on these observations, to place them in a low-risk category for future CVD (mean C-reactive protein levels <1 mg/l), SFA intake should be decreased to <7 % of energy intake in adolescent Asian Indians. Further, high intakes of SFA and trans-fatty acids (TFA; see below) were reported to be independently associated with increased subclinical atherosclerosis in migrant South Asians living in Canada19.

Table 1. Fatty acid content of cooked food items frequently consumed by South Asians

<table>
<thead>
<tr>
<th>Nutrients‡</th>
<th>Parantha§</th>
<th>Bhatura§</th>
<th>Pulaa§</th>
<th>Pakora‖</th>
<th>Dosai‖</th>
<th>Samosa‡</th>
<th>Fried potato chaat§</th>
<th>Halwa‖</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical serving (g)</td>
<td>80</td>
<td>60</td>
<td>275</td>
<td>100</td>
<td>90</td>
<td>70</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Total fat (g)</td>
<td>12·03</td>
<td>20·32</td>
<td>11·61</td>
<td>17·40</td>
<td>11·36</td>
<td>15·64</td>
<td>15·6</td>
<td>20·73</td>
</tr>
<tr>
<td>SFA (g)</td>
<td>2·01</td>
<td>2·52</td>
<td>2·76</td>
<td>1·65</td>
<td>1·23</td>
<td>2·28</td>
<td>3·75</td>
<td>4·91</td>
</tr>
<tr>
<td>MUFA (g)</td>
<td>2·42</td>
<td>4·58</td>
<td>2·22</td>
<td>4·21</td>
<td>2·77</td>
<td>3·60</td>
<td>3·00</td>
<td>3·90</td>
</tr>
<tr>
<td>PUFA (g)</td>
<td>4·38</td>
<td>11·04</td>
<td>0·76</td>
<td>11·11</td>
<td>7·12</td>
<td>7·12</td>
<td>0·65</td>
<td>0·81</td>
</tr>
<tr>
<td>n-3 PUFA (g)</td>
<td>0·15</td>
<td>12·16</td>
<td>0·06</td>
<td>0·09</td>
<td>0·22</td>
<td>0·09</td>
<td>0·12</td>
<td>0·33</td>
</tr>
<tr>
<td>n-6 PUFA (g)</td>
<td>4·23</td>
<td>10·90</td>
<td>0·70</td>
<td>11·07</td>
<td>6·90</td>
<td>7·03</td>
<td>0·53</td>
<td>0·73</td>
</tr>
<tr>
<td>Trans-fatty acids (g)</td>
<td>2·72</td>
<td>4·45</td>
<td>5·30</td>
<td>0·21</td>
<td>0·14</td>
<td>2·79</td>
<td>7·95</td>
<td>10·6</td>
</tr>
</tbody>
</table>

* Recipes compiled from published literature on Indian foods25,26. Fatty acid contents compiled from published literature on Indian foods27,28. Parantha is an Indian bread prepared on a griddle using fat; bhatura is an Indian bread prepared by deep frying; pulao is a rice preparation; pakora is a sliced/mashed potato cooked with Bengal gram flour batter and deep fried; dosa is a preparation consisting of rice and black gram flour, prepared like a pancake on a griddle; samosa is a snack prepared by stuffing potato in refined wheat flour dough cones and deep fried; fried potato chaat is boiled potatoes shallow fried for crispiness; halwa is a dessert prepared using refined wheat flour (semolina).
† Values for fatty acid content are given for a typical serving.
‡ Prepared in a combination of Vanaspati (hydrogenated fat) as shortening and refined sunflower-seed oil as the frying medium.
§ Prepared in Vanaspati.
‖ Prepared in refined sunflower-seed oil.

Polysaturated fatty acids

Evidence from experimental studies has indicated the beneficial effect of long-chain n-3 PUFA (fish oils) over n-6 PUFA (safflower-seed oil) in preventing insulin resistance, but robust evidence from human studies is lacking. Some human studies indicate a protective effect of fish intake on the development of insulin resistance20–22 but results from dietary intervention studies have not been consistent23. Supplementation with long-chain n-3 PUFA appears to improve insulin sensitivity in subjects with impaired glucose tolerance22,24 and in patients with T2DM22. Even though it has not been well investigated in healthy individuals, long-chain n-3 supplementation clearly lowers levels of serum TAG even when no positive effect on glycaemia and peripheral glucose utilisation were seen25.

More than a decade ago, Sevak et al.26 showed that South Asians consumed significantly lower n-3 PUFA (0·08 v. 0·13 % energy, respectively; P = 0·02), but higher n-6 PUFA (5·4 v. 5·0 % energy, respectively; P = 0·05) than white Caucasians in the UK. Lovegrove et al.27 have also reported similar findings, in addition to a significantly higher...
dietary n-6:n-3 PUFA ratio in diets of Indo-Asian Sikhs in the UK as compared with white Caucasians (11.2 v. 6.7, respectively; P<0.001). Furthermore, these investigators have shown that South Asians had a higher proportion of total fatty acids as n-6 PUFA and a lower proportion of long-chain n-3 PUFA in plasma and cellular membrane phospholipids as compared with white Caucasians.(27) While this could be due to dietary imbalance, low activities of Δ5- and Δ6-desaturases necessary for the formation of long-chain n-3 PUFA and/or the presence of certain dietary factors that interfere with the formation of EPA and DHA could be other explanations.(28) It has been suggested that an imbalance in dietary n-6 and n-3 PUFA may be important for the development of insulin resistance and dyslipidaemia in South Asians(29). We have previously shown a low intake of n-3 PUFA, a low energy intake of n-6 and a low n-6:n-3 PUFA ratio (1.9–2.1, which was much lower than the recommended ratio of 5–10) in Asian Indians belonging to the low socio-economic stratum in North India(30). Furthermore, this underprivileged population had a high prevalence of insulin resistance, dyslipidaemia, hypertension and T2DM(30). In adolescent and young Asian Indians, we have recently shown that the mean percentage of energy intake contributed by PUFA was significantly higher in those with fasting hyperinsulinaemia as compared with those with normal insulin levels (9.2%; P=0.021). However, the percentage energy contribution of n-3 PUFA and n-6 PUFA in the hyperinsulinaemic (0.8 and 4.8%, respectively) and normoinsulinaemic groups (0.7 and 4.2%, respectively) was similar. An important observation was that a higher intake of PUFA was associated with higher fasting serum insulin levels (OR 2.2). Specifically, n-6 PUFA intake was a significant independent predictor of fasting hyperinsulinaemia. In view of these observations in Asian Indian adolescents and young adults, it would be prudent to restrict the intake of n-6 PUFA. Importantly, the role of n-6 PUFA in the pathogenesis of insulin resistance in Indian Asians needs to be investigated further(17).

Interestingly, intervention studies with n-3 PUFA have not yielded encouraging results in South Asians. Lovegrove et al. (27) compared the impact of long-chain n-3 PUFA supplementation in white Caucasians and Indo-Asian Sikhs in the UK. These investigators showed that with supplementation, concentrations of plasma TAG, apo B-48, platelet phospholipids and arachidonic acid decreased, and HDL-cholesterol, platelet phospholipids, EPA and DHA significantly increased in Indo-Asians; however, no effect on insulin sensitivity was seen(27). In another study, Brady et al. (31) reported the effect of fish-oil supplementation against a background of high or moderate intake of n-6 PUFA on fasting and postprandial blood lipids and on insulin resistance in Indian Asians in the UK. They showed that a high dietary intake of n-6 PUFA did not attenuate the beneficial effects of fish-oil supplementation on plasma TAG levels. Further, in line with the study of Lovegrove et al. (27), long-chain n-3 PUFA supplementation, whether given in combination with a background dietary intake of high or moderate n-6 PUFA, had no effect on insulin sensitivity in Indo-Asians. In yet another study by this group, no effect of a high or moderate n-6:n-3 PUFA ratio diet on clinically relevant insulin sensitivity and dyslipidaemia was reported in Indian Asians in the UK. However, there was a trend towards a loss of insulin sensitivity on the high-n-6:n-3 PUFA diet, and lower EPA and DHA levels were observed following the high-n-6:n-3 PUFA diet(32).

Low intakes of n-3 PUFA in Asian Indians could be due to the vegetarian status. Even in those individuals who are non-vegetarians, fish intake is infrequent. Interestingly, those living in the coastal area of India and having high fish intake have a better lipid profile, specifically low serum TAG levels.(33,34) In vegetarian Asian Indians, a higher intake of n-3 PUFA could be achieved by the addition of several low-cost vegetarian dietary items containing n-3 PUFA, for example, green leafy vegetables, rajmah (kidney beans), bajra (Sorghum vulgare) and chana (black gram). These food items are widely available at low cost in India.

**Dietary monounsaturated fatty acids**

A MUFA-enriched diet resulted in significant increases in insulin sensitivity with modest total fat intake in healthy subjects.(35) Further, MUFA-rich diets lowered mean plasma glucose and plasma TAG levels and reduced insulin requirements in patients with T2DM.(36,37). Improvement of lipoprotein and glycaemic profiles with a high-MUFA diet may not be related to changes in insulin sensitivity but could be due to a reduction in the dietary carbohydrate load.(38) The increase in insulin sensitivity induced by MUFA-rich diets may be due to their effect on gastric emptying and increased basal glucose uptake(37). Overall, high-MUFA diets have shown beneficial effects in T2DM, but their influence on insulin resistance, although appearing beneficial, is still inconclusive.

Some 14 years ago, Sevak et al. (20) showed a lower dietary intake of MUFA (% of energy) in South Asians as compared with white Caucasians (11.9 v. 14.7, respectively; P<0.001) in the UK. In a relatively recent study on Indian Asians living in the UK, the mean MUFA intake in subjects with a high-n-6:n-3 PUFA diet was 25 g/d, while in those consuming a moderate-n-6:n-3 PUFA diet, MUFA intake was 43 g/d.(32) Asian Indians belonging to the low socio-economic stratum in India consumed low MUFA (% of energy): males, 4.7%; females, 5.7%.(39) Non-significantly higher MUFA intake (% energy) was seen in hyperinsulinaemic adolescents and young adults in India than those with normal fasting plasma insulin levels (7.4 v. 6.9, respectively; P=0.26)(17). A study of adult urban males from three different states in India (North India, Uttar Pradesh; South India, Goa; West India, Kolkata) showed low MUFA intake(38). In North India (Rajasthan), the daily intake of MUFA (% energy) was higher in illiterate individuals than in educated individuals (15.6 v. 8.6, respectively; P<0.005)(39). In South India, however, the middle income group consumed higher MUFA (11.9 g/d) as compared with the low income group (8.5 g/d)(40). Our data showed a low intake of MUFA (<4% energy) in an urban slum population (19–49 years) in New Delhi (Table 2).

**Trans-fatty acids**

Specifically, dietary TFA intake has been associated with dyslipidaemia and an increased risk of T2DM and CVD.(41,42). Studies in patients with T2DM have showed an elevated postprandial insulin response with a TFA-rich diet as compared with a cis-MUFA-rich diet(43). However, data regarding the
Table 2. Consumption of fats and fatty acids in urban adolescents and adults in India
(Mean values and standard deviations)

<table>
<thead>
<tr>
<th>Age group</th>
<th>Total fat (% energy)</th>
<th>SFA (% energy)</th>
<th>PUFA (% energy)</th>
<th>n-6 PUFA (% energy)</th>
<th>MUFA (% energy)</th>
<th>TFA (% energy)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RDA 15–30%</td>
<td>Male</td>
<td>SD</td>
<td>Female</td>
<td>SD</td>
<td>Male</td>
</tr>
<tr>
<td>Adolescents</td>
<td>13–18 years</td>
<td>32.4</td>
<td>7</td>
<td>35.6</td>
<td>6.3</td>
<td>116</td>
</tr>
<tr>
<td>(n 797)*†‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adults</td>
<td>19–49 years</td>
<td>30.8</td>
<td>8.4</td>
<td>34.1</td>
<td>6.7</td>
<td>89</td>
</tr>
<tr>
<td>(n 326)*†‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>50 years</td>
<td>31.2</td>
<td>6.8</td>
<td>33.5</td>
<td>6.2</td>
<td>92</td>
</tr>
<tr>
<td>(n 124)*†§</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>18–69 years</td>
<td>24.7</td>
<td>12</td>
<td>28.7</td>
<td>10.5</td>
<td>166</td>
</tr>
<tr>
<td>(n 227)*‡</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
| RDA, RDA for Asian Indians(89); TFA, trans-fatty acids.
* Representative sample from the general population in New Delhi, India (unpublished results).
† Design: cross-sectional stratified cluster sampling according to WHO methodology. Dietary intake methodology: 24 h dietary recall and food-frequency and amount questionnaire.
‡ Funded by the Department of Science and Technology, Ministry of Science and Technology, Government of India.
§ Indo-US study funded by the US Congress and carried out in collaboration with Texas A&M University, USA (R. Misra and A. Misra, unpublished results).
|| Individuals belonging to the low socio-economic stratum living in urban slums, New Delhi(9).
| Range. |
Asian Indians and reported that increased carbohydrate intake (above the threshold of total carbohydrate intake of 282 g/d) in them resulted in high serum TAG, particularly in insulin-resistant subjects. Even in Asian Indians belonging to the low socio-economic stratum living in urban slums, a higher percentage of energy from carbohydrate intake was positively correlated with serum TAG levels(9).

The consumption of large carbohydrate meals is very common in Asian Indians, especially at dinner time. This permits hyperinsulinaemia to occur, and also causes postprandial hyperglycaemia and hypertriacylglycerolaemia. Therefore, a rational strategy would be to distribute carbohydrate evenly through three to five meals per d, especially in patients with diabetes, so as to avoid high carbohydrate loading.

Dietary fibre

Evidence from epidemiological studies supports the beneficial effects of high intakes of fruits and vegetables, with possible reductions of over 80% in CHD, 70% in stroke and 90% in T2DM by following Mediterranean diets, which are low in energy and high in fibre(54). Higher intakes of fruit and vegetables have been shown to lower the risk of the metabolic syndrome(55).

Few studies in India have reported data on fibre intake; however, methodologies of analysis of fibre intake have differed. Poor intake of fruit and vegetables resulting in low fibre intake was reported in less educated urban Asian Indians in North India(39), in subjects residing in West Bengal, India (reported as crude fibre intake; 5.7 g/d)(56) and in an urban slum population in North India (reported as crude fibre intake; males, 8.5 g/d; females, 4.1 g/d)(9). In a recent study on adolescents and young adults from North India, we showed crude dietary fibre consumption to be 8.6 g daily(17).

Fibre intake may also depend on socio-economic stratum and ability to buy relatively expensive fruits and vegetables, being higher in the middle income group (8.6 g/d) than the low income group (4.7 g/d)(40). Many studies in rural populations in India(57), including a subset of rural pregnant women(58), have shown a poor intake of fruits and vegetables. Even in migrant Asian Indians or South Asians, fibre intake has been reported to be low, particularly in vegetarians; however, it varied by region of origin in India and dietary profile of the migrants from India and other South Asian countries(59). Intervention with high-fibre diets in Asian Indians has been poorly investigated. Mean blood glucose values in North Indian subjects residing in the UK after a high-fibre (32 g/d) mixed meal was lower than after the standard glucose load. According to the authors, these findings suggest the potential of high-fibre constituents of a typical North Indian diet in improving glucose tolerance(60). Clearly, more data are needed from South Asians in this area of nutrition.

Micronutrients and trace elements

Trace elements (micronutrients) such as Mg have been postulated to play a role in glucose homeostasis and insulin action(61,62). Some investigators have shown that dietary fibre and Mg explain most of the beneficial effect of whole grains on insulin sensitivity(63–65). In this context, it is important to note that both Mg and Ca intakes were significantly lower for the Asian Indian vegetarians than for the white Caucasian vegetarians(66). Vitamin D and Ca intakes were less than two-thirds of the recommended intake in Gujarati Asian Indian migrants in the USA, and BMI was negatively correlated with Ca intake(67). Nearly 95% of ethnic South Asians in the UK (predominantly British Bangladeshis), whose random blood glucose level indicated ‘high risk’ of diabetes, had vitamin D deficiency(68). The implications of trace element deficiency on the metabolic syndrome and CVD are not clear in South Asians and long-term studies are required to address this issue.

Effect of nutrition during the perinatal period and early childhood on insulin resistance and the metabolic syndrome

Asian Indian mothers residing in rural areas in India consume lower energy (approximately 7.53 v. 10.04 MJ, respectively) and protein (45 v. 90 g/d, respectively) but higher percentage energy from carbohydrates (72 v. 50%, respectively) as compared with British mothers(69). Such inadequate maternal dietary intake during intra-uterine fetal development leads to fetal undernutrition and smaller neonatal size. Further, some evidence exists that there is an increased susceptibility of low-birth weight babies to insulin resistance, hypertension and CVD in adult life. Some scientists believe that widespread maternal undernutrition has contributed to the diabetes epidemic in Asian countries, supported by the fact that more than half of low-birth-weight babies are South-east Asians(70). This line of thought appears to be an oversimplified explanation to diseases, which have complex causation. At the same time, it is interesting to note that better nourished and heavier urban Asian Indian babies (mean weight 2.9 kg) have an almost five times higher susceptibility to T2DM as compared with rural babies (mean weight, 2.6 kg)(71), implicating overriding roles of dietary excess and lifestyle factors. Indeed, the evidence now suggests that those who have a low birth weight have an increased tendency to develop hyperglycaemia and the metabolic syndrome only when fed excess energy and those who have increased velocity of weight gain and adiposity in childhood(72,73).

The focus is gradually shifting to micronutrient deficiencies in maternal diets and their relationship to insulin resistance in children and adults. It is important to note that the intake of most vitamins and minerals, particularly vitamin D, total folate, vitamin B₆, and Mg, were lower in pregnant Asian Indian women in the UK as compared with the intakes of most nutrients consumed by pregnant European women(74). Interestingly, in a recent study in women residing in West India, high maternal erythrocyte folate concentrations predicted greater adiposity and higher insulin resistance, and low vitamin B₁₂ levels predicted higher insulin resistance in offspring(75). Maternal nutrition during pregnancy, both macronutrients as well as micronutrients, in rural Indians has been reported to affect fetal growth, body composition and disease risk in adulthood(69).

Overall, both maternal undernutrition and excess nutrition in children appear to be important in the development of adiposity, insulin resistance and related diseases in childhood and adults; however, it would be premature to implicate any specific micronutrient. The preventive implications on
components of the metabolic syndrome would involve better fetal growth through improved maternal nutrition and reducing over-nutrition in the early years of life.

**Dietary patterns and socio-economic status in Asian Indians and the insulin resistance syndrome**

In South India, individuals belonging to the middle income group (most having a sedentary job profile, average monthly income 8075 rupees (approximately US$ 200)) consumed significantly higher amounts of energy (7855 v. 6577 KJ; 1877 v. 1572 kcal), total fat (60.7 v. 32.3 g/d), SFA (15.9 v. 11.3 g/d) and sugar (73.1 v. 43.8 g/d) as compared with the low income group (most in labour-intensive jobs, average monthly income 1400 rupees (approximately US$ 35))\(^{(40)}\). Further, age-standardised prevalence rates of obesity, impaired glucose tolerance, T2DM, hypertension, dyslipidaemia, the metabolic syndrome and CVD were significantly higher in the middle income group as compared with the low income group\(^{(40)}\). Along with imbalanced dietary intake, including low intakes of MUFA, n-3 PUFA and fibre and a high intake of SFA, the urban slum dwellers (low socio-economic stratum, 90 % having monthly income < US$ 70 monthly)\(^{(9,70)}\) in North India showed a high prevalence of abdominal obesity, hypercholesterolaemia, hypertriglyceridaemia and T2DM and low levels of HDL-cholesterol\(^{(9)}\). Interestingly, one study has shown a higher intake of total energy and SFA in rural v. urban subjects; however, the prevalence of CVD and hypercholesterolaemia was higher in urban subjects and hypertriglyceridaemia and low levels of HDL was seen more in rural subjects\(^{(77)}\). Recent data show that the prevalence of dyslipidaemia, obesity and the metabolic syndrome in rural areas of India is increasing. A study of the rural population of Andhra Pradesh (Central India) showed that 18.4 % of men and 26.3 % of women were overweight, and 26.9 % of men and 18.4 % of women had the metabolic syndrome\(^{(78)}\).

**Regional and geographical dietary patterns and habits and insulin resistance**

Regional differences in dietary patterns and food habits may have some influence on the occurrence of obesity and hyperglycaemia in Asian Indians. Data from a study carried out nearly 35 years ago show differences in North and South Indian diets\(^{(79)}\). While South Indians predominantly eat a diet consisting of boiled rice and sambar (thin lentil soup), use coconut oil in cooking and consume a small quantity of milk, North Indians eat a wheat-based diet, consisting of wheat chapattis (Indian bread), vegetables cooked with ghee (clarified butter), milk and yoghurt. These authors also showed that North Indians consumed 2700 calories and 150 g fat per d, and South Indians consumed 2400 calories and 12 g fats per d, including mainly seed oils. The higher carbohydrate intake in South Indians v. North Indians was probably due to rice-based diets in the former. In this study, a higher prevalence of T2DM (8.8 %) was shown in the South Indians v. the North Indians (2.7 %)\(^{(79)}\).

It is important to note that currently, while most traditional South and North Indian families continue to consume diets as above, many young individuals in many parts of India consume a wide variety of diets; North and South Indian, and ‘Westernised’ diets and snacks, in line with rapid nutrition transition and the increasing presence of aggressive marketing by transnational food companies.

**Community lifestyle intervention studies in Asian Indians**

Given imbalanced nutrition and the increase in obesity, the metabolic syndrome and T2DM in Asian Indians, community-based non-pharmacological intervention through the promotion of healthy food choices and increase in physical activity are needed. A relative risk reduction of 28.5 % (P = 0.018) in cumulative incidences of diabetes through lifestyle and dietary changes, higher than that achieved by metformin alone (26.4 %, P = 0.029), has been shown in South Indian patients\(^{(80)}\). Improved dietary patterns of individuals with hyperglycaemia, a decrease in obesity, and a reduction of fasting blood glucose levels in adults and adolescents (aged 10–17 years) with pre-diabetes and adults with T2DM by 11, 17 and 25 %, respectively, were seen in a South Indian population with dietary interventions. The dietary modifications included: increase in fibre and protein intake from local low-cost resources such as the nutritionally rich drumstick leaves, millets, legumes/lentils and whole grains; avoidance of sweetened drinks; substitution of polished white rice with millets, sprouted legumes and vegetables; reduction of fat content and portion control\(^{(81)}\). Asian Indian migrants in New Zealand also showed a decrease in obesity, blood pressure, and beneficial effects on dyslipidaemia but no effect on insulin sensitivity with dietary interventions, which included encouraged use of rapeseed oil, fat removal from meat and increase in fish consumption\(^{(82)}\).

We have initiated programmes aimed at awareness and prevention of childhood obesity, namely ‘CHETNA’ (Hindi for ‘The Awareness’) (Children Health Education Through Nutrition and Health Awareness programme) and ‘MARG’ (Hindi for ‘The Path’) (Medical Education for Children/Adolescents for Realistic Prevention of Obesity and Diabetes and for Healthy Ageing)\(^{(83,84)}\). Dietary modifications advised include: reduction in fried snacks, commercial/fast foods containing TFA, polished rice, refined carbohydrate flour (by using whole grains and mixing protein and fibre-based flour such as Bengal/black gram flour and bajra (Surghum vulgare)); promotion of fruit and vegetable intake; replacing aerated/sweetened fruit drinks with healthy alternatives such as lemonade and skimmed buttermilk. These dietary changes are emphasised through lectures and printed leaflets, and through involvement of students in debates, skits and cookery contests. This large-scale programme for the first time in South Asia aims to cover 500 000 children in about fifteen cities of North India.

**Conclusions**

Dietary factors are likely to have greater and often overriding influence in the generation of insulin resistance, the metabolic syndrome and T2DM in Asian Indians and South Asians than genetic factors. Several studies in Asian Indians have established the link between dietary nutrients and insulin resistance. Higher intakes of carbohydrate, SFA, TFA and n-6 PUFA, and lower intakes of n-3 PUFA and fibre, and a higher n-3 : n-6 PUFA ratio have been reported in South Asians, as compared with other populations. Intervention studies with n-3 PUFA...
increased the EPA and DHA content of membrane phospholipids, improved lipid profile but did not show a beneficial effect on insulin resistance. Further, high dietary n-6 PUFA and SFA are significant independent predictors of fasting hyperinsulinaemia and high levels of C-reactive protein, respectively, in adolescent Asian Indians. Asian Indians consume large carbohydrate meals, which may lead to high concentrations of plasma TAG, decreased levels of HDL-cholesterol and also cause postprandial hyperinsulinaemia. Maternal and fetal undernutrition and excess adiposity in early childhood increase the risk of hyperglycaemia and insulin resistance later in life.

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