

## Thematic Article

# Nutrition and diabetes in the Asia–Pacific region with reference to cardiovascular disease

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In many parts of the Asia–Pacific region, diabetes prevalence is increasing and seems destined to become a major risk factor for cardiovascular disease. The phenomenon seems predicated on insulin resistance (IR), partly attributable to an early impact of abdominal (visceral) adiposity than in Caucasian populations. Food intake along with physical activity and emotional stress are all determinants of glycaemic status. The glycaemic index (GI) of foods indicates that a number of food factors other than glucose content are important for good glycaemic response to foods and meals. These include (i) low GI foods could also be ones low in fat, (ii) foods that have the lowest GI which include lentils, pasta, noodles, multigrain breads and some fruits (e.g. grapefruit, plums) and (iii) fruits are to be preferred to their juices. The nutritional management of diabetes is best served by counselling changes in a sociocultural context and step-wise fashion by negotiation rather than prescription. It needs to be accompanied by advice to engage in regular physical activity, both aerobic and strength training. The same concept applies to the prevention of abdominal adiposity and diabetes mellitus type II in the Asia–Pacific region, but with particular reference to protective regional food.

**Key words:** abdominal adiposity, Asia–Pacific, cardiovascular disease, diabetes, insulin resistance.

## Introduction

The nutritional prevention of most diabetes may start as early as fetal life with maternal nutrition. Reduced food variety, excessive refining of food and saturated fat, with sedentary lifestyle unmasks predisposition to non-insulin dependent diabetes (diabetes mellitus type II) and increase the likelihood of complications. Food pattern is important, with preference for small, more frequent meals rather than large infrequent meals being an advantage. For short-term (acute meal response) and longer term glycaemic control, as well as an aid to satiety, low-fat low glycaemic index (GI) foods are encouraged. Some sucrose and alcohol are compatible with good glycaemic control. Nutritional behaviours are more likely to change if the sociocultural context is respected and negotiation rather than prescription is used. Regular physical activities, like walking and strength training, are crucial in prevention and management. Informed self-monitoring reinforces adherence.

## The increasing prevalence of diabetes

Most diabetes is diagnosed as diabetes mellitus type II or that associated with insulin resistance (IR) and non-insulin-dependence, although insulin may be required in the later stages of the disease. Ahead of frank diabetes, diagnosed by WHO criteria (changed in recent times),<sup>1–3</sup> there is usually as much again or more impaired glucose tolerance (IGT) or impaired fasting glycaemia (IFG). The latter is an increasingly used screening test for ‘dysglycaemia’ (either IFG or diabetes mellitus type II).

Both IFG and diabetes mellitus type II were uncommon among the peoples of Asia and the Pacific until recent times. While diabetes prevalence doubled in many Caucasian populations during the 1980s, its prevalence in Pacific Islanders, for example in Nauruans, South Asians and Mauritians (of

various ethnicity – African, South Asian, Chinese and Caucasian) increased more dramatically (Tables 1a, 1b).<sup>4,5–7,8–11</sup> By the 1990s, some south-east Asian (Malay and Chinese) populations were reaching diabetes prevalences of around 10%.<sup>11,12</sup> A similar phenomenon was happening among Chinese and South Asians in Australia.<sup>13</sup> Hakka Chinese in Meixian and in China at the end of the 1980s had virtually no diabetes, but their migrant relatives in Mauritius had a prevalence of 11%.<sup>9,10,14–16</sup>

The prevalence of diabetes can reach upwards to 40% (as in Polynesia) and it is uncertain how high it may go. The potential community health impact, through diabetes complications, notably cardiovascular disease (CVD), nephropathy, neuropathy and blindness of this rising prevalence of diabetes is enormous and may cripple health budgets.

The Swedish diabetes register has provided some evidence that food processing may be important in the development of insulin-dependent or type I diabetes.<sup>17</sup> More specifically, an energy dense (especially high fat, high alcohol, low dietary fibre) diet predisposes towards obesity and therefore to type II diabetes.<sup>18,19</sup> More work is required to understand the complex relationships between urbanisation, obesity and type II diabetes, but factors that predispose towards less lean mass and more bodyfat, especially more visceral fat appear critical.<sup>20,21</sup> These understandings increasingly provide a basis for nutritional intervention.

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Accepted 15 January 2001

Swedish studies have also looked at the role of exercise in the prevention and management of diabetes mellitus type II, suggesting a direct link.<sup>22,23</sup>

In planning a nutritional approach to diabetes management an overall philosophy is needed (Table 1, Fig. 1).

**Risk factors for dysglycaemia and insulin resistance in the Asia-Pacific region**

It has become increasingly clear that at an early stage in the increment of visceral (abdominal) fatness among some Asian peoples (Singapore, Vietnam), the risk of dysglycaemia is greater than for contemporary Caucasian populations.<sup>24,25</sup> This may not have always been the case, as it may be attributable to the lifelong effects of maternal nutrition deprivation and consequential low birthweight (LBW) and associated

developmental abnormalities, as the child is born into a nutritional environment with a biological expectation that it will be deprived, yet later food becomes abundant.<sup>26-30</sup> This has happened throughout the 20th century, as a consequence of war, but the risk of visceral obesity may have been minimised when populations were relatively more active. Children who have had LBW may encounter, even in adolescence, an abundant food supply accompanied by sedentariness. As an example, short stature among Chinese Australians predisposes to abdominal obesity and in turn to diabetes.<sup>31</sup>

Even so, food patterns still matter. Food variety in Chinese can protect against abdominal fatness.<sup>32</sup> For reasons inter alia, reduced exposure to low-energy density foods (energy density is the energy value of a food per unit mass of

**Table 1a.** Prevalence of diabetes mellitus in the South-East Asian Peninsula<sup>11</sup>

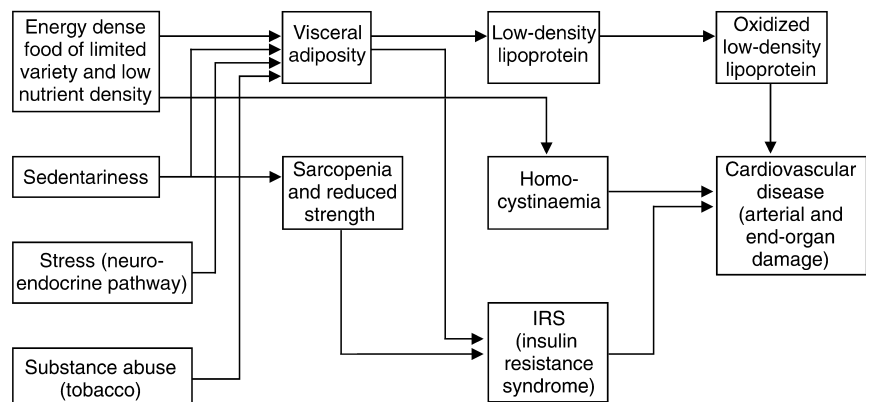
Country	Prevalence (%)	Year	Comment
Vietnam	1.4*†	1990	Hanoi
	2.5†	1992	Ho Chi Minh City (urban)
Indonesia	5.7	1992	Jakarta (urban)
Malaysia	> 8.0*	1997	National survey
Singapore	8.1*	1992	Ethnic Chinese; higher in Indians and Malays
Thailand	11.9	1995	North-East Thailand (rural); age 30-74 years

\*Age-adjusted figure; † Screening test used.

**Table 1b.** Prevalence of diabetes mellitus among Chinese populations in East Asia, since 1990<sup>11</sup>

Country	Prevalence (%)	Year	Comment
China	1.6	1993	30-64
China*	2.5	1994	25-64
Zhejiang, China	3.2	1993	30-64
Beijing, China	3.63	1992	30-64
Hong Kong	7.7	1990	Age-standardised
Hong Kong	8.9	1995	Age-standardised
Taiwan	9.0	1994	> 40
Taiwan	11.0	1995	Age-standardised
Singapore	8.1	1992	Age-standardised

\*19 provinces; n = 213, 515.



**Figure 1.** The role of visceral adiposity, sarcopenia and the insulin resistance syndrome (IRS) in cardiovascular disease.

that food), to the intake of phytochemicals (in particular phytoestrogens), with effects on fat distribution or other factors, are yet to be explored. There is a tendency for cigarette smoking to reduce the interest in food variety and also to predispose to abdominal obesity, although this latter phenomenon possibly relates also to hormonal properties of factors in cigarette smoke.

### Dysglycaemia, insulin resistance and risk for cardiovascular disease

The extent to which abnormalities of glucose metabolism (reflected in hyperglycaemia) and IR (reflected in hyperinsulinaemia) contribute independently and directly to CVD has been a matter of debate and depends very much on the coexistence of other risk factors.

It must first be understood that the site of adverse action of these factors may be either at the arterial wall or at the end-organ (heart, brain, peripheral nerves, kidney, gut, limbs, genital organs, eyes). This is because not only does arterial damage reduce the capacity for blood flow, but also the use of glucose as a fuel is critical for anaerobic metabolism in the face of ischaemia with reduced oxygen supply.<sup>33,34</sup> Epidemiological studies pointed to the role of hyperinsulinaemia in coronary heart disease (CHD) events.<sup>35</sup> Arterial wall studies emphasise the importance of both blood glucose and insulin independent of lipids, blood pressure and cigarettes smoking in macrovascular disease.<sup>36,37</sup> However, the permissive role of hyper-low-density lipoprotein-aemia (hyper-LDL-aemia), the principle basis of hypercholesterolaemia, is generally acknowledged.

Insulin resistance does also contribute to dyslipidaemia, with hypertriglyceridaemia and low high-density lipoprotein (HDL) cholesterol concentrations, both of which increase atherogenicity and probably hypertension and hyperuricaemia. This is the so-called insulin resistance syndrome (IRS), usually seen in the presence of visceral adiposity. It is plausible that visceral adiposity accounts for much of the IRS (Fig. 1).<sup>38</sup>

### Food patterns

In healthy individuals, there is a relatively greater resistance to insulin in the latter part of the day compared with the earlier part.<sup>39</sup> However, this may not apply once diabetes mellitus type II is established.<sup>40</sup>

Nevertheless, at any time of the day, once insulin response or action is in any way compromised, the glycaemic response of increasing food portion size needs to be considered. Thus,

spreading food out across the day remains an important strategy in the nutrition management of type II diabetes.

Jenkins has shown that small, frequent, non-fatty meals also decrease overall cardiovascular risk factor profiles.<sup>41</sup> From a practical point of view, this often means substituting snacks like sweet and even savoury biscuits (which are often high in fat) with fruit.

There is increasing evidence that a variety of foods may be associated with reduced overall glycaemic response and status (Table 2).<sup>42–44</sup> These findings about food variety may depend on a number of food properties which includes both nutrient and non-nutrient (phytochemicals). Phytochemicals are increasingly of interest as cardioprotective agents.<sup>45</sup>

Individual foods, like fish, have also been found protective against arterial wall damage.<sup>46,47</sup>

### Diabetes mellitus type II versus diabetes mellitus type I

The difference between type I and type II diabetes in relation to food pattern is that where pharmacological agents are not used, the glycaemic response is principally determined (for a given level of physical activity and state of psychosocial stress)<sup>48</sup> by the food intake and pattern. A recent study has shown that cereal fibre has a particularly strong inverse relationship to type II.<sup>49</sup>

Where insulin is used, especially by a 'Basal-Bolus' regimen, some flexibility in food intake is possible. If fixed insulin regimens, for example single or twice daily with medium and short acting components, meal and snacking times need to relate to the insulin dose.

### Food choices

The major development of relevance to food choice in diabetes relates to the fact that a given amount of carbohydrate may be followed by a very different glycaemic response depending on the phytochemical and other chemical modifying factors in the food or meal.<sup>50–52</sup>

### Glycaemic index

As a consequence of the glycaemic response differentials between foods, the GI has been developed. This relates the area under the blood glucose response curve from a food with equivalent carbohydrate, to that with a reference food, such as glucose or white bread. In general, a simple sugar which contains both glucose and fructose (e.g., sucrose) or which contains glucose and galactose (e.g., lactose) will have a lower glycaemic response than one that has only glucose.<sup>53</sup> Foods that are more viscous or where particle size is greater

**Table 2.** Rationale for nutrition in diabetes care

- Preventing the development of diabetes in susceptible individuals or for secondary reasons, such as those with pancreatic disease or using glucocorticosteroids;<sup>22</sup>
- To reduce the damaging effects of elevated blood glucose on tissues such as the eye, kidneys, nervous system and arteries;<sup>75</sup>
- To keep the blood fats (cholesterol, triglycerides, high-density lipoprotein cholesterol) as normal as possible;<sup>76,77</sup>
- To reduce damaging effects on tissues by any other mechanism, such as oxidation;<sup>68,78</sup>
- To improve the action of available insulin by:
  - minimising abdominal fatness
  - improving the action of the insulin receptor in cell membranes<sup>79</sup>
  - improving the action of insulin in the cell
  - reducing the amount of circulating free fatty acids (FFA)
  - increasing the utilisation of FFA in ways that do not interfere with glucose metabolism<sup>23,80,81</sup>

(e.g., multigrain bread) will have lower glycaemic indices. Legumes as a food category have the lowest GI (Table 3).<sup>54–57</sup>

Fatty foods tend to delay gastric emptying and are generally excluded from food plans for diabetics as they are not nutrient dense (nutritious) and they contribute to the development of long-term macrovascular complications. Another problem about a high-fat intake is that it predisposes to IR, unless it is rich in omega-3 fatty acids.<sup>58,59</sup> Monounsaturated fats also have a place.<sup>60</sup>

The total glycaemic load of foods as meals and snacks, along with cereal fibre intake and magnesium status (Mg comes especially from chlorophyll-containing plant food or greens and seeds), predicts the development of diabetes over the long-term.<sup>61</sup> Other studies indicate that n-3 fatty acids rich foods (fish) may protect against the development of IR and diabetes, possibly because of the importance of these fatty acids in insulin receptor function.<sup>62,63</sup>

### Alcohol

Alcohol can reduce blood glucose because it reduces gluconeogenesis. This effect can be reduced if it is taken with food. If the particular alcoholic drink contains carbohydrate, the patient can evaluate this with blood glucose monitoring. While there is no specific reason to exclude people with diabetes from drinking alcohol, consideration should be given to the likelihood of further pancreatic beta cell damage or cirrhosis (which itself impairs glucose tolerance) and the more general sociomedical concerns with alcohol.

Recent data indicate that there may be an optimal intake of alcohol in relation to diabetes risk.<sup>64</sup>

### Changing nutritional behaviours

Initially, it is important to elicit and record what a patient eats, then negotiate change and review what is achieved with further counselling. The process of recording food intake by way of a diary can be part of the process of behavioural change.<sup>65</sup>

Working with a person's food culture, family and work place needs is essential for adherence to agreed change.

**Table 3.** Dietary food variety list

Plant-derived food
1. Cereals – wheat based, rye based, oats, rice, barley
2. Vegetables – green leafy, flower type (broccoli, cauliflower), root, runner-yellow (pumpkin, squash), tomatoes
3. Fruits – citrus, stone, bananas, tropical, berries
4. Legumes
5. Nuts – almonds, peanuts, cashew
6. Infusions, beverages – tea, coffee
Fungi and yeasts
1. Mushrooms
2. Yeast – brewers, bakers
Animal derived
1. Finned fish, fresh or tinned
2. Crustaceans
3. Shellfish
4. Ruminant animals – sheep, cattle, deer
5. Monogastric animals – pig
6. Avian – poultry, pigeon
Confectionery – sugar based, chocolate
Alcoholic beverages

**Table 4.** The glycaemic index of some foods

Food	Glycaemic index
Breakfast cereals	
Kellogg's All-Bran	30
Kellogg's Corn Flakes	77
Porridge	42
Grains/pastas	
Rice	
Calrose	83
Basmati	58
Brown	76
Long-grain (white)	56
Glutinous	98
Jasmine	109
Noodles	
Instant	47
Mung bean	39
Rice (fresh)	40
Rice (dried)	61
Vermicelli	58
Pasta	
Egg fettuccine	32
Spaghetti	41
Bread	
Bagel	72
Mixed grain bread	45
Wholemeal bread	77
Crackers	
Ryvita	69
Jazz	55
Biscuits	
Arrowroot	69
Oatmeal	55
Vegetables	
Carrots	49
Sweet potato (Pacific Island)	44
Sweet potato (other)	54
Yam	51
Taro	58
Swede	72
Potato baked	85
French fries	75
Legumes	
Lentils	29
Soya beans	18
Broad beans	79
Chick peas (boiled)	33
Fruit	
Cherries	22
Plum	24
Grapefruit	25
Apricot	31
Apple	36
Apple juice	40
Grapes	43
Oranges	44
Orange juice	46
Kiwifruit	52
Banana	53
Mango	55
Chico	57
Pawpaw	58

**Table 4.** Continued

Food	Glycaemic index
Rockmelon	65
Pineapple	66
Watermelon	72
Dates (dried)	103
Dairy foods	
Milk	
Whole	27
Skim	32
Yoghurt	
Low-fat fruit	33
Indian foods	
Varagu	68
Varagu and greengram dal	78
Varagu and whole greengram	57
Bajra	55
Jowar	77
Ragi	104
Indian snack foods	
Bengal gram cheela	42
Green gram cheela	36
Fermented Bengal gram cheela	45
Fermented green gram cheela	38

The above data were taken from references 82–85.

'Diets' should not be 'prescribed'. Indeed, there is evidence that slavish compliance may actually be associated with increased morbidity and mortality.<sup>36,66,67</sup>

Small changes in food habits in a sociocultural context and aligned with other behaviours conducive to physical fitness and mental health, can lead to substantial gains in metabolic fitness.<sup>68</sup>

### **Gastroparesis**

Where autonomic neuropathy supervenes as a diabetic complication, gastroparesis can present considerable difficulty for nutritional management because of the unpredictability of gastric emptying, with hyper- and hypoglycaemia. Sometimes prokinetic agents like cisapride (now restricted in use because of side-effects) can be helpful.

### **Maintaining outcomes**

The most valuable aid to long-term success in nutritional management is the informed independence of the patient. This can be aided by interest in prevention from first degree relatives (who are themselves at risk of developing diabetes mellitus type II). Behaviours that seek and help achieve a preferred body composition need to be encouraged as this will also aid euglycaemia and low macrovascular risk factor profiles.

Blood glucose monitoring which accounts for short- and long-term effects of dietary change is important – the benefits of a higher unrefined carbohydrate (or high monounsaturated fat, like olive oil) eating pattern may unfold over a couple of weeks for glycaemic status and months for body

composition.<sup>60,69</sup> Therefore, checking blood glucose after meal tells only part of the potential benefit of nutritional change – fasting blood glucose falls more slowly and decreased body fatness even more slowly.

Where the patient and family take more day-to-day control, the doctor and dietitian can take more strategic and surveillance roles.

### **Reducing the future burden of insulin resistance syndrome-related disease in the Asia-Pacific region**

The potential for reduction in prevalence of IRS-related disease in the Asia-Pacific region has undoubtedly been underestimated for several reasons:<sup>70</sup>

1. The staple food, principally rice, but sometimes wheat (as in the north of China) or cassava (as in parts of Indonesia) used has not heretofore been associated with other than sporadic or low prevalence of type II diabetes. As it plays a role in dysglycaemia (inappropriately high insulin response for a given blood glucose concentration) observed with lifestyle changes, energy sources could be diversified and include a range of lower glycaemic foods, snacks and meals.
2. Ageing populations are more prone to IRS, not only because of increased visceral adiposity, but also because of sarcopenia (loss of muscle mass) and reduced muscle strength, both of which are now known to affect glucose handling. If physical fitness (aerobic and strength training) are encouraged as the demography of the Asia-Pacific region changes to an older population, traditional diets and unfavourable introduced food choices may be better tolerated.<sup>71</sup>
3. Changing quality of fat intake as well as increased amounts can affect both insulin action at its receptor and insulin secretion by the pancreatic beta cell, respectively.<sup>70,72</sup> Fat intake also slows gastric emptying and lowers GI, although the effects of this beyond the eating episode in question may not be as favourable.<sup>73</sup> These dietary changes are low on the agenda of diabetes prevention and management in the Asia-Pacific region at the present time.
4. The loss of foods potentially protective against IRS for example, legumes of which soy is an important regional example, green leafy vegetables as a source of magnesium and fish as a source of n-3 fatty acids has not received much attention. This is also evident in protection against the complications of diabetes, for example, through plant foods of sufficient amount and variety to provide a range of antioxidant phytochemicals – carotenoids and polyphenolics, along with tea (oo-long in China or green in Japan).
5. The importance of maternal nutrition and gestational diabetes in fetal development and lifelong genetic programming for metabolic status is only now being understood. With the maintenance of maternal and child health programs directed more at these emergent health issues, substantial progress may be made to reduce IRS and related CVD.
6. There has been an excessive focus on the reduction of added sugar as the preferred prevention and management strategy for diabetes. We now know that it is not causal and in amounts up to at least 30 g/day, spread across the day, has no measurable impact on glycaemic status.

The combination of diet and exercise approaches, in those who already have IGT in Da Qing, China, reduces the progression to type II diabetes, but most of the gain was achieved

through exercise.<sup>74</sup> A more comprehensive approach to nutrition measures is likely to provide for more effective prevention and management.

## References

- Colman PG, Thomas DW, Zimmet PZ, Welborn TA, Garcia-Webb P, Moore MP. New classification and criteria for diagnosis of diabetes mellitus. The Australasian Working Party on Diagnostic Criteria for Diabetes Mellitus. *N Z Med J* 1999; 112: 139–41.
- Zimmet PZ, Colman PG, Welborn TA. Problems with new criteria for diagnosis of diabetes mellitus. *Med J Aust* 1999; 171: 108–109.
- Colman PG, Thomas DW, Zimmet PZ, Welborn TA, Garcia-Webb P, Moore MP. New classification and criteria for diagnosis of diabetes mellitus. *Med J Aust* 1999; 170: 375–378.
- Welborn TA, Knuiman MW, Bartholomew HC, Whittall DE. 1989–90 National Health Survey: prevalence of self-reported diabetes in Australia. *Med J Aust* 1995; 163: 129–32.
- Sekikawa A, LaPorte RE. Epidemiology of insulin dependent diabetes mellitus. In: Alberti KGMM, Zimmet P, Defronzo RA, eds. *International textbook of diabetes mellitus*. London: John Wiley & Sons, 1997; 89–96.
- Valle T, Tuomilehto J, Eriksson J. Epidemiology of NIDDM in europids. In: Alberti KGMM, Zimmet P, Defronzo RA, eds. *International textbook of diabetes mellitus*. London: John Wiley & Sons, 1997; 125–142.
- De Courten M, Bennett PH, Tuomilehto J, Zimmet P. Epidemiology of NIDDM in non-europids. In: Alberti KGMM, Zimmet P, Defronzo RA, eds. *International textbook of diabetes mellitus*. London: John Wiley & Sons, 1997; 143–170.
- Ibibebe TI, Hsu-Hage BH-H, Wahlqvist ML, Wattanapenpaiboon N. Prevalence of non insulin dependent diabetes mellitus in South Asian Australians using fasting blood glucose. *Int J Diabetes* 2000; 8: 56–68.
- Shaw JE, Zimmet PZ, de Courten M, Dowse GK, Chitson P, Gareeboo H, Hemraj F, Fareed D, Tuomilehto J, Alberti KG. Impaired fasting glucose or impaired glucose tolerance. What best predicts future diabetes in Mauritius? *Diabetes Care* 1999; 22: 399–402.
- Shaw JE, Hodge AM, de Courten M, Dowse GK, Gareeboo H, Tuomilehto J, Alberti KG, Zimmet PZ. Diabetic neuropathy in Mauritius: prevalence and risk factors. *Diabetes Res Clin Prac* 1998; 42: 131–139.
- Cockram CS. The epidemiology of diabetes mellitus in the Asia-Pacific region. *Hong Kong Med J* 2000; 6: 43–52.
- Nawawi HM, Yazid TN, Ismail F, Khalid BAK. Acute effects of acarbose on post-prandial glucose and triglycerides in type 2 diabetics following intake of different Malaysian foods. *Asia Pac J Clin Nutr* 2000; 9: 41–45.
- Khor GL, Hsu-Hage BH-H, Sundram K, Wahlqvist ML. Prevalence of coronary risk factors in a sample of Chinese women in Kuala Lumpur. *Med J Malaysia* 1997; 52: 367–376.
- Chen X, Hsu-Hage BH-H, Wahlqvist ML, Li Y, Liu X. Cardiovascular risk factor prevalence in three Chinese communities in 1989. *Asia Pac J Clin Nutr* 1995; 4: 278–286.
- Kapantow NH, Rumawas JSP, Schultink WJ, Hsu-Hage BH, Wahlqvist ML. Cardiovascular disease risk profile in adult Chinese living in north Jakarta, Indonesia (with emphasis on coronary heart disease). *Asia Pac J Clin Nutr* 1996; 5: 233–238.
- Hsu-Hage BH-H, Wahlqvist ML. Cardiovascular risk in adult Melbourne Chinese. *Aust J Pub Hlth* 1993; 17: 306–313.
- Dahlquist GG, Blom LG, Persson LA, Sandstrom AI, Wall SG. Dietary factors and the risk of developing insulin dependent diabetes in childhood. *BMJ* 1990; 300: 1302–1306.
- Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? *BMJ* 1995; 311: 437–439.
- Hodge AM, Dowse GK, Zimmet PZ. Diet does not predict incidence or prevalence of non-insulin-dependent diabetes in Nauruans. *Asia Pac J Clin Nutr* 1993; 2: 35–42.
- Husband AJ, Bryden WL. Nutrition, stress and immune activation. *Proc Nutr Soc Aust* 1996; 20: 60–70.
- Pethick DW, Dunshea FR. The partitioning of fat in farm animals. *Proc Nutr Soc Aust* 1996; 20: 3–13.
- Eriksson K-F, Lindgarde F. Prevention of Type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. *Diabetologia* 1991; 34: 891–898.
- Krotkiewski M, Lonroth P, Mandroukas K, Wroblewski Z, Rebuffe-Serive M, Hoi G, Smith U, Bjorntorp P. The effects of physical training on insulin secretion and effectiveness and on glucose metabolism in obesity and Type 2 (non-insulin-dependent) diabetes mellitus. *Diabetologia* 1985; 28: 881–890.
- Deurenberg-Yap M, Li T, Tan WL, van Staveren WA, Deurenberg P. Validation of a semiquantitative food frequency questionnaire for estimation of intakes of energy, fats and cholesterol among Singaporeans. *Asia Pac J Clin Nutr* 2000; 9: 282–288.
- Tam TTT, Gross R, Lukito W, Rumawas JSP. Chronic energy deficiency and relative abdominal overfatness coexist in free-living elderly individuals in Ho Chi Minh City, Vietnam. *Asia Pac J Clin Nutr* 1999; 8: 129–135.
- Barker DJ. Maternal nutrition and cardiovascular disease. *Nutr Health* 1993; 9: 99–106.
- Barker DJ, Osmond C, Simmonds SJ, Wield GA. The relation of small head circumference and thinness at birth to death from cardiovascular disease in adult life. *BMJ* 1993; 306: 422–426.
- Barker DJ, Martyn CN, Osmond C, Hales CN, Fall CH. Growth in utero and serum cholesterol concentrations in adult life. *BMJ* 1993; 307: 1524–1527.
- Barker DJ. The intrauterine origins of cardiovascular disease. *Acta Paediatr Suppl* 1993; 82: 93–99.
- Osmond C, Barker DJ, Winter PD, Fall CH, Simmonds SJ. Early growth and death from cardiovascular disease in women. *BMJ* 1993; 307: 1519–1524.
- Wahlqvist ML. Options in obesity management. *Asia Pac J Clin Nutr* 1992; 1: 183–190.
- Hsu-Hage B, Wahlqvist ML. Food variety of adult Melbourne Chinese: A case study of a population in transition. In: Simopoulous R (eds). *Dietary patterns of selected countries, tea and coffee: metabolic consequences*. World review of nutrition and dietetics, Vol. 79. Basel: Karger, 1996; 53–69.
- Wahlqvist ML. Nutritional pathways to coronary heart disease – An overview. *Patient Management* 1986; 10: 136–143.
- Wahlqvist ML, Dalais FS. Nutrition and cardiovascular disease (Editorial). *Asia Pac J Clin Nutr* 1999; 8: 2–3.
- Welborn TA, Weame K. Coronary heart disease incidence and cardiovascular mortality in Busselton with reference to glucose and insulin concentrations. *Diabetes Care* 1979; 2: 154–160.
- Wahlqvist ML, Lo CS, Myers K, Simpson RW. Plasma insulin and free fatty acids as risk factors for arterial compliance in Type-2 diabetes. *Recent Adv Clin Nutr* 1986; 2: 330–333.
- Wahlqvist ML, Lo CS, Myers KA, Simpson RW, Simpson JM. Putative determinants of arterial wall compliance in NIDDM. *Diabetes Care* 1988; 11: 787–790.
- Carroll KF, Nestel PJ. Diurnal variation in glucose tolerance and insulin secretion in man. *Diabetes* 1973; 22: 333–348.
- Wahlqvist ML, Simpson RW, Lo CS, Cooper P. Preferred meal patterns in non-insulin-dependent diabetes. *Asia Pac J Clin Nutr* 1993; 2: 191–194.
- Jenkins DJA, Wolever TMS, Vuksan V. Nibbling versus gorging: metabolic advantages of increased meal frequency. *N Engl J Med* 1989; 321: 929–934.
- Hodgson JM, Hage B, Wahlqvist ML, Kouris-Blazos A, Lo CS. Development of two food variety scores as measures for the prediction of health outcomes. *Proc Nutr Soc Aust* 1991; 16: 62–65.
- Defronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* 1991; 14: 173–194.
- Hodgson JM, Hsu-Hage BH-H, Wahlqvist ML. Food variety as a quantitative descriptor of food intake. *Ecol Food Nutr* 1994; 32: 137–148.
- Wahlqvist ML, Hsu-Hage B. Food variety of adult Melbourne Chinese: A case study of a population in transition. In: Simopoulous R (eds). *Dietary patterns of selected countries, tea and coffee: metabolic consequences*. World review of nutrition and dietetics, Vol. 79. Basel: Karger, 1996.

45. Wahlqvist ML, Lo CS, Myers KA. Fish intake and arterial wall characteristics in healthy people and diabetic patients. *Lancet* 1989; 2: 944–946.
46. Surwit RS, Feinglos MN. Stress and autonomic nervous system in type II diabetes: a hypothesis. *Diabetes Care* 1988; 11: 83–85.
47. Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC. Dietary fiber, glycaemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* 1997; 222: 472–477.
48. Wahlqvist ML, Wilmshurst EG, Murton CR, Richardson EN. The effect of chain length on glucose absorption and the related metabolic response. *Am J Clin Nutrition* 1978; 31: 1998–2001.
49. Jenkins DJ, Goff DV, Leeds AR, Alberti KG, Wolever TM, Gassull MA, Hockaday TD. Unabsorbable carbohydrates and diabetes: decreased post-prandial hyperglycaemia. *Lancet* 1976; 2: 172–174.
50. Wahlqvist ML, Lo CS, Myers KA. Food variety is associated with less macrovascular disease in those with Type II diabetes and their healthy controls. *J Am Coll Nutr* 1989; 8: 515–523.
51. Wahlqvist ML. Nutritional aspects of diabetes (Editorial). *Int J Diabetes* 2001; in press.
52. Cooper PL, Wahlqvist ML, Simpson RW. Sucrose versus saccharin as an added sweetener in non-insulin-dependent diabetes: short and medium term metabolic effects. *Diabetic Med* 1988; 5: 676–680.
53. Burr ML, Fehily AM, Gilbert JF, Rogers S, Holliday RM, Sweetnam PM, Elwood PC, Deadman NM. Effects of changes in fat, fish, and fibre intakes on death and myocardial infarction. Diet Reinfarction Trial (DART). *Lancet* 1989; 2: 757–761.
54. Brand JC. The glycaemic index of foods. *Asia Pac J Clin Nutr* 1993; 2: 107–110.
55. Truswell AS. Glycaemic index of foods. *Eur J Clin Nutr* 1992; 46: S91–S101.
56. Brand JC, Colagiuri S, Crossman S, Allen A, Roberts DCK, Truswell AS. Low-glycemic index foods improve long-term glycemic control in NIDDM. *Diabetes Care* 1991; 14: 95–101.
57. Brand Miller JC, Foster-Powell K, Colagiuri S. The GI factor. Sydney: Hodder and Stoughton, 1996.
58. Swinburn BA, Boyce VL, Bergman RN, Howard BV, Bogardus C. Deterioration in carbohydrate metabolism and lipoprotein changes induced by modern, high fat diet in pima Indians and Caucasians. *J Clin Endocrinol Metab* 1991; 73: 156–165.
59. Storlien L, Jenkins A, Chisholm D, Pascoe W, Khouri S, Kraegen E. Influence of dietary fat composition on development of insulin resistance in rats. Relationship to muscle triglyceride and  $\omega$ -3 fatty acids in muscle phospholipid. *Diabetes* 1991; 40: 280–289.
60. Garg A, Bonanome A, Grundy SM, Zhang ZJ, Unger RH. Comparison of a high-carbohydrate diet with a high-monounsaturated-fat diet in patients with non-insulin-dependent diabetes mellitus. *N Engl J Med* 1988; 319: 829–834.
61. Borkman M, Storlien LH, Pan DA, Jenkins AB, Chisholm DJ, Campbell LV. The relation between insulin sensitivity and the fatty-acid composition of skeletal-muscle phospholipids. *N Engl J Med* 1993; 328: 238–244.
62. Dahlquist G, Blom L, Lonnberg G. The Swedish childhood diabetes study – a multivariate analysis of risk determinants for diabetes in different age groups. *Diabetologia* 1991; 34: 757–762.
63. Wei M, Gibbons LW, Mitchell TL, Kampert JB, Blair SN. Alcohol intake and risk of diabetes. *Diabetes Care* 2000; 23: 18–22.
64. Parker DR, McPhillips JB, Lapane KL, Lasater TM, Carleton RA. Nutrition and health practices of diabetic and nondiabetic men and women from two southeastern New England communities. *Nutr & Health* 1995; 10: 255–268.
65. Davis WK, Hess GE, Hiss RG. Psychosocial correlates of survival in diabetes. *Diabetes Care* 1988; 11: 538–545.
66. Glasgow RE, Toobert DJ. Social environment and regimen adherence among type II diabetic patients. *Diabetes Care* 1988; 11: 377–386.
67. Wahlqvist ML. Clinicians changing individual food habits. *Asia Pac J Clin Nutr* 2000; 9: S55–S59.
68. Simpson RW, McDonald J, Wahlqvist ML, Balasz N, Sissons M, Atley L. Temporal study of metabolic change when non-insulin dependent diabetics changed from a low to high carbohydrate-fibre diet. *Am J Clin Nutr* 1988; 48: 104–109.
69. Mann JI. The role of nutritional modifications in the prevention of macrovascular complications of diabetes. *Diabetes* 1997; 46: S125–S130.
70. Storlien LH, Kriketos AD, Jenkins AB, Baur LA, Pan DA, Tapsell LC, Calvert GD. Does dietary fat influence insulin action? *Ann N Y Acad Sci* 1997; 827: 287–301.
71. Wells AS, Read NW, Uvnas-Moberg K, Alster P. Influences of fat and carbohydrate on postprandial sleepiness, mood, and hormones. *Physiol Behav* 1997; 61: 679–86.
72. Tsihlias EB, Gibbs AL, McBurney MI, Wolever TM. Comparison of high- and low-glycemic-index breakfast cereals with mono-unsaturated fat in the long-term dietary management of type 2 diabetes. *Am J Clin Nutr* 2000; 72: 439–49.
73. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and diabetes study. *Diabetes Care* 1997; 20: 537–44.
74. DCCT Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* 1993; 329: 977–986.
75. Ginsberg HN. Lipoprotein physiology in non-diabetic and diabetic states. Relationship to atherogenesis. *Diabetes Care* 1991; 14: 839–855.
76. Stern MP, Haffner SM. Dyslipidemia in Type II diabetes. Implications for therapeutic intervention. *Diabetes Care* 1991; 14: 1144–1159.
77. Jones AF, Lunec J. Protein fluorescence and its relationship to free radical activity. *Br J Cancer* 1987; 55: 60–65.
78. Duncan JJ, Gordon NF, Scott CB. Women walking to: health and fitness. How much is enough? *JAMA* 1991; 266: 3295–3299.
79. Borkman M, Storlien LH, Pan DA, Jenkins AB, Chisholm DJ, Campbell LV. The relation between insulin sensitivity and the fatty-acid composition of skeletal-muscle phospholipids. *N Engl J Med* 1993; 328: 238–244.
80. Depres JP. Metabolic dysfunction and exercise. In: Wahlqvist M, Hills A, eds. Exercise and obesity. London: Smith-Gordon, 1995.
81. Lassers BW, Wahlqvist ML, Kaijser L, Carison LA. Relationship in man between plasma free fatty acids and myocardial metabolism of carbohydrate substrates. *Lancet* 1971; 2: 448–450.
82. Brand Miller J, Foster-Powell K. GI Plus. Sydney: Hodder and Stoughton, 2000.
83. Guevarra MTB, Paniasigui LN. Blood glucose responses of diabetes mellitus type II patients to some local fruits. *Asia Pac J Clin Nutr* 2000; 9: 303–308.
84. Mani UV, Prabhu BM, Damle SS, Mani I. Glycaemic index of some commonly consumed foods in western India. *Asia Pac J Clin Nutr* 1993; 2: 111–114.
85. Batra M, Sharma S, Seth V. The glycaemic index of fermented and non-fermented legume based snack food. *Asia Pac J Clin Nutr* 1994; 3: 151–154.