The topic of obesity in relation to nutrition was discussed in two recently published articles in this Bulletin1, 2. This review will recapitulate some of the earlier points made and will present evidence to show that malnutrition in childhood, a persisting problem in developing countries in Asia, is causally linked to obesity in adulthood. Adult onset obesity, and more particularly abdominal obesity, is associated with increased risk of morbidity in South Asians. These include the importance of abdominal obesity in the development of insulin resistance, and in the metabolic syndrome X which is characterised by hyperinsulinaemia, dyslipidaemia, glucose intolerance and hypertension.

This review will briefly outline the anthropometric criteria for obesity and for abdominal obesity in adults and will then highlight the important morphological and metabolic differences in abdominal adipose tissue as compared to subcutaneous adipose tissue. In the last section of this review, evidence, both animal experimental and human, will be presented to show the link between malnutrition and obesity with the predisposition to abdominal obesity in particular.

The Nutrition Foundation of India recently brought out a Scientific Report3 detailing the results of a study on obesity carried out among the ‘middle class’ and among the slum dwellers of Delhi. The report highlights the growing problem of obesity in India and in other developing countries. It also lays special emphasis on abdominal obesity.

INDICATORS OF OBESITY

Obesity is defined as a condition in which excess body fat is accumulated, leading to adverse effects on health as a result of associated co-morbidities such as coronary heart disease (CHD), non-insulin dependent diabetes mellitus (NIDDM), certain cancers, gall bladder disease, osteo-arthritis, etc. Body Mass Index (BMI; weight in kg/height in metres2) provides the most useful population level measure of obesity. Within populations, a BMI greater than 30 is associated with elevated blood pressure and an increased risk of NIDDM and CHD. Hence, the WHO Expert Committee Report4 accepted a BMI of 18.5 to 24.9 as the normal range and classified overweight in three grades from a BMI above 25. A more recent report of a WHO Consultation on Obesity, published in 1998, modified this classification by categorising a BMI between 25 to 29.9 as pre-obese with three further grades of obesity with BMIs over 30, 35 and 40, respectively.

Abdominal accumulation of fat results in abdominal obesity. The abdominal fat mass in any individual can vary for the same BMI and for the same amount of total body fat. There are marked sex differences in the proportion of abdominal fat mass; for any accumulation of total body fat, males on an average have twice as much abdominal fat as compared to pre-menopausal females. In addition, different population or ethnic groups have marked differences in the risk of co-morbidities associated with abdominal fat accumulation. Abdominal obesity is less strongly associated with risk factors for CHD and NIDDM in black African-American women as compared to white European women in the USA. In the Asian region, South Asians are more prone to the associated co-morbidities of abdominal obesity and also have excess abdominal fat for a comparable BMI than Europeans5.

The criterion for the clinical diagnosis of abdominal obesity has been the use of the waist-to-hip ratio (WHR). A WHR of > 1.0 for men and > 0.85 for women has been accepted as an indicator of abdominal obesity. More recently, the use of waist circumference alone has been advocated6. The latter is a convenient and easy measurement to make. It is independent of height and correlates well with both BMI and WHR and thus is a reasonably good indicator of intra-abdominal body fat and total body fat7.

SUBCUTANEOUS AND ABDOMINAL ADIPOSE TISSUES

There are marked morphological differences between intra-abdominal adipose tissue (IAT) and subcutaneous adipose tissue (SAT) depots. IAT fat cells are smaller and hence have more cells per unit mass of adipose tissue and a higher blood flow. IAT also has more glucocorticoid or cortisol receptors as well as more androgen or testosterone receptors and demonstrate a greater catecholamine-induced lipolysis. These morphological differences in IAT cells result in metabolic and hormonal differences in their function, which may have important implications. SAT cells are more susceptible to changes in lipid accumulation and to hormonal stimulation. Since IAT adipocytes are upstream to the liver in the portal circulation, specific hormonal stimulation can increase the flux of fatty acids to the liver via the portal circulation, particularly in those with abdominal obesity.

Adipocytes are not passive stores of energy in the body. They function like endocrine cells and secrete hormones that act locally and also distal to the point of production. An example is leptin, which is secreted by adipocytes. Adipocytes are also the target cells for the actions of a wide range of hormones in the body and hence have numerous hormone receptors on their cell surfaces. Hence, abdominal obesity is associated with several hormonal abnormalities7 such as insulin resistance and the consequent increased insulin secretion which may occur as an adaptation to limit the further accumulation of body fat deposition and the resultant increase in body weight.

There are several abnormalities related to reproductive hormone functions associated with abdominal obesity. These include a decrease in sex hormone binding globulin levels in women resulting in an increase in the clearance of free testosterone and oestradiol. Abdominal obesity in women is associated with an increase in free testosterone and free androstenedione levels and a decrease in progesterone levels. In men, on the other hand, abdominal obesity is associated with a reduction in testosterone levels. These hormonal abnormalities may explain why abdominal obesity is associated with ovulatory dysfunction, hyperandrogenism, polycystic ovaries and some...
hormone sensitive cancers in women. Increased cortisol production, which may contribute to the manifestation of insulin resistance and decreased growth hormone levels, are other abnormalities seen in abdominal obesity.

MALNUTRITION AND OBESITY

The risk of intra-abdominal accumulation of fat can begin early in life and may be concurrent with the development of obesity in childhood. Obesity in children is also associated with higher insulin levels indicating that the consequence of abdominal fat accumulation is also evident quite early in childhood. Prenatal influences other than malnutrition can increase the risk of later obesity. These include the fact that children of diabetic mothers are fatter at birth and at ages five to nine years, 10-14 years and 15-19 years. More recent studies by Whitaker and others have shown that infants of mothers who required insulin had a greater prevalence of obesity at age seven compared to mothers with glucose intolerance who did not need insulin. Either the severity of the glucose intolerance or the use of insulin during pregnancy may represent important risk factors for childhood obesity.

Protein intake at two years of age was associated with the development of fatness and an early adiposity rebound which may be suggestive of early maturation and association with increased adiposity in adulthood.

ABDOMINAL OBESITY

Prenatal malnutrition: The Dutch famine was a natural experiment that occurred during the Second World War and demonstrates the effects of food restriction, its timing and duration as well as the effect of refeeding on subsequent obesity. The cohort born to mothers who were exposed to food restriction in the last trimester had a reduced prevalence of obesity at the age of 18 years as compared to those who were born to mothers who were exposed to undernutrition during the first two trimesters of pregnancy. The third trimester represents a period of rapid adipocyte replication and thus is a time of rapid increase in body fat. Food restriction during this period impairs fat cell development that may lead to consequent leanness. In contrast, the increased prevalence of obesity among individuals who were born to mothers exposed to famine in the first and second trimester may be explained by their impact on the development of the hypothalamus and a sympathetic nervous system which may influence subsequent energy metabolism and feeding behaviour. An alternate explanation is that refeeding may promote adipocyte replication and development leading to obesity in adult life. Whatever the mechanism, it is evident that prenatal undernutrition increases the risk of later obesity.

Low birth-weights and obesity in adult life: Barker's hypothesis links low birth-weight and size at birth resulting from poor maternal nutrition as being important markers that programme the individual to having an increased risk of a range of chronic non-communicable diseases such as CHD and NIDDM. Law and others have looked at the possibility that abdominal obesity is associated with retarded growth in foetal life and, subsequently, during infancy. They found a higher WHR and BMI with decreased growth during early life and infancy. They also showed that for any level of obesity there was more abdominal fat in men who had weighed less at birth. These findings suggest that abdominal obesity is programmed and inversely related to early growth.

Childhood undernutrition and abdominal obesity: There is considerable animal experimental evidence to support the hypothesis that undernutrition or food restriction post-natally can influence the later development of obesity. There are several studies which demonstrate that food restricted rats following access to ad libitum food intakes, deposit increased body fat. Ozelci and others showed that the increased body-weight gain and body fat accumulation was only seen when energy deficient rats were pair fed on an intake basis and not when they were pair fed on a body weight basis as compared to controls. A similar increase in body fat gain has been demonstrated even in malnourished children who were nutritionally rehabilitated. For instance, Ashworth reported that growth rates were 15 times as fast during recovery in malnourished children as compared to that of normal children of similar age and five times as fast as those of normal children with similar height and weight. She also reported an increase in per cent body fat once the expected height and weight was reached. Fjeld and others showed a rise in fat deposition when they expressed fat gained as a proportion of the total weight gained during catch-up growth in malnourished children.

This was influenced very little by either the rate of recovery and was seen both during early and late recovery following nutritional rehabilitation.

Whether there is an increased tendency to deposit fat intra-abdominally or whether the nature of the diet during rehabilitation influences this fat gain in any way has not been investigated in children. However, animal experiments on rats following preweaning nutritional deprivation show that there is a tendency to selectively deposit fat intra-abdominally even when the total fat gain is less than that of normal rats of a similar age.

These studies also show that the intra-abdominal fat cell numbers are restored while the deficits in subcutaneous fat cell numbers persist at 28 weeks of age. Animal experiments also seem to suggest that the type of fat in the diet during refeeding can influence the gain in visceral adipose tissue. The latter study suggests that n-3 polyunsaturated fats lead to significantly lower visceral fat deposition as compared to n-6 polyunsaturated and saturated fats in the diet.

Hypothalamic-pituitary-adrenal axis: Björntorp has suggested that the pattern of endocrine perturbations associated with abdominal obesity, that is, elevated cortisol, increased insulin secretion and raised androgens (in women) along with low levels of growth hormone, may contribute both to the abdominal fat accumulation and insulin resistance.

There is compelling evidence to show that this is due to a hypersensitive hypothalamic-pituitary-adrenal (HPA) axis. This feature and the increase in abdominal obesity is seen in psycho-social conditions associated with stress and is a feature of life in lower social classes with low education, unemployment and work stress as well as low levels of physical activity. This may explain the increase in the prevalence of obesity and related co-morbidities among lower social
classes in the Whitehall studies21. There is more recent evidence suggesting that salivary cortisol levels are higher in stunted children and that stress levels are also higher during psychological testing of such stunted children in Jamaica as compared to normal children22. This is further evidence of a mechanism that may operate to perpetuate obesity and abdominal obesity in malnourished populations in developing societies.

Stunting and obesity: There is considerable evidence now that suggests a close link between increase in stunting in populations in developing societies and the emerging epidemic of obesity in their midst. As discussed above, several factors may be operating to further this link between malnutrition in childhood and obesity in these populations. Stunting in children is likely to alter the relationship of the appropriateness of weight for height in individuals with short stature and thus exaggerate the apparent prevalence of obesity in a population.

Irrespective of the method of classification utilised, the use of this commonly used indicator of pre-school childhood malnutrition (weight for height) may exaggerate the problem of obesity among the stunted children. It is also believed that the discordance between linear growth and adipocyte development will enhance adipocyte development when linear growth is affected by malnutrition. Thus, despite the fact that BMI is chosen as an indicator for the diagnosis of obesity because of its relative independence from height, a stunted individual with a short stature has to gain relatively little weight as compared to taller individuals to end up with a higher BMI. These smaller gains in weight over time resulting from the economic development can result in an apparent exaggerated problem of obesity in developing societies.

CONCLUSIONS

Malnourished populations in developing societies, particularly in Asia, have an increased predisposition to obesity and, more specifically, to abdominal obesity. The causal link between the two operates by a range of mechanisms. Recovery from one or several episodes of undernutrition results in alterations in body composition, increase in the amount of fat gained and probably an increase in intra-abdominal fat, deposition also. This altered body composition, manifesting with a reduced lean tissue mass, results in a lower Basal Metabolic Rate and reduced energy costs of physical activity. There may be changes in the ability to regulate food intake and also alterations in the ability to oxidise fat. All these changes, as a result of prenatal and post-natal malnutrition, will increase susceptibility to obesity under the right environmental influences such as an increased intake of fat in the diet and reduced levels of physical activity. These environmental changes are now characteristic of economic development and urbanisation in these countries and will hence fuel the epidemic of obesity currently seen in these societies.

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References


NUTRITION NEWS

The XXXII Annual Meeting of the Nutrition Society was held in Coimbatore on November 25-26. Dr Rajamall P. Devadas, Chancellor of Avinashilingam University, was felicitated on her 80th birth anniversary. The meeting was attended by over 500 scientists. The programme included two special symposia, the Gopalan Oration and the Srikantia Memorial Lecture, and free presentations by young scientists.

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