Editorial

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Diabetes and nutrition are inseparably linked. It is usually accepted that overnutrition and obesity play an important role in the pathogenesis of 'type 2' diabetes, especially in the developed world. It was therefore, a surprise when workers in the developing countries reported a 'type' of diabetes common amongst the poor and the undernourished. In 1950's and 60's Hugh Jones, Zuidema, Tripathy, Ahuja and Geevarghese reported from different tropical countries a number of severely malnourished young patients with diabetes. These patients were severely hyperglycaemic and needed insulin treatment, sometimes in large doses. Surprisingly, they did not develop ketosis when insulin treatment was stopped for prolonged periods (because of socioeconomic reasons). Some of these patients suffered from chronic pancreatitis and pancreatic calculi could be demonstrated on X-ray of the abdomen (tropical pancreatic diabetes), in others there was no evidence of exocrine pancreatic involvement (J – type). Since severe malnutrition was the most striking clinical feature of these patients it was suggested that malnutrition was an aetiological factor, though there was no proof of this. The temporal relationship between nutritional deprivation and development of diabetes was not known. The relationship, if any, between the two major types described ('J-type' and the 'tropical pancreatic diabetes') was also not clear. 'Nutritional diabetes' remained an intriguing clinical syndrome.

Stronger representation from the developing countries in the international organizations led to a broad recognition that diabetes in the developing countries ('third world') could have a different profile than that in the developed world . It was suggested that undernutrition must be an important factor in these differences. The above mentioned two types of diabetes ('protein deficient pancreatic diabetes-PDPD' and 'fibrocalculous pancreatic diabetes-FCPD') were clubbed under a new class of diabetes called 'malnutrition related diabetes (MRDM)'. Geopolitical rather than scientific interests diverted the attention from the basic and the more interesting issues of aetiopathogenesis of diabetes in the developing world to definition of a new syndrome. Changing nutritional status in different populations altered the clinical picture and over a period of time fewer patients with classical description were seen. Aetiological role of malnutrition in human diabetes remained unproven for lack of good prospective studies, the term MRDM reflected more an article of faith.

In recent years young workers in the developing countries have helped improve our understanding of the so called MRDM. They did not restrict their studies to clinical (phenotypic) characteristics of patients. International collaborations helped application of modern techniques. Analysis of the more specific markers of type 1diabetes (HLA and pancreatic islet cell antibodies), revealed an overlap between MRDM (J-type as well as FCPD) and type 1 diabetes. Absence of such markers for 'type 2' diabetes (and rather poor overall understanding of this type of diabetes) make such studies difficult. Classically, the syndrome of type 2 diabetes is thought to result when modern life-style factors (overnutrition, poor exercise etc.) act on the background of a genetic predisposition ('thrifty genotype'). No genetic markers are however, yet described. In general it is thought to be a disease of the affluent but recent experience suggests a rapid rise in some developing countries, probably due to changes in lifestyle. A new thought about possible role of 'malnutrition' in the aetiology of 'type2' diabetes has raised considerable interest. Retrospective, epidemiologic studies in the U.K. demonstrated an association of low birth weight and poor early growth with development of diabetes (and hypertension and coronary artery disease) in the adult life (a 'thirty phenotype' or 'small baby syndrome'). Animal experiments suggest that maternal and early life malnutrition impair B-cell function in later life. It is not difficult to envisage the enormous influence early life (intrauterine and early childhood) factors could have on the profile of disease in adult life if we remember that a major share (up to 90%) of the divisions that the fertilized ovum undergoes to reach adult size take place before birth. Environmental influences acting at crucial developmental stages could have a profound 'programming' effect on different tissues and organs of the body Postnatal lifestyle factors could then act on the background of such developmental susceptibility (as opposed to the 'genetic' predisposition). If proved correct, this hypothesis offers possibilities for prevention of the so called diseases of civilization by treatment of pregnant mothers. Need for prospective studies in humans cannot be overemphasized. Diabetes secondary to early life malnutrition and maldevelopment appears a real possibility. Nutritional

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status at the time of diagnosis of diabetes (the mainstay of diagnosis of present day MRDM) obviously doesn't tell us much by way of aetiology. The stress should be on studying the effect of malnutrition in early life on later development of diabetes rather than defining a 'type' of diabetes based on nutritional status at the time of diagnosis.

In this issue various authors contribute their views on different aspect of MRDM. Profs. Tripathy and Samal report their experience of PDDM from Orissa, India, in defence of the original concept of MRDM. Mohan reviews FCPD from India. Abdulkadir narrates the experience from Africa, including his studies which show an overlap between PDPD and 'type 1' insulin dependent diabetes. Srikanta summarises the clinical, biochemical, endocrine and immunological studies of childhood diabetes in North India and again suggests that PDPD is type 1 diabetes indisguise. Ramchandran highlights the characteristics of 'type 2' diabetes in the young Indians to suggest reasons for the possible overlap with MRDM. Prof. Seshiah's group from Madras narrates the experience from a public hospital. Finally, Prof. Barker reviews his studies suggesting a link between low birth weight and adult diabetes.

Obviously, the last chapter in MRDM is still some distance away.