

# EDITORIAL

## EXPANDED SCOPE OF DIABETES BULLETIN

Learning resource material, especially in subjects such as diabetes mellitus is scarcely available in Asian and other developing countries. Available information often is not relevant or applicable to local conditions and is also very expensive.

Ethnic and ecological factors contribute to a rather low sensitivity to precepts of health awareness or health care, especially relating to a chronic ailment in these countries, even amongst professionals. Population profile in age structure (children constitute 40% of population), nutritional status (poor resources, ten to twenty times less than the West), and life expectancy (at least two decades less than in the West) add to alter or modify the natural history of diseases like diabetes in the developing countries. There is a tremendous scope to present the facts on the variability of natural history of complication of diabetes in these geographical regions.

With advancements of science and induction of new technology, there is need to harness resources to make the new information of relevance and practical value available to the community. This information should be used optimally and be cost effective. For this purpose, many innovations need to be promoted and their usefulness be known to the practising physician at an affordable cost through the medium of a scientific publication.

Emphasis as such would deserve to be on preventive aspects of diabetes and a holistic approach to the disease and its social sequelae relevant to the developing countries.

This publication has the onus of fulfilling these objectives. It can serve as a resource material and practical guide for those who are engaged in the care of diabetics in the developing countries.

## CHILDHOOD DIABETES IN THE DEVELOPING COUNTRIES

Insulin dependent diabetes (IDDM) is a universal disease. While heterogeneity is well recognized in IDDM, conventional IDDM, characterized by immunogenic associations, absolute insulin deficiency (loss of C-peptide reserve) and presence of islet cell antibodies, is distinctive.

HLA typing (population and family studies) has been indicative of DR3 and/or DR4 to confer significantly high risk for IDDM in most populations (in Japanese DR4, W7, W53, BW54). In all races studied so far, DR2 has been shown to have a "protective" influence against the development of IDDM.

Islet cell autoantibodies show positivity in upto 75% of IDDM subjects at the time of clinical diagnosis, declining to 25% by one year, and then on continuing the same trend. Spontaneous insulin autoantibodies are additional markers of islet cell autoimmunity and beta cell destruction, particularly in younger children; its levels appear to reflect the rate of beta cell destruction. More important, these two autoantibodies appear in the circulation five to ten years or even more prior to presentation with clinical IDDM or ketoacidosis. Early screening may identity the onslaught of autoimmune beta cell damage at a stage much earlier to the advent of clinical diabetes.

Environment factors speculated include infectious agents (congenital rubella, mumps) and dietary factors (complex proteins). Risk of 'endemicity' is being related to the average yearly temperature of environment in a population. In some countries, seasonality and epidemic forms have been recognized, but this is not universal.

Incidence differences in IDDM in Finland and Japan are exemplary, 36 times more frequent in Finland than Japan; again countries close to the equator have the lowest risk.

The incidence of IDDM is seemingly increasing world-wide, and it has been observed that in some countries, such as Poland and New Zealand, the rates of childhood diabetes have doubled in a span of 5 years.

With this background, the developing countries are so far 'apparently' low risk populations for IDDM. The scene is already changing, and today with increasing awareness and available diagnostic facilities, there is now better recognition of IDDM amongst children in these economically poorer countries. The "childhood registries" now launched under the auspices of Diabetes Epidemiology Research International & W.H.O. are unraveling the correct magnitude.

Based on this limited data, are there possible hypotheses for the lower rates observed in certain populations? In some underdeveloped and developing societies, with limited health care facilities, "death before diagnosis" can significantly contribute to the lower rates observed.

Is it possible that the genetic population profile is at variation? Anthropological ethnic admixture is protective as far as diabetes prevalence rates are concerned.

Racial types recognized in India include Indo-Aryans, Dravidians and Mongolians who over time have freely mixed with each other. Again, a number of 'foreign' invasions over centuries has led to further admixture of population as it exists today.

Amongst the environmental factors, the rates of various infections are high in developing countries; this may have diverse effects on the immune system. Malnutrition related immunodeficiency and other nutritional variables can be potentially protective.

Furthermore, data suggest that the profile of IDDM, as observed in developing countries, is distinctive in certain aspects: age of onset is late and in a subset of patients, residual beta cell function (C-peptide reserve) is relatively better preserved, so that ketoacidosis at the onset of diabetes may not be frequent.

There is need for further investigative research in these aspects as epidemiological studies may provide useful clues towards the prevention or more effective management of IDDM.