graduate training for all doctors. Clinicians will increasingly recognise that they have interests in common with community physicians to improve the health of the district in their specialty through prevention, and to sustain their patients in the community after hospital care, with the general practitioner.

The Chief Medical Officer designate for England and Wales, Professor Donald Acheson, has been at the forefront of raising interest in clinical epidemiology.¹⁴ We can expect him to support the royal colleges' initiative; but can he also move the Körner steering group along more fruitful paths?

MARK MCCARTHY

Senior Lecturer in Community Medicine,
University College London,
London WC1E 6JJ

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Update on insulin dependent diabetes

The pace of research into the aetiology and pathogenesis of insulin dependent (type I) diabetes has increased over the past 10 years and this has been reflected in a series of international symposia and workshops. The most recent was the fourth international workshop on "The immunology of insulin dependent diabetes mellitus," held in London in April. The need for progress in our understanding of the aetiology of this form of diabetes was emphasised at the outset by J Nerup (Denmark), who pointed to the failure of modern insulin treatment appreciably to reduce long term morbidity.

The characteristic presentation of insulin dependent diabetes has fostered the assumption that most cases result from an acute pathological event occurring just before the onset of symptoms. Perhaps the most important change in our concept of the disorder is the realisation that, in some cases at least, damage to the islet cells probably occurs over several years before the severe metabolic breakdown that results in symptoms. This may occur even in those patients

who have had an acute infective episode before the clinical onset of diabetes.

W Gepts (Belgium) reviewed the histological findings and emphasised that these may vary from case to case. Cellular infiltration of the islet cells is only one of the features seen; other changes include pseudoatrophy, which suggests that damage to the islet cells occurs over a long time. Studies of diabetes induced by viruses and chemical agents—for example, cytomegalovirus, reovirus, streptozotocin (A L Notkins, United States; A D Toniolo, Italy)—have suggested that sequential exposure to such agents may lead to cumulative damage to the β cells of the pancreas. D C Gajdusek (United States) pointed out that some viruses may remain in the tissues for many years before they exert a cytopathic effect, the target tissue often being highly specific for a particular virus. F Ginsberg-Fellner (United States) reported the development of diabetes in 18 people out of 600 who had suffered from congenital rubella. In some cases the diabetes had presented as late as 14 years of age.

Further evidence comes from advances in our knowledge of the genetics of insulin dependent diabetes with the possibility through HLA typing of ascertaining certain highly susceptible subjects. This has been done in the Barts-Windsor family study, and some of the findings from this study were reported by K Spencer and P Pozzilli (Britain). Complement fixing islet cell antibodies, particularly in the healthy haploidentical siblings of probands, were detectable for as long as three years before the clinical onset of diabetes. Moreover, there was preliminary evidence in some of these subjects of an increased number of activated T cells and of a fall in T suppressor-cytotoxic cells in the peripheral blood.

It is possible that the destruction of β cells is mediated via immunological processes irrespective of the environmental trigger. This theory was raised several years ago and is supported by the presence of islet cell antibodies in the serum of most patients at the time of clinical presentation. G F Bottazzo (Britain) described further findings such as the presence of IgG at the surface of and within islet cells, with IgG bearing lymphocytes and activated T lymphocytes infiltrating the islets in patchy fashion. These findings are consistent with the concept that destruction of β cells may result from the synergistic action of both humoral and cell mediated attack. The potential pathological effects of antibody were shown by experiments reported by A Lernmark (Denmark), in which insulin release by isolated islet cells after a glucose stimulus was appreciably reduced when the cells were perfused with IgG fractions of sera from diabetics.

Review of the genetic aspects of insulin dependent diabetes showed general agreement that more than one HLA linked susceptibility gene is implicated. Three high risk haplotypes are known, two including DR3 and one DR4. We do not know where exactly along the chromosome the relevant genes are, but rapidly developing knowledge of the closely linked polymorphisms in the DR region should bring us closer to the loci which actually determine who is susceptible to diabetes.

Looking to the future, we should like to be able to detect susceptible people and protect them from the environmental trigger factors or to block the processes that lead to destruction of β cells. Only an optimist would claim that we shall be able to achieve this in the near future, but current research is—at the very least—marking out the directions in which further information is urgently sought.

J C WOODROW

Professor of Medicine,
Department of Medicine,
University of Liverpool,
Liverpool L69 3BX