

Epidemiological data suggesting an infectious origin of diabetes pre-date the discovery of insulin; indeed it was the variation in mortality rates from diabetes that led Gunderson to hypothesise that a virus with 'selective affinity for the pancreas' may cause 'acute diabetes' in youth (1). He noted an increase in deaths from diabetes in young people aged 10-20 yr in Norway from 1900 to 1921 following epidemics of parotitis, with a lag time of 3-4 yr between infection and death. In Norway, Denmark, France, and America, the increase in deaths from diabetes exceeded the expected number based on population growth; lending further weight to the proposal that diabetes was caused by infection. Since that time, a large body of epidemiological, clinical and experimental research, in humans, cellular and animal models, has provided further insights into the contribution of infections in the development of type 1 diabetes. Epidemiological evidence for a viral aetiology of diabetes A substantial body of epidemiological data point to a significant contribution of the environment in the development of type 1 diabetes, although much of the evidence is not specific to viruses per se. These data include rising rates of type 1 diabetes in both developed and developing countries in recent decades (2, 3) and a reduced contribution of high risk human leucocyte antigen (HLA) genotypes (4, 5), indicating that non-genetic factors are important. Similarly, the pairwise concordance between monozygotic twins for type 1 diabetes of less than 40%, and the observation that the incidence of diabetes in migrant children reflects that of their adopted country (6, 7), provide circumstantial evidence that environmental agents contribute to the disease. Space-time clustering in the presentation of type 1 diabetes (8-10) and clustering of births in children who subsequently develop diabetes (11) support a direct role for infections in the initiation and acceleration of the disease process.

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