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Insulin resistance in the prevention and treatment of type 1 diabetes

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Diabetes was considered a single entity until 40 years ago, when three observations made largely in children (lymphocytic insulitis, islet cell antibodies and HLA linkage) were interpreted by opinion leaders at the time to mean that childhood diabetes was caused by dysregulation of the immune system (autoimmunity). A previously single disorder was now viewed as two categorically distinct entities of different aetiology, and the autoimmune paradigm has become deeply rooted since. Importantly, however, the new classification into T1D (autoimmune) and T2D (metabolic) diabetes was based on observation, not on experiment, and some 20 human trials using immunotherapy to test the autoimmunity paradigm since have proved disappointing. None has translated into patient benefit, and it seems appropriate to ask whether the original interpretation was correct. An alternative paradigm (the accelerator hypothesis) proposes that insulin resistance, linked largely to body fat, drives all diabetes, and that the tempo of beta cell loss is modulated by the HLA-dependent immune response. Indeed, children who go on to develop islet cell autoimmunity and T1D tend to be heavier, and insulin resistance is an established independent risk factor for T1D. T2D and T1D are viewed by the hypothesis as the polar extremes of a continuous spectrum that extends from slow (presents late, because there is little immune response) to fast (presents early, because there is an intense immune response). A prevention trial using metformin ('APT for childhood diabetes') is currently under review to establish whether insulin resensitisation can slow beta cell loss and prevent T1D.

Biography

Terence Wilkin was trained at the Universities of St Andrews and Dundee in Scotland. His research was subsequently funded for 15 years by the Wellcome Trust at the Universities of Montpellier (France) and Southampton (UK). He s currently professor of endocrinology at the University of Exeter Medical School, and formulated the accelerator hypothesis in 2001.

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