**Electronic supplementary material**

## Methods

Populations studied

*Type 1 diabetes family collection* Type 1 diabetes families were white European or of white European descent, with two parents and at least one affected child comprising DNA samples from up to 919 Finnish multiplex/simplex families [1], 469 multiplex Diabetes UK Warren I families [2], 335 multiplex Human Biological Data Interchange families [3], 263 Belfast multiplex/simplex families [4], 261 Romanian simplex families [5], 112 Norwegian simplex families [6] and 80 Yorkshire simplex families [7]. The populations studied have been described previously [8]. All DNA samples were collected after informed consent was obtained.

*The Avon Longitudinal Study of Parents And Children* Details of this birth cohort were previously reported [9] and are also available on the ALSPAC website (www.alspac.bris.ac.uk). The children in this study are from a 10% 'Children in Focus' sub-cohort (1,335 full-term singleton infants) randomly selected from the last 6 months of recruitment for more detailed measurements of infant and childhood growth. Ethical approval was obtained from the three local Ethics Committees covering the study area and from the ALSPAC Ethics committee. DNA was prepared as described previously [10].

ALSPAC growth data

Birthweight was noted from hospital records, and length and head circumference were measured soon after birth by the ALSPAC study team [11]. At age 7 years (mean

age 7.5±0.1 years), body weight was measured using electronic scales and standing height was measured by stadiometer (Leicester height measure; Child Growth Foundation, London, UK) [12]. Body composition was assessed at age 9 years by whole-body dual-energy x-ray absorptiometry [13]. Internal SD scores were calculated for all parameters of growth as previously described [11] in order to adjust for age and sex.

ALSPAC IGF-1 concentrations

IGF-1 concentrations were measured in cord blood samples collected at birth, and also in venous blood samples collected at ages 7 or 8 years as previously described [11, 12]. Fasting insulin sensitivity was assessed by the homeostatic model assessment index and insulin secretion at 30 min post-oral glucose by the insulinogenic index at age 8 years as previously described [13].

Genotyping

Genotyping of selected tag-SNPs in both populations was undertaken by means of Invader (Third Wave Technologies, Madison, WI, USA) or TaqMan (Applied Biosystems, Warrington, UK). Variation at a previously described 5′ *IGF1* CA repeat was determined after PCR amplification using previously described fluorescently labelled primers [14]. The amplified sequences were subsequently analysed using an Applied Biosystems 3700 capillary sequencer (Applied Biosystems, Foster City, CA, USA). All genotyping data were double-scored to minimise error. All genotypes were in Hardy–Weinberg equilibrium ($p>0.05$).

Wellcome Trust Case Control Consortium data

To determine whether there was any further evidence of an association with type 1 diabetes in an extended *IGF1* region, we used data from a recent genome-wide association study (Wellcome Trust Case–Control Consortium [WTCCC]; www.wtccc.org.uk) [15]. We defined the extended *IGF1* region by moving the boundaries of *IGF1* (chromosome 12; 101,292,143–101,376,791 base pairs in NCBI build 35) out by 21,162 base pairs (25% of the gene) on either side of the gene. This extended region contained 23 WTCCC SNPs, one of which was monomorphic. In HapMap (www.hapmap.org), 20 of 23 WTCCC SNPs had a minor allele frequency (MAF) ≥0.05 in 60 Centre d'Etude du Polymorphisme Humain (CEPH) parents; these 20 WTCCC SNPs had a maximum $r^2 \geq 0.5$ with 26 of 37 additional HapMap SNPs with a MAF ≥0.05 in the extended *IGF1* region. In 2,893 controls, 15 of 23 WTCCC SNPs had a MAF ≥0.05. When the WTCCC case and control data were analysed using a logistic regression model adjusted for 12 broad geographical regions within Great Britain to minimise any confounding due to variation in allele frequencies across the country, there was no evidence of association of these SNPs within the linkage disequilibrium region containing *IGF1* (minimum $p$=0.0366 from 22 WTCCC SNPs tested; ESM Table 4).

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