Supplementary Methods

Study populations

The individuals were recruited into various collections, UK-based individuals from the type 1 diabetes genetics consortium (T1DGC) [1], the genetic resource investigating diabetes (GRID), the Warren cohort [2], the northern Irish GRID [3], the 1958 birth control cohort and the UK blood service [1], individuals from central Europe, Asia-Pacific and the USA from the T1DGC and individuals from Finland from the IDDMGEN and T1DGEN cohorts [4].

We grouped the individuals into three groups, individuals from the UK, individuals from Finland and individuals recruited into the T1DGC study. From each group, we removed individuals related to closer than second degree by pruning variants correlated to r^2 >0.2 and removing the major histocompatility (MHC) region. We then calculated relatedness using the KING 2.0.9 software [5], leaving the total of 26,991 individuals for analysis. After removing variants in linkage disequilibrium (LD) and the MHC region (chromosome 6, positions 25000000-35000000 genome build 36), 29,101 variants were used for relationship inference in the UK group, 28,597 in the T1DGC group and 26,809 in the Finnish group.

Genotyping

All samples were genotyped using the Illumina ImmunoChip[1], a specialised platform which densely genotypes regions of interest for autoimmune diseases.

Imputation

To impute the classical HLA alleles of individuals on our dataset, HiBag [6] R Bioconductor package was used with the classifiers as calculated from the training dataset in the original publication used. Alleles called with a probability of less than 0.5 were treated as missing.

Nine non-HLA variants that we wanted to examine failed genotype quality control (QC) from direct genotyping using the ImmunoChip; either for having a minor allele frequency <0.01, a Hardy-Weinberg equilibrium exact test p value $<5\times10^{-4}$ or ≥5% missing values. In these instances, we imputed the variants using the IMPUTE2[7] software; with the 1000 Genomes Project data as the reference haplotypes. We used this same imputation strategy to impute around index variants for non-HLA regions found to be differentially associated between the <7 and ≥13 groups for fine mapping.

Heterogeneity tests

In order to examine heterogeneity in effect size between the <7 and ≥13 groups, we fitted two multinomial logistic regression models including all individuals from the three AAD groups and controls in both models, assessing the probability of being in each AAD group relative to controls. In the first model, we fitted the multinomial logistic regression with no constraints, adjusting for the top ten largest genetic principal components and the examined variant, where a different effect size for the disease-associated genetic variant was estimated in the <7, 7-13 and ≥13 groups. In the second model, as suggested in [8], we constrained the effect size of the genetic variant in the <7 and ≥13 groups to be equal to each other. If the unconstrained model fitted the data significantly better than the constrained model, this would suggest there are differences in effect sizes for the genetic variant between the two groups. We tested for heterogeneity between the <7 and ≥13 groups using a likelihood ratio test comparing these two models. This analysis was performed using the multinomRob R package [9].

For HLA analyses, we additionally adjusted for other HLA haplotypes/alleles to account for the high levels of LD in the region. When examining the HLA class II haplotype effects other than DR3-DQ2/DR4-DQ8, we examined only individuals

without the DR3-DQ2/DR4-DQ8 haplotype in order to remove any confounding effects of this diplotype. Supplementary Table 1 shows which individuals were included in each analysis, as well as the classical haplotypes/alleles adjusted for, with some rare haplotypes/classical alleles not adjusted for in models due to failure of the logistic regression algorithm to converge when these were included.

The top 10 genetic principal components were adjusted for in each analysis and were generated using ImmunoChip data filtered so that a set of independent variants (r^2<0.2) were kept and the MHC region was excluded entirely (chromosome 6, positions 25000000-35000000 genome build 36), leaving a total of 25,745 variants for which to calculate principal components from. The PLINK software was used to carry out this procedure [10].

The false discovery rate (FDR) applied throughout is the Benjamini-Hochberg FDR procedure [11].

Binomial test examining variants not associated to FDR <0.1

Given there are 3 AAD groups in our analysis, there are 6 possible ways the log-odds ratio estimates could be ordered. If there were to be no differences in genetic effect between AAD groups, we would expect approximately one sixth of the variants tested to be in each of the possible orders. We therefore performed a binomial test comparing the number of times the ordering was the largest effect estimate in the <7s, the intermediate effect estimate in the 7-13s and the smallest effect estimate in the ≥13s to that expected by chance (1/6=0.167).

Fine-mapping

At regions selected for fine mapping due to their heterogeneity in effect sizes between the <7 and ≥13 group, we implemented GUESSFM [12]. We imputed a 0.5 Mb region around each index variant, then to guard against identifying false positive associations

due to imputation, we removed variants from the stochastic search that had a certainty call rate of <75%, a minor allele frequency of <0.005, an absolute Hardy-Weinberg equilibrium z score of >8 (in controls only) and a variant imputation information score of <0.8 or a difference in imputation information score between cases and controls of more than 0.01. We also excluded variants with a concordance rate between genotypes and imputed genotype call of <0.8 and variants where the ratio of the variance compared to the approximate expected variance for a variant with that minor allele frequency (MAF) (≈2×MAF×(1-MAF)) was greater than 0.05.

We adjusted for the top five principal components, set the prior mean number of expected causal variants in the region to be three and saved the top 10,000 most visited models for downstream analysis.

Colocalisation analyses

Using the imputed genotype data as described above, we tested each imputed variant that passed imputation QC for an association with type 1 diabetes diagnosed at <7 years compared to controls using logistic regression. We then took association summary statistics for these variants from an eQTL analysis in whole blood of over 30,000 individuals [13]. Summary statistics from both association studied (disease risk and eQTL) were converted to approximate Bayes Factors (ABFs). Posterior probabilities were then calculated for each of the following scenarios: i) association with disease only, ii) association with eQTL only, iii) association with disease and eQTL but distinct signals or iv) shared disease and eQTL signal in the region [14]. We focus on scenario iv), colocalisation of disease and eQTL signal, which is calculated by multiplying the ABFs for disease and eQTL together for each variant (to get the likelihood of colocalisation) and multiplying this by the prior probability of colocalisation, which we took to be the product of the prior probability of association

of each variant for disease and eQTL, set at 0.001, meaning the prior probability for colocalisation was 0.000001. Dividing this product by the normalising factor, the overall probability of all combinations of each of the four hypotheses and also the probability of no association with disease or eQTL, gives the posterior probability of colocalisation. The coloc R package was used to carry out this analysis.

Supplementary Table 1: Classical HLA alleles/haplotypes examined in analysis.

Haplotype/allele name	Haplotype/allele definition	Individuals included	Conditioned on	Enough individuals to include?
DR3-DQ2	DRB1*03:01-DQB1*02:01	Non-DR3/4 only	A*02:01, A*24:02, B*39:06	Yes
DR4-DQ8	DRB1*04:01-DQB1*03:02 DRB1*04:02-DQB1*03:02 DRB1*04:04-DQB1*03:02 DRB1*04:05-DQB1*03:02 DRB1*04:08-DQB1*03:02 DRB1*04:01-DQB1*03:04 DRB1*04:02-DQB1*03:04 DRB1*04:04-DQB1*03:04 DRB1*04:05-DQB1*03:04 DRB1*04:08-DQB1*03:04 DRB1*04:01-DQB1*02:02 DRB1*04:02-DQB1*02:02 DRB1*04:04-DQB1*02:02 DRB1*04:05-DQB1*02:02 DRB1*04:08-DQB1*02:02	Non-DR3/4 only	A*02:01, A*24:02, B*39:06	Yes
DR3-DQ2/DR4-DQ8	Any combination of rows 1 and 2 above, one on either chromosome.	All individuals	A*02:01, A*24:02, B*39:06	Yes
DRB1*13:03-DQB1*03:01	DRB1*13:03-DQB1*03:01	Non-DR3/4 only		No
DRB1*11:04-DQB1*03:01	DRB1*11:04-DQB1*03:01	Non-DR3/4 only		No
DRB1*15:01-DQB1*06:02	DRB1*15:01-DQB1*06:02	Non-DR3/4 only		Yes
DRB1*07:01-DQB1*03:03	DRB1*07:01-DQB1*03:03	Non-DR3/4 only		Yes
DRB1*14:01-DQB1*05:03	DRB1*14:01-DQB1*05:03	Non-DR3/4 only		No
A*02:01	A*02:01	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, B*39:06	Yes
A*24:02	A*24:02	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, B*39:06	Yes
A*11:01	A*11:01	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, B*39:06	Yes
A*32:01	A*32:01	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, B*39:06	Yes
DPB1*03:01	DPB1*03:01	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, A*02:01, A24*02, B*39:06	Yes
DPB1*04:02	DPB1*04:02	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, A*02:01, A24*02, B*39:06	Yes
B*18:01	B*18:01	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, A*02:01, A24*02	Yes
B*39:06	B*39:06	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, A*02:01, A24*02	Yes
B*44:03	B*44:03	All individuals	DR3-DQ2, DR4-DQ8, DR3-DQ2/DR4-DQ8, A*02:01, A24*02	Yes

Supplementary Table 2: Non-HLA variants examined in analysis.

Variant name	Chr	Position (genome build 37)	Publication(s) where association identified*	Locus name in Figures in this manuscript
rs2476601	1	114377568	Onengut-Gumuscu	*PTPN22*
rs78037977	1	172681031	Fortune	*TNFSF4***
rs6691977	1	200814959	Onengut-Gumuscu	*CAMSAP2*
rs3024505	1	206939904	Onengut-Gumuscu	*IL10*
rs4849135	1	111615079	Onengut-Gumuscu	*ACOXL*
rs13415583	2	100764087	Onengut-Gumuscu	*AFF3*
rs2111485	2	163110536	Onengut-Gumuscu	*IFIH1 (1)*
rs35667974	2	163124637	Onengut-Gumuscu	*IFIH1 (2)*
rs72871627	2	163136942	Onengut-Gumuscu	*IFIH1 (3)*
rs3087243	2	204738919	Onengut-Gumuscu	*CTLA4*
rs113010081	3	46457412	Onengut-Gumuscu	*CCR5*
rs6819058	4	123114622	Onengut-Gumuscu/ Burren	*IL2/IL21 (1)*
rs67797421	4	123116177	Onengut-Gumuscu/ Burren	*IL2/IL21 (2)*
rs2611215	4	166574267	Onengut-Gumuscu	*CPE*
rs11954020	5	35883251	Onengut-Gumuscu	*IL7R*
rs72975913	6	128293932	Inshaw	*PTPRK/THEMIS*
rs72928038	6	90976768	Onengut-Gumuscu	*BACH2*
rs1538171	6	126752884	Onengut-Gumuscu	*CENPW*
rs62447205	7	50465830	Onengut-Gumuscu	*IKZF1*
rs10277986	7	51028987	Onengut-Gumuscu	*COBL*
rs6476839	9	4290823	Onengut-Gumuscu	*GLIS3*
rs61839660	10	6094697	Onengut-Gumuscu, Wallace	*IL2RA (1)*
rs11594656	10	6122009	Onengut-Gumuscu, Wallace	*IL2RA (2)*
rs6602437	10	6130077	Onengut-Gumuscu, Wallace	*IL2RA (3)*
rs41295121	10	6129643	Onengut-Gumuscu, Wallace	*IL2RA (4)*
rs12416116	10	90035654	Onengut-Gumuscu	*PTEN*
rs689	11	2182224	Onengut-Gumuscu	*INS (1)*
rs72853903	11	2198665	Onengut-Gumuscu	*INS (2)*
rs917911	12	9905851	Onengut-Gumuscu	*CD69*
rs705705	12	56435504	Onengut-Gumuscu	*IKZF4*
rs653178	12	112007756	Onengut-Gumuscu	*SH2B3*
rs9585056	13	100081766	Onengut-Gumuscu	*GPR183*
rs1456988	14	98488007	Onengut-Gumuscu	*LINC01550*
rs56994090	14	101306447	Onengut-Gumuscu	*MEG3*
rs72727394	15	38847022	Onengut-Gumuscu	*RASGRP1*
rs34593439	15	79234957	Onengut-Gumuscu	*CTSH*
rs151234	16	28505660	Onengut-Gumuscu	*IL27*
rs12927355	16	11194771	Onengut-Gumuscu	*DEXI (1)*
rs193778	16	11351211	Onengut-Gumuscu	*DEXI (2)*
rs8056814	16	75252327	Onengut-Gumuscu	*CTRB1***
rs12453507	17	38053207	Onengut-Gumuscu	*IKZF3*
rs757411	17	38775150	Onengut-Gumuscu	*CCR7*

rs1052553	17	44073889	Onengut-Gumuscu	*MAPT*
rs1893217	18	12809340	Onengut-Gumuscu	*PTPN2 (1)*
rs12971201	18	12830538	Onengut-Gumuscu	*PTPN2 (2)*
rs1615504	18	67526644	Onengut-Gumuscu	*CD226*
rs34536443	19	10463118	Onengut-Gumuscu	*TYK2 (1)*
rs12720356	19	10469975	Onengut-Gumuscu	*TYK2 (2)*
rs402072	19	47219122	Onengut-Gumuscu	*PRKD2*
rs516246	19	49206172	Onengut-Gumuscu	*FUT2*
rs6043409	20	1616206	Onengut-Gumuscu	*SIRPG*
rs11203202	21	43825357	Onengut-Gumuscu	*UBASH3A*
rs6518350	21	45621817	Onengut-Gumuscu	*ICOSLG*
rs4820830	22	30531091	Onengut-Gumuscu	*HORMAD2*
rs229533	22	37587111	Onengut-Gumuscu	*C1QTNF6*

*Most recent publication listed. Onengut-Gumuscu [1], Fortune [15], Burren [16], Inshaw [3], Wallace [12].
**Candidate gene different from previous reports based on publically available gene expression (https://dice-database.org/) and promoter-capture Hi-C data (https://www.chicp.org).

Supplementary Table 3: Non-HLA region variants with evidence of heterogeneity in effect size between the <7 and ≥13 groups: Promoter Capture Hi-C (PCHi-C) candidate genes.

Locus name/ karyotype band	Tag/index variant	Candidate causal genes	PCHi-C prioritised protein coding genes*	PCHi-C prioritised non-protein coding transcripts
IKZF3 / 17q21	rs12453507	*IKZF3* *ORMDL3* *GSDMB*	*ORMDL3* *GSDMB* *ZPBP2*	-
CTSH / 15q25.1	rs34593439	*CTSH*	*CTSH* *BCL2A1*	-
GLIS3 / 9p24.2	rs6476839	*GLIS3*	*GLIS3***	*GLIS3-AS1***
IL2RA (3) / 10p15.1	rs6602437	*IL2RA*	*RBM17* *IL2RA* *GDI2* *PRKCQ* *ANKRD16* *FAM208B* *FBX018*	*RP11-536K7.3* *PRKCQ-AS1*
IL10 / 1q32.1	rs3024505	*IL10* *FAIM3*	*IL10* *FCAMR* *IL20* *FAIM3* *PIGR* *CD55* *IL24* *IL19*	-
PTPRK/THEMIS / 6q22.33	rs72975913	*PTPRK* *THEMIS*	*PTPRK* *THEMIS*	
SIRPG / 20p13	rs6043409	*SIRPG* *SIRPG-AS1*	-	

*Inshaw et. al [17]

** Miguel-Escalada et al. [18]

Supplementary Table 4: Details of non-HLA variants with evidence of heterogeneity in effect size between the <7 and ≥13 groups.

Locus name/ karyotype band	Tag/index variant	Candidate causal genes	Tissues expressed*	BLUEPRINT [19] / DICE** database immune cell types expressed***	Function
IKZF3 / 17q21	rs12453507	*IKZF3* *ORMDL3* *GSDMB*	*IKZF3*: Small intestine, spleen *ORMDL3*: Ubiquitous *GSDMB*: Ubiquitous	*IKZF3*: thymocytes, B, T and NK cells *ORMDL3*: thymocytes, T, B, and NK cells, eosinophils *GSDMB*: thymocytes, T, B and NK cells, eosinophils	*IKZF3 (Ailos)*: A transcriptional regulator and a member of the Ikaros gene family [20]. IKZF3 regulates B cell activation thresholds and differentiation [21] and is required for generation of high affinity plasma cells [22]. In T cells, IKZF3 acts as a transcriptional repressor to promote Th17 differentiation [23] and can modulate the activity of FoxP3 [24]. *ORMDL3*: regulates sphingolipid biosynthesis and expression is increased by inflammatory stimuli [25]. ORMDL3 is a negative regulator of store operated calcium entry [26] and modulates lymphocyte activation and cytokine production [27]. *GSDMB*: GSDMB can be cleaved by apoptotic caspases [28] and the active form of GSDMB induces pyroptotic cell death in epithelial cells [29].
CTSH / 15q25.1	rs34593439	*CTSH*	Ubiquitous	Macrophages, DCs, T and B cells, monocytes	CTSH has amino- and endopeptidase activity [30] and has been reported to process pro-surfactant protein B [31] and pro-granzyme B [32] into their active mature forms. Proteolytic cleavage of TLR3 by CTSH alters the stability, localization and/or the regulation of TLR3 activity [33, 34]. CTSH-deficient mice have reduced TLR3 expression and type 1 IFN release in response to poly(I:C) stimulation [35] and T1D associated SNPs colocalise with a monocyte eQTL signal [36].

GLIS3 / 9p24.2	rs6476839	*GLIS3*	Pancreas, thyroid gland, kidney, ovary	-	GLIS3 is a transcriptional regulator [37], required for insulin expression [38], function and transcriptional program of mature beta cells [39]. A key molecule regulating the response to unfolded protein stress and protection of beta cells from apoptosis [40, 39].
IL2RA (3) / 10p15.1	rs6602437	*IL2RA*	Bone marrow, Lymph nodes, spleen	Thymocytes, T, B, NK	*IL2RA* encodes CD25 the alpha chain of the trimeric high affinity IL-2 receptor. IL-2 signaling controls T cell growth and differentiation, NK cell and activated B cell proliferation and is essential for the survival and function of regulatory T cells (Tregs) [41, 42]
IL10 / 1q32.1	rs3024505	*IL10* *FAIM3*	*IL10:* Spleen *FAIM3:* Spleen, small intestine	*IL10*: Monocytes, activated T, Treg, macrophages and B cells. *FAIM3*: T and B cells	*IL10*: Interleukin 10 is a key cytokine that modulates immune and non-immune cell function [43]. IL10 can be produced by specialized regulatory T [44] and B cell populations [45]. Primary mechanisms include downregulation of MHC class II and cytokine responses of antigen-presenting cells [46] and induction of anergy in T cells [47]. IL10 acts as a potent growth and differentiation factor for B cells [48]. *FAIM3:* Encodes FCmR a receptor for natural IgM [49] and involved in B cell homeostasis [50], control of IL10 production [51] and supports early B cell activation events and plasma cell development [52].
PTPRK/THEMIS / 6q22.33	rs72975913	*PTPRK* *THEMIS*	*PTPRK*: Ubiquitous *THEMIS*: Spleen, small intestine, thymus	*PTPRK*: Thymocytes, naïve T cells, B cells *THEMIS*: T cells	*PTPRK*: Protein tyrosine phosphatase receptor type kappa may be involved in thymic selection of single positive CD4 T cells [53]. *THEMIS*: Thymocyte-expressed molecule involved in selection regulates phosphatase activity (Shp1) in developing thymocytes to enable positive selection [54, 55].
SIRPG /	rs6043409	*SIRPG*	*SIRPG*: Spleen,	*SIRPG*: T cell	*SIRPG*: Signaling receptor protein gamma binds to

20p13		*SIRPG-AS1*	small intestine, testis	*SIRPG-AS1:* T cell	CD47[56], a ubiquitously expressed integrin associated protein. Interaction of SIRPG with CD47 controls transendothelial migration of T cells [57]. The T1D associated variant rs2281808 reduces expression of SIRPG in T cells and modulates the activation threshold and differentiation state of CD8 T cells [58].

*Human protein atlas: https://www.proteinatlas.org
** DICE database: https://dice-database.org/
*** T=T cells, B= B cells, NK = Natural Killer cells, DC= Dendritic cells.

INCLUDE:
(i) candidate genes nominated in Onengut-Gumuscu et al [1] and Inshaw et al [17].
(ii) genes prioritised by promoter capture Hi-C (PCHi-C) in immune cells [17, 59], or gene promoter interactions with credible SNPs in islet PCHi-C [18].

FILTER ON:
(i) expression in immune cells (https://dice-database.org/) or islets [60]
and/or
(ii) eQTL in whole blood [13] passing a Bonferroni corrected p value threshold ($p < 2.78 \times 10^{-13}$).

Supplementary Table 5: Most likely variants causally associated with T1D at the *IKZF3* locus from GUESSFM fine mapping analysis.

Variant	Position (gr37)	Log-odds ratio	Reference allele	Effect allele	Variant posterior probability	Group posterior probability
rs1008723	38066267	-0.1729	T	G	3.55e-02	0.9557
rs1011082	38068514	-0.1664	T	C	1.02e-02	0.9557
rs1031458	38072173	-0.1661	G	T	9.82e-03	0.9557
rs1031460	38072247	-0.1691	T	G	1.75e-02	0.9557
rs12453507	38053207	-0.1688	G	C	1.65e-02	0.9557
rs12603332	38082807	-0.1627	T	C	5.51e-03	0.9557
rs12950209	38049102	-0.1774	C	T	8.33e-02	0.9557
rs12950743	38049233	-0.179	C	T	1.10e-01	0.9557
rs141758348	38069364	-0.1476	TCAAAA	T	6.60e-04	0.9557
rs143385463	38071855	-0.1629	ATTT	A	5.78e-03	0.9557
rs150597688	38071086	-0.1653	C	CTT	7.76e-03	0.9557
rs2290400	38066240	-0.1767	C	T	7.43e-02	0.9557
rs2305479	38062217	-0.1731	T	C	3.74e-02	0.9557
rs2872516	38072727	-0.1422	C	T	1.75e-04	0.9557
rs35196450	38062942	-0.1566	AC	A	1.77e-03	0.9557
rs36000226	38063929	-0.1665	C	T	7.02e-03	0.9557
rs36084703	38063980	-0.1665	C	CA	7.10e-03	0.9557
rs4065275	38080865	-0.1662	A	G	1.06e-02	0.9557
rs62067034	38063738	-0.1668	T	C	7.44e-03	0.9557
rs6503524	38069809	-0.1673	C	T	1.24e-02	0.9557
rs68122720	38050092	-0.157	A	AAG	3.62e-03	0.9557
rs7216389	38069949	-0.1678	C	T	1.35e-02	0.9557
rs7216558	38070071	-0.1709	C	T	2.44e-02	0.9557
rs7219923	38074518	-0.1696	C	T	1.94e-02	0.9557
rs7224129	38075426	-0.1694	G	A	1.88e-02	0.9557
rs7359623	38049589	-0.1644	T	C	7.71e-03	0.9557
rs8065777	38072402	-0.1667	C	T	1.10e-02	0.9557
rs8067378	38051348	-0.1818	G	A	1.93e-01	0.9557
rs8074437	38076137	-0.1677	T	G	1.28e-02	0.9557
rs869402	38068043	-0.1677	T	C	1.30e-02	0.9557
rs883770	38063381	-0.1723	T	C	3.20e-02	0.9557
rs921649	38069274	-0.1669	C	T	1.16e-02	0.9557
rs921650	38069076	-0.1686	G	A	1.53e-02	0.9557
rs9906951	38048244	-0.1794	C	T	1.19e-01	0.9557

Supplementary Table 6: Most likely variants causally associated with T1D at the *CTSH* locus from GUESSFM fine mapping analysis.

Variant	Position (gr37)	Log-odds ratio	Reference allele	Effect allele	Variant posterior probability	Group posterior probability
rs12148472	79231478	-0.2763	T	C	1.76e-02	1
rs12592898	79229199	-0.2736	G	A	9.89e-03	1
rs2289702	79237293	-0.3513	C	T	4.47e-01	1
rs34593439	79234957	-0.3342	G	A	2.42e-01	1
rs34843303	79234470	-0.3283	T	C	2.80e-01	1
rs60254670	79229959	-0.2648	TGGCCAGAATG	T	4.10e-03	1

Supplementary Table 7: Most likely variants causally associated with T1D at the *GLIS3* locus from GUESSFM fine mapping analysis.

Variant	Position (gr37)	Log-odds ratio	Reference allele	Effect allele	Variant posterior probability	Group posterior probability
rs10116772	4290541	-0.129	A	C	1.43e-02	0.9751
rs10758593	4292083	-0.1338	A	G	2.96e-02	0.9751
rs10758594	4295583	-0.1421	A	G	1.26e-01	0.9751
rs10814915	4290544	-0.1358	T	C	4.60e-02	0.9751
rs10814916	4293150	-0.1371	C	A	5.94e-02	0.9751
rs10814917	4296430	-0.1399	A	G	9.18e-02	0.9751
rs10974438	4291928	-0.1439	C	A	8.95e-02	0.9751
rs34706136	4294707	-0.1522	TG	T	2.99e-01	0.9751
rs35338539	4298589	-0.1268	A	G	5.74e-03	0.9751
rs4339696	4295880	-0.1266	G	T	1.17e-02	0.9751
rs6476839	4290823	-0.1352	T	A	3.72e-02	0.9751
rs6476842	4291268	-0.1396	C	T	8.45e-02	0.9751
rs7020673	4291747	-0.1389	G	C	8.03e-02	0.9751

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