

ESM Table 10 In silico replication in existing GWAS meta-analysis databases for eight associations with nominal replication in stage 3 data.

Trait	rs (dbSNP 129)	Chr-Position (build 36)	Gene	EAF (%)	Proxy (r^2)	N	Effect/P	Consistent direction	Reason for missing results
Fasting serum HDL-cholesterol	None	17-39281652	<i>CD300LG</i>	3.5					No proxy
Type 2 diabetes	rs7607980	2-165259447	<i>COBLL1</i>	12.5		22570	0.93 (0.87-0.99), $P=0.016$	Y	
Type 2 diabetes	rs2296172	1-39608404	<i>MACF1</i>	23.4		22570	1.06 (1.01-1.11), $P=0.016$	Y	
Type 2 diabetes	rs60980157	9-138355236	<i>GPSM1</i>	24.6					No proxy
Fasting serum triacylglycerol	rs41273264	6-31708085	<i>PRRC2A</i>	4.2					No proxy
Obesity	rs11553746	2-262203	<i>ACP1</i>	37.8					No data for this trait
BMI	rs11553746	2-262203	<i>ACP1</i>	37.8	rs3791221 (1)	123842	$P=0.093$	Y	

Due to a minor overlap in samples between stage 3 and the GWAS meta-analysis data these data were not meta-analysed with stage 2 and stage 3 data and consequently they represent semi-independent replication. Data for type 2 diabetes were contributed by the DIAGRAM Consortium [27], data on BMI by GIANT Consortium [28] (http://www.broadinstitute.org/collaboration/giant/index.php/GIANT_consortium) and data on glycaemic traits were contributed by MAGIC investigators [29] and were downloaded from www.magicinvestigators.org. For two SNPs the lead SNPs were in the GWAS meta-analyses data while *in silico* replication for two SNPs was based on proxies. Here LD was estimated from data from the 1000 Genomes Project. Three SNPs had no good proxy ($r^2 > 0.4$). For one trait no GWAS data could be investigated.