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Common Variation in the LMNA Gene (Encoding Lamin A/C) and Type 2 Diabetes: Association Analyses in 9,518 Subjects.

[Owen KR](#), [Groves CJ](#), [Hanson RL](#), [Knowler WC](#), [Shuldiner AR](#), [Elbein SC](#), [Mitchell BD](#), [Froquel P](#), [Ng MC](#), [Chan JC](#), [Jia W](#), [Deloukas P](#), [Hitman GA](#), [Walker M](#), [Frayling TM](#), [Hattersley AT](#), [Zeggini E](#), [McCarthy MI](#).

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Mutations in the LMNA gene (encoding lamin A/C) underlie familial partial lipodystrophy, a syndrome of monogenic insulin resistance and diabetes. LMNA maps to the well-replicated diabetes-linkage region on chromosome 1q, and there are reported associations between LMNA single nucleotide polymorphisms (SNPs) (particularly rs4641; H566H) and metabolic syndrome components. We examined the relationship between LMNA variation and type 2 diabetes (using six tag SNPs capturing >90% of common variation) in several large datasets. Analysis of 2,490 U.K. diabetic case and 2,556 control subjects revealed no significant associations at either genotype or haplotype level: the minor allele at rs4641 was no more frequent in case subjects (allelic odds ratio [OR] 1.07 [95% CI 0.98-1.17], $P = 0.15$). In 390 U.K. trios, family-based association analyses revealed nominally significant overtransmission of the major allele at rs12063564 ($P = 0.01$), which was not corroborated in other samples. Finally, genotypes for 2,817 additional subjects from the International 1q Consortium revealed no consistent case-control or family-based associations with LMNA variants. Across all our data, the OR for the rs4641 minor allele approached but did not attain significance (1.07 [0.99-1.15], $P = 0.08$). Our data do not therefore support a major effect of LMNA variation on diabetes risk. However, in a meta-analysis including other available data, there is evidence that rs4641 has a modest effect on diabetes susceptibility (1.10 [1.04-1.16], $P = 0.001$).

PMID: 17327460 [PubMed - in process]

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