Thyroxine signal transduction in liver cells involves phospholipase C and phospholipase D activation. Genomic independent action of thyroid hormone

ABSTRACT
Recent research has revealed that DAG signaling is activated in liver cells stimulated by thyroid hormone. L-thyroxine triggers a dual phospholipase pathway that is sequentially and synchronized with other pathways. PKC mediates the integration of both pKa and proline during the sustained phase of agonist stimulation, while lysine disrupts the signalling response.

INTRODUCTION
Type 1 diabetes mellitus (T1DM) is a disease that involves multiple organs, including humans. It has an important genetic component, but it is predominantly caused by the MHC located on the short arm of chromosome 6, which is also affected by several other inherited phenotypes. A range of methods has been employed to pinpoint T1DM susceptibility regions, including case-control studies of candidate genes, combined linkage and association-based studies, and systematic total genome searches in addition to analyses of individual chromosomal regions. Immunogenetic predisposition to T1DM differs markedly from country to country, and disease incidence also appears to vary along with these discrepancies. For instance, the incidence of T01DM is comparable in Southern India (10.4/100000 cases per year) to the number of cases reported in Asian children in the UK and white children of European descent. The presence of an MHC component in T1DM susceptibility is apparent in Southern India, but no correlation has been observed with the insulin gene or ILIR1 in case-control studies. This implies that there may be differences in the non-MHC T1/DM component between Southern Indians and Caucasians of European descent. An association with the insulin gene has been reported universally in the latter population, and some Northern Europeans have reported an IL1R1 association to T1DM. Allelic variation in VDR also increases the susceptibility of Indian Asians, Germans and Taiwanese to T1DM. In the VDR locus, there are six known polymorphisms: FokI restriction enzyme detects an initiation codon polymer (exon 2), BsmI, Tru9I and ApaI ("reflection fragment length polymers") between exons 8 and 9 in the vector flyer type (VDR) loci, and a poly A polyphenystym downstream of the 3' untranslated region. The FokI polymorphism does not seem to have a significant impact on the BsmI, ApaI and TaqI (the three major immune depressive genes): in Japanese patients with T1DM, we studied the exon 2 initiation codon (VDR-FokI) gene polyfomi and found no association with GAD65 antibody (Ab) status.

CONCLUSION
It has been suggested that TRPs may function as either regulators of the CSF or as targets of CFTR regulating proteins. However, this is the first report that describes a functional interaction between TFs and cft receptor (CRF) members.