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USC Researchers Identify Gene that Regulates Glucose Levels and Increases Risk for Diabetes

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San Francisco, Calif., June 7, 2008— Researchers at the University of Southern California (USC) have helped identify a genetic variant that regulates glucose levels and also increases the risk of type 2 diabetes. The results of the study were presented as an oral presentation on Saturday, June 7, at the American Diabetes Association 68th Scientific Sessions held in San Francisco.

“We tested for an association between genetic variants across the human genome and fasting glucose and insulin,” says Richard M. Watanabe, Ph.D., associate professor of preventive medicine and physiology & biophysics at the Keck School of Medicine of USC and lead author of the paper. “We found a novel association between fasting glucose and the melatonin receptor 1B (MTNR1B). It’s novel because this is the first time a genetic variant has been associated with both glucose and increased risk of diabetes.”

The study examined genetic information from 6,543 people participating in three independent genome-wide association studies for fasting glucose and insulin. The studies included Finland-United States Investigation of Non-insulin-dependent Diabetes Mellitus (FUSION) study and the SardiNIA study of aging and the Diabetes Genetics Initiative.

“The MTNR1B finding is interesting because melatonin is involved with regulating circadian rhythms, like sleep cycles, and people with sleep disorders, like sleep apnea, tend to develop obesity and insulin resistance, which are risk factors for type 2 diabetes,” continues Watanabe. “More studies will be needed to understand how MTNR1B is involved in regulating glucose and associated risk for diabetes.”

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Richard M. Watanabe, Wei-Min Chen, Michael R. Erdos, Richa Saxena, Anne U. Jackson, Valerie Lyssenko, Manuela Uda, Thomas A. Buchanan, David Schlessinger, Leif Groop, Francis S. Collins, David Altshuler, Goncalo Abecasis, Micheal Boehnke, Angelo Scuteri, “Novel Genetic Loci for Fasting Glucose and Insulin Identified by Genome-wide Association in Caucasians.” Abstract # 137-OR.

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