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THE DISTILLERY

This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Endocrine/metabolic disease				
Diabetes	Calcium calmodulin-dependent protein kinase IIγ (CAMK2G)	Mouse studies suggest inhibiting CAMK2G could help improve hyperglycemia and insulin sensitivity in patients with type 2 diabetes. CAMK2G stimulates glucagon-induced, calcium-mediated hepatic glucose production. In three mouse models of obesity and type 2 diabetes, liver-specific inactivation of *Camk2g* improved blood glucose response and decreased hepatic glucose production and blood insulin levels compared with no *Camk2g* inactivation. In mouse livers, *Camk2g* inactivation enhanced insulin signaling through MAP kinase–activated protein kinase 2 (MAPKAPK2; MK2)-mediated activation of the eukaryotic translation initiator factor 2α kinase 3 (EIF2AK3; PERK) branch of the unfolded protein response. Next steps include developing inhibitors of MK2 and testing them in animal models of diabetes. The authors have cofounded Tabomedex Biosciences LLC to develop MK2 inhibitors licensed from Columbia University. *SciBX* 7(1); doi:10.1038/scibx.2014.16 Published online Jan. 9, 2014	Patent application filed by Columbia University; additional application in preparation covering unpublished MK2 inhibitors; licensed by Tabomedex Biosciences	Ozcan, L. *et al. Cell Metab.*; published online Nov. 21, 2013; doi:10.1016/j.cmet.2013.10.011 **Contact:** Ira Tabas, Columbia University, New York, N.Y. e-mail: iat1@columbia.edu **Contact:** Lale Ozcan, same affiliation as above e-mail: lo2192@columbia.edu