This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cardiovascular disease				
Left ventricular hypertrophy (LVH)	G protein–coupled receptor kinase 5 (GRK5); guanine nucleotide binding protein, q polypeptide (GNAQ; G_q); histone deacetylase 5 (HDAC5); myocyte enhancing factor 2 (Mef2; D-Mef2)	Studies in mice and in cell culture suggest that targeting GRK5 could help treat LVH associated with heart failure. In mouse models of pressure-overload cardiac stress, GRK5 overexpression led to cardiac hypertrophy and increased the incidence of heart failure compared with what was seen in wild-type mice or mice that overexpressed a GRK5 variant that did not enter the nucleus. *In vitro*, cardiac stress–induced G protein–coupled receptor signaling led to upregulation of GRK5 and its subsequent translocation to the nucleus, where it induced HDAC5-mediated expression of hypertrophy genes. Ongoing studies will assess the specificity of GRK5's activation of HDAC5 and the effects of targeting GRK5 with a GRK5-specific microRNA. Many companies market or are developing compounds to treat or prevent hypertrophy via treatment of hypertension and/or ischemia, two conditions that can lead to LVH and subsequent heart failure.	Not patented; licensing status undisclosed	Martini, J. *et al. Proc Natl. Acad. Sci. USA*; published online Aug. 4, 2008; doi:10.1073/pnas.0803153105 **Contact:** Walter J. Koch, Thomas Jefferson University, Philadelphia, Pa. e-mail: walter.koch@jefferson.edu