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Articles of Significant Interest in This Issue

Estrogen-Related Receptor Alpha Regulates Angiogenesis

The function of the estrogen-related receptors (ERRs) in the vasculature is largely unknown. Likhite et al. (e00411-18) report the discovery that vascular endothelial cells exclusively express the ERRα subtype and that endothelial ERRα deletion facilitates angiogenesis, particularly in response to angiogenic growth factors. They show that ERRα acts predominantly as a transcriptional repressor of gene expression in endothelial cells, targeting genes linked with processes involved in blood vessel morphogenesis, including angiogenesis, migration, and cell adhesion. Finally, they show that vascular endothelial growth factor A regulates ERRα expression and that it is recruited to angiogenic gene promoters, consistent with a negative feedback role for ERRα in growth factor-mediated angiogenesis in endothelial cells.

Rapid Recapitulation of Nonalcoholic Steatohepatitis upon Loss of Host Cell Factor 1 in Mouse Hepatocytes

Nonalcoholic fatty liver disease (NAFLD) encompasses a range of adverse liver conditions caused by excessive fat accumulation (steatosis) within hepatocytes. Despite the increasing prevalence of NAFLD in developed countries, the mechanisms underlying NAFLD progression remain incompletely understood. Host cell factor 1 (HCF-1), encoded by the X-linked gene *Hcfc1*, is an epigenetic coregulator important for cell proliferation and development. By inactivating the *Hcfc1* gene in mouse hepatocytes, Minocha et al. (e00405-18) recapitulated rapid NAFLD progression, including the development of steatosis, mitochondrial defects, inflammation, hepatocyte ballooning, fibrosis, progenitor cell activation, and metabolic dysfunction. Hepatocyte HCF-1 loss also led to the loss of PGC1α protein and to the broad dysregulation of genes involved in mitochondrial function. These findings outline an essential role for HCF-1 in hepatocyte function and provide a new model for the study of NAFLD pathogenesis.

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