Elevated circulating cholesterol levels is a major risk factor for cardiovascular diseases (CVD), and therefore understanding pathways that affect cholesterol metabolism are important for potential treatment of CVD. The major route for cholesterol excretion is through its catabolism to bile acids. Specific bile acids are also potent signaling molecules that modulate metabolic pathways affecting lipid, glucose and bile acid homeostasis. Bile acids are synthesized from cholesterol in the liver, and the key enzymes involved in bile acid synthesis (Cyp7a1, Cyp8b1) are regulated transcriptionally by the nuclear receptor FXR. We have identified an FXR-regulated pathway upstream of a transcriptional repressor that controls multiple bile acid metabolism genes. We identify MafG as an FXR target gene and show that hepatic MAFG overexpression represses genes of the bile acid synthetic pathway, and modifies the biliary bile acid composition. In contrast, MafG loss-of-function studies cause de-repression of the bile acid genes with concordant changes in biliary bile acid levels. Finally, we identify functional MafG response elements in bile acid metabolism genes using ChIP-Seq analysis. Our studies identify a molecular mechanism for the complex feedback regulation of bile acid synthesis controlled by FXR. The identification of this pathway will likely have important implications in metabolic diseases.