The farnesoid X receptor (FXR) is a nuclear receptor expressed highly in the liver and intestine. FXR is a transcriptional regulator activated endogenously by bile acids and regulates the expression of genes involved in cholesterol and bile acid homeostasis (via CYP7A1), hepatic gluconeogenesis/lipogenesis, and inflammation. The purpose of this study was to evaluate therapeutic targets that utilize FXR activation to treat nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatosis (NASH) using literature review of current preclinical studies in mice and clinical research. NASH is characterized by hepatic steatosis, cellular damage, inflammation and progressive development of liver fibrosis and the potential to progress to hepatocellular carcinoma. However, there is currently no FDA approved treatment option for NASH. While there are multiple factors contributing to NASH pathophysiology, bile acid regulation is proposed to have a major role in NASH onset. FXR agonists are currently in phase two or phase three clinical trials and these ligands have demonstrated improvement on NASH symptoms. The forerunner of these candidates is obeticholic acid (OCA) where the treatment group observed a significant decrease in NAFLD activity score compared to placebo group. A concern with OCA is pruritis and unfavorable alterations in cholesterol levels. Other agonists like cilofexor, tropifexor, nidufexor, and EDP-305 have displayed favorable effects on NAFLD/NASH with similar or without the same side effects as OCA. FXR antagonism has also been studied, but antagonists like ursodeoxycholic acid (UDCA) and glycine-β-muricholic acid (Gly-MCA) are in pre-clinical stages. Evaluating and organizing the literature available on FXR ligands and their potential as therapeutic candidates for NASH provides better clinical understanding for a disease that has no FDA approved treatment currently. Future research in cholesterol dysregulation, pruritis effects, and other potential risks from FXR activation could be further studied as unfavorable risk to benefit ratio can prevent or delay drug approval. Funded by Grant Award Number NIH ES020721 and the ASPET Fellow Program.