Figure 1 Schematic representation of the immunometabolic crosstalk in the gut-liver axis linking inflammatory bowel disease and metabolic dysfunction-associated steatotic liver disease. The diagram illustrates three critical mechanisms: (1) Dysbiosis: In the inflammatory bowel disease gut, dysbiosis leads to the production of pathogenic metabolites (*e.g.*, trimethylamine N-oxide, endogenous ethanol) and a functional downregulation of short-chain fatty acid receptors (G protein-coupled receptors 41 and 43), creating a pro-inflammatory environment; (2) Pathogenic Th17: An interleukin-23-rich milieu promotes the differentiation of pathogenic Th17 cells. This axis can be therapeutically targeted by the interleukin-23 inhibitor Guselkumab; and (3) Sphingosine-1-phosphate modulation: Sphingosine-1-phosphate receptor modulators, such as Etrasimod, prevent lymphocyte egress from lymphoid tissues, thereby reducing immune cell infiltration into the liver. Center (gut-liver crosstalk): These metabolites and immune cells translocate *via* the portal vein to the liver, driving hepatic inflammation and steatosis. Parts of this figure were adapted from Servier Medical Art (https://smart.servier.com/), licensed under a Creative Commons Attribution 4.0 International (CC BY 4.0) (Supplementary material). GPR: G protein-coupled receptors; S1P: Sphingosine-1-phosphate; TMAO: Trimethylamine N-oxide.