

Metabolic Associated Fatty Liver Disease and Gut Microbiota a Close Relationship: The Gut Liver Axis

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The gut-liver axis (GLAx) establish an effective communication between the liver and the intestine [1]. The liver obtains much of its blood from the gastrointestinal tract through the portal axis where the portal axis where the portal vein as a fundamental link ensures that bacteria reach the liver [2]. This further confirms the crucial role of the gut-liver axis in the pathogenesis of MAFLD.

Specify improvements in GLAx, possibility liver protection against intestinal pathogens, which may be mediated by probiotics. Therefore, the role of the GLAx as a determinant in the pathophysiology of GLAx in MAFLD is evidence, helping in the search for strategies for prevention and treatment [3].

It is important to know some elements of GLAx such as: Intestinal permeability determines what can be transported from gut to liver and influences NAFLD progression. It is dependent on the intestinal barrier consisting of mucus layer, intestinal epithelium, mucosal immune system, and the gut vascular barrier (GVB). The central roles of the intestinal barrier are enterocytes and GVB in charge of entry into the portal vein and access to the liver [3].

The portal vein a systemic circulation connects the intestine directly with the liver, which in physiological conditions allows the products of the intestinal microbiota to reach the liver. The Bile Acids (BAs) circulation enables bile acids, a molecule synthesized in the liver, to reach the intestine during the interdigestive period. On the other hand, the sterilization of bile acids in the intestine is possible through salt hydrolase (BSH), which is active on a variety of bacterial genera [3]. The microbiome is understood as the relationship of the intestinal microbiota with its genomic element in the environment of the host and probiotics [9].

Inflammation constitutes an important pathophysiological element, which we are going to name Metainflammation. This peculiar characteristic in MAFLD as chronic infection. Its genesis lies in the bacterial overgrowth in the intestine (SIBO) that it is consider sterile and non-infectious. An essential role in the progression of the disease that reaches a spectrum that goes from simple steatosis to the most advance stages [4,5]. Therefore, due to influence of the microbiota on MAFLD, the scientific community has carried out numerous studies with the aim That these intervention make it possible to have a better approach in terms of managements and treatments [6,7].

Analyzing the qualitative and quantitative changes in intestinal bacteria is of great relevance; this is what we call dysbiosis. Consequently, alterations in intestinal permeability are evidence, which allows the presence of endotoxins in the portal tract, which activates inflammatory cytokines [10].

Now it is important to keep in mind that in personalized medicine the intestinal microbiota plays an essential role [11]. Making a correct stratification base on the microbiome when selecting different therapeutic methods in patients with MAFLD is of great importance

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considering that it depends on the microbial characteristics of the metagenome and the metabolic profile of the patients. Currently there are not many studies that support this premise [12].

Finally, it would be of great importance to incorporate the measurement of intestinal permeability *in vivo* into clinical interventions for patients with MAFLD, bringing with it the determination of patients with greater benefits from this intervention [8].

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91