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Research progress on the effect of intestinal microbiota on liver injury caused by particulate matter exposure and the intervention mechanism of lactic acid bacteria

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ABSTRACT: Food processing activities emit a number of air pollutants, including fine particulate matter (PM), which has emerged as a significant environmental risk factor for human health. In recent years, increased emphasis has been paid to the detrimental health impacts of air pollution. PM, being a primary component of air pollution, has historically been seen as predominantly damaging to the respiratory system. Still, with the deepening of the research, PM enters the intestinal tract through the internal circulation, causing intestinal damage and gut microbiome (GM) disorders, which affects the function of GM, and through the portal or intestinal barriers, it brings a harmful effect on the liver of the human body. Therefore, this paper summarizes the research progress in recent years through the intestinal flora, the mechanism of PM-induced liver injury and the intervention mechanism of lactobacilli (LAB), and explores the effects of PM on the gut-liver axis, and the intervention role of lactobacilli (LAB) through those mechanisms, to provide scientific new ideas for the study of LAB intervention in PM-induced GM disorders leading to liver injury.

Keywords: air pollutants, GM, PM, liver damage, gut-liver axis,LAB

1. Introduction

Food manufacturing and processing operations are increasingly recognized as non-negligible sources of ambient air pollution. Processes such as baking, frying, drying, smoking, and industrial food packing release considerable volumes of particulate matter (PM), volatile organic compounds (VOCs), and polycyclic aromatic hydrocarbons (PAHs) into the environment (Abdullahi, Delgado-Saborit, & Harrison, 2013; Arrighini, Guariso, Volta, & Zecchi, 2023). For example, thermal food processing generates ultrafine particles and lipid-derived fumes, which contribute not only to occupational exposure in food factories but also to ambient air pollution in urban areas (Singh & Agarwal, 2022). Additionally, biomass burning in agricultural food production (such as crop straw burning and animal waste incineration) significantly aggravates PM emissions and deteriorates air quality (Fig 1) (Cupertino et al., 2023). These contaminants,

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despite coming from the food sector, have systemic health consequences similar to those from traffic or industrial sources and pose a substantial public health danger.

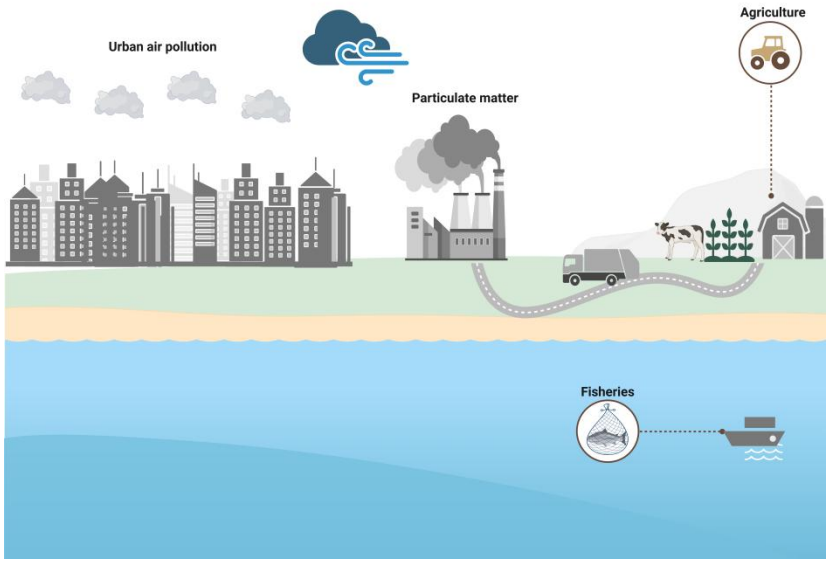

Fig 1 The impact of food processing on PM in urban air.

Air pollution has become a severe public health problem in the world, and, according to the World Health Organization (WHO), about 90 percent of the world's urban residents are exposed to air containing high levels of pollutants, and about 4.2 million people die each year as a result of outdoor air pollution (Organization, 2016). As a result, it is listed as the 13th highest global mortality rate by the WHO (Araujo, 2011). Many previous studies showed that air pollutants not only cause cardiovascular and respiratory disorders, but also directly or indirectly induce liver diseases such as fatty liver, cirrhosis and fibrosis (Chan et al., 2019; Shao et al., 2019; Srimuruganandam & Nagendra, 2012; B.-Y. Yang et al., 2019). Air pollution consists of a wide range of substances in three main categories, namely gaseous constituents, volatile organic compounds (VOCs) and respirable particulate matter (PM). Among them mentioned above, the PM is the most significant factor affecting human health (Garrett & Casimiro, 2011; Perez et al., 2012).

PM comes mainly from solid fuel combustion, industrial activities, and transportation (Organization, 2013). Based on previous studies, PM exposure has been found to cause respiratory symptoms, exacerbation of chronic respiratory and cardiovascular diseases, decreased lung function, and premature death (Halonen et al., 2009; Samoli et al., 2008). Beseides, current researches also found that PM could affect the gastrointestinal tract by altering the permeability of the gastrointestinal membrane, releasing inflammatory cytokines, and inducing apoptosis in colonic cells (Dai, Zanobetti, Koutrakis, & Schwartz, 2014; Mutlu et al., 2018). Meanwhile, PM have an impact on the GM. Results from animal experiments indicated that PM could enter the circulatory system through the lungs, thereby disrupting intestinal microecology, such as the diversity of the GM (Mutlu, et al., 2018). In population-based studies, increased abundance of *Mycobacterium avium* in the gut resulted in disruption of ornithine biosynthesis, coenzyme A biosynthesis I and glycolytic pathways, and findings suggest that GM disorders lead to dysfunction in amino acid synthesis or glucose metabolism, and that the liver is a major site for these biotransformation processes (Fouladi, et al., 2020). Other studies

have found that GM plays an important role in inducing liver disease, and changes in the abundance of Thick-walled *Mycobacteria* and *Mycobacterium*, the most prominent microorganisms in GM, play an important role in liver pathology (Tremaroli, et al., 2012). A decrease in the abundance of *B. thicketi* and an increase in the abundance of *B. anthropophilus* produce large amounts of ethanol and pro-inflammatory factors, which are transported to the liver through the portal vein and induce liver lesions (e.g., ALD, NAFLD, and hepatitis, etc.) (Faria Ghetti, et al., 2018; Acharya, et al., 2017). Thus, alterations in GM can induce liver lesions. However, investigations on the mechanisms of GM on liver lesions are limited according to current researches.

Therefore, this paper combines the changes in GM in recent years, the effects of GM on the liver, and the effects produced by PM on the liver to illustrate the relationship between the liver-intestinal axis and liver lesions caused by PM and to provide an idea for further research on the changes in GM due to air pollution that cause liver injury.

2. The effects of particulate matter on gut microbiome

The GM is composed of a complex tribe of microbiota and also an essential source of metabolites, hormones, and neurotransmitters in the body that significantly impacts human health (Jandhyala et al., 2015). The human gut exhibits over 100 trillion highly metabolically active microorganisms (Round & Mazmanian, 2009) and the GM can indirectly affect the function of other extraintestinal organs of the human body by directly regulating the part of the gut (Park, 2018). This means that good GM plays a crucial role in human health. However, GM in the human body is not stable, and a variety of events can increase the risk of GM dysregulation, such as diet, stress, antibiotic use, and environmental changes et al (Sommer & Bäckhed, 2013).

Previous studies on PM mainly focused on pulmonary and cardiovascular diseases. More profound research on organ damage by PM in humans revealed that PM can be transferred from the lungs to the gastrointestinal tract via mucociliary clearance (Albano, Montalbano, Gagliardo, Anzalone, & Profita, 2022; Feng, Cavallero, Hsiai, & Li, 2020). When PM enters the intestinal tract, it increases the permeability of the intestinal mucosa by effecting the diversity and abundance of GM, which can lead to an increase in mitochondrial reactive oxygen species (ROS), the release of inflammatory factors, and the induction of apoptosis (Mutlu et al., 2011; epommier et al., 2019; R. Li et al., 2017; Plovier et al., 2017; Yassour et al., 2016). In addition, PM comes into touch with intestinal epithelial cells, immune cells and 10^{14} bacteria (Mutlu et al., 2018), and enhances the spread of inflammatory cells. It can be found a decrease in the alpha diversity of human GM exposure to $PM_{2.5}$ and PM_{1}, with a negative correlation between $PM_{2.5}$ and PM_{1} and the relative abundance of *Proteobacteria* using 16S rRNA sequencing (T. Liu et al., 2019; Roslund et al., 2019). These findings indicate that PM significantly alters the composition and abundance of the GM upon entering the intestinal tract. So, how does PM lead to changes in the GM? Current research reveals that PM impacts GM through multiple major processes. First, PM changes host metabolism, thereby generating GM dysbiosis. For instance, Wanjun Wang and colleagues observed that long-term exposure to $PM_{2.5}$ in mice reduced glucose

tolerance and insulin sensitivity, ultimately leading to aberrant glucose metabolism. This metabolic disruption was followed by a reduction in GM diversity, as shown by lower ACE and Chao1 indices (W. Wang et al., 2018). Second, PM promotes intestinal inflammation, which further changes GM composition. Chronic $PM_{2.5}$ exposure lowers the fraction of regulatory T cells (Tregs) and increases Th17 cells, altering the Treg/Th17 balance. Consequently, the expression of anti-inflammatory cytokines such as interleukin-10 (IL-10) and transforming growth factor-β (TGF-β) is downregulated, whereas pro-inflammatory cytokines including IL-17 and IL-6 are considerably increased (Braak et al., 2012). This inflammatory milieu negatively affects beneficial gut microbes; for example, 16S rDNA sequencing indicated a dramatic drop in *L.actobacillus* acidophilus abundance. Moreover, treatment with *L.acidophilus* was observed to decrease PM-induced intestinal inflammation, indicating its protective effect (Jie Xu, Wang, He, Chen, & Meng, 2024). Finally, PM interacts with GM metabolism and inhibits microbial activity. Its chemical components can block important enzymes involved in short-chain fatty acid (SCFA) synthesis [e.g., glycosidase, phosphotransacetylase (PTA), and acetate kinase (ACK)], resulting to lower SCFA levels and further impairing gut homeostasis (Bhardwaj et al., 2024). Meanwhile, the effect of PM on GM was also relatived with age, and the adolescents exposed to $PM_{2.5}$ and PM_{1} showed a decrease in the abundance of *Bacteroidetes* and an increase in the quantity of *Coriobacteriaceae* in the gut (Alderete et al., 2018). However, in adults, $PM_{2.5}$ and PM_{1} induced a reduction in the abundance of *Firmicutes*, *Proteobacteria*, and *Ruminococcaceae* warts in the gut (T. Liu et al., 2019). These results mentioned above indicate that PM can significantly change the structure and quantity of GM after entering the intestine.

In order to properly grasp the effects of PM on intestinal bacteria, we created inclusion and exclusion criteria based on a thorough literature search of PubMed other Web scientific sites. Utilizing MeSH keywords and entry phrases, we created search techniques concentrating on the relationships between PM and GM. To expand our research, we also evaluated additional components of PM, such as PAHs, since they constitute the most important component of $PM_{2.5.}$ The inclusion and exclusion criteria are defined in Table 1, and the search results are presented in Table 2.

It is obvious from these studies in the literature that PM may dramatically promote dysregulation of GM. However, these studies contain problems. First, this literature responds to just part of the changes in colonization for comparison(Dai et al., 2024). Second, the description of colonization largely focuses on the changes in quantity and does not represent the structural changes of colonization in the gut(H. Liu et al., 2024). Finally, the effects of GM on mice treated using the same PM ($PM_{2.5}$) are inconsistent or contradictory(X. Liu et al., 2020). This means that future studies of GM need to explore not only changes in the number of colonies but also their structural alterations, allowing for a relationship between various PMs on GM.

3. Mechanism of liver damage by gut microbiome

Currently, the most common chronic liver diseases are alcoholic liver disease (ALD) and non-alcoholic fatty liver disease (NAFLD), and they can progress to cirrhosis or liver cancer. Recent studies have shown that

the altered composition of GM plays a vital role in developing NAFLD. Typical changes in the GM of patients with NAFLD are an increase in *Bacteroidetes*, a decrease in *Firmicutes*, and an increase in pro-inflammatory taxa such as *Proteobacteria* and *Bacteroides* (de Faria Ghetti et al., 2018). And, patients with NAFLD exposure to PM2.5 can lead to a significant decrease in the GM in both the *Firmicutes* and *Lactobacillus*, and an increase in the *Bacteroidetes* (J. Chen et al., 2021). These changes in GM produce harmful substances that can enter the bloodstream from the intestines via the portal vein, making the liver more susceptible to toxic substances that can lead to inflammation (Macpherson, Heikenwalder, & Ganal-Vonarburg, 2016). In addition, intestinal permeability is an essential factor that influences the impact of GM on the liver, and intestinal permeability prevents harmful bacteria from entering the bloodstream (Turner, 2009). However, when PM enters the gut, it first increases intestinal permeability, which can lead to the leakage of harmful bacteria and their toxic metabolites. This effect further leads to inflammation, leading to non-alcoholic hepatitis, and other complications such as obesity and type 2 diabetes (T2D) (Fasano, 2008; Hong Li et al., 2009; Wong et al., 2012). Therefore, by researches on the changes in GM caused by PM mentioned above, it is known that changes in GM can cause NAFLD and some related complications. Meanwhile, changes in GM can also contribute to ALD, when *E. coli* are increased in the gut, it increases endogenous ethanol, which enters the liver through the portal vein to induce validation activation (Zhu et al., 2013).

Although some studies focused on the consequences of changes in GM on liver injury, the exact processes remain poorly described. Currently, experimental and clinical results suggest that the liver-gut axis plays a vital role in the pathogenesis of NAFLD. Firstly, anatomically, the intestine and the liver are connected through the portal vein. Thus, the liver needs to respond to nutrients, toxins, and antigens in food and to the GM and its products (Baffy, 2019). Secondly, when intestinal permeability is increased, harmful bacteria and inflammatory factors in the gut diffuse to the liver, inducing the onset and progression of NAFLD (Fig 2) (Wigg et al., 2001). Regarding the influence of GM on liver injury, there are currently three predominant pathways, namely gut barrier, metabolites and dysbiosis.

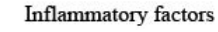

Fig 2 Effects of GM on the enterohepatic axis and the liver. When intestinal permeability is enhanced, dangerous bacteria and inflammatory substances in the intestines will enter the portal vein through the intestinal epidermis and circulate through the portal vein into the liver, leading to metabolic abnormalities in the liver and, thus, the establishment of NAFLD.

3.1 Gut microbiome on intestinal barrier dysfunction

Over the last decade, various results have indicated that the intestinal barrier prevents the transmission of bacteria or bacterial products and is a crucial component in the control of the gut-liver axis. The intestinal barrier is a complex structure that consists of four main components respectively, they are luminal intestinal alkaline phosphatase (IAP), mucus layer, epithelial cell layer, and antimicrobial proteins produced by Paneth cells (Fig 3) (Ghosh, He, Wang, Gehr, & Ghosh, 2018), which work together to block the entry of harmful bacteria or bacterial products into the body's internal circulation. Intestinal barriers also can be defined according to their constituent functions, inciuding extracellular, cellular, immune, and vascular barriers. The

extracellular or luminal barrier prevents the entry of toxins by catalyzing the dephosphorylation of lipopolysaccharide (LPS) through the secretion of IAP. And, it dephosphorylates LPS anddephosphorylates adenosine triphosphate (ATP), which promotes the growth of commensal bacteria and maintains microbial homeostasis (Malo et al., 2014). And, the cellular barrier is mainly responsible for the transportation of substances, in which the villi at the tip of the epithelial cells are negatively charged, and the cellular border can exert electrostatic repulsion on bacteria because of the negatively charged cell wall of bacteria. In addition to the negatively charged epithelial cells that can stop bacteria, the cuprates, and Paneth cells are the additional components that provide the intestinal barrier by producing mucus and antimicrobial proteins, respectively. Occludin and ZO-1 are the most important proteins of the intestinal wall, they are also located on the cellular barrier, and in the case of GM disorders, the GM inhibits the expression of specific tight junction proteins, i.e., obstruction proteins and ZO-1, which leads to an increase in intestinal permeability (Cani et al., 2007). The most crucial role of the immune barrier is to disrupt it by binding directly to bacteria through the production and secretion of proteins such as defensins, antimicrobial peptides, resistin-like molecules, bactericidal permeability-inducing proteins, and lectins, as well as attenuating the ability of bacteria to bind directly to each other and the epithelial cell layer. The vascular barrier is a significant component of the enteric nervous system. Its primary function is to effectively organize the entry of bacteria into the liver through the portal vein when the intestinal epithelial barrier is disrupted (Spadoni et al., 2015).

Fig 3 Functional components of the intestinal barrier. (a) Extracellular barrier is composed of the mucus layer secreted by the lumen and the bacteria in the gut, which is mainly responsible for the regulation of local antimicrobial peptides and the production of immune proteins, (b) Cellular barrier is composed of the epithelium, which is mainly responsible for the restriction of intracellular transport, (c)Immunological barrier is composed of antimicrobial proteins secreted by the immune cells, which can bind directly to the bacteria, (d) Vascular barrier is composed of the endothelium of the vessels, which is the essential part of the enteric nervous system.

3.2 Metabolites of gut microbiome

After the food is digested by stomach acid, it enters the intestines, where the GM breaks down the rehashed material to produce a variety of metabolites, which are transported through the body to regulate the organs of the human body, for the liver metabolites enter the liver mainly through the portal vein to regulate its metabolism and health.

Ethanol is an essential product of gut microbial metabolism. Several studies have shown significantly increased levels both of ethanol in the gut of patients with NAFLD and abundance of some ethanol-producing flora (grown lot of γ-Propionibacterium and Prevotella) (Del Chierico et al., 2017; Michail et al., 2015; Zhu et al., 2013). And, the GM also contains alcohol-metabolizing enzymes, including alcohol dehydrogenase, which converts ethanol to acetaldehyde and acetate (Chaudhry et al., 2016; Mir et al., 2016). The resulting acetaldehyde can weaken tight junction protein connections, damaging the gut barrier and translocating gut microbes. To verify whether ethanol metabolized by GM causes liver damage, Yuan, Jet et al. cited a high

alcohol-producing strain of *Klebsiellapneumoniae* (HiAlc Kpn) that caused NAFLD in mice (Yuan et al., 2019). The metabolites of ethanol may contribute to the oxidative stress of free fatty acids, which, in turn, affects the progression of NAFLD (Z. Chen, Tian, She, Cai, & Li, 2020). GM produces other hepatotoxic alcohols besides ethanol, such as methanol, which is produced as a byproduct of pectin metabolism and vitamin B_{12} in some microorganisms (Dorokhov, Shindyapina, Sheshukova, & Komarova, 2015).

Hepatotoxic alcohols are not the most important metabolites produced by GM, but other metabolites are also created, among which the substance with the most serious impact on liver disease is GNB. GNB have a high relative abundance (relative abundance ≥30%) in the GM (Stoma et al., 2021). Therefore, bacterial LPS, an essential component of GNB, is a metabolite of GM with high intestinal content. LPS can increase the concentration of portal vein LPS through intestinal damage induced by endogenous ethanol produced by GM and thus by portal vein LPS (Malaguarnera, Giordano, Nunnari, Bertino, & Malaguarnera, 2014). In addition to the activation of blastic cells and hepatic stellate cells, which can induce hepatitis, LPS also affects the toll-like receptor-4 (TLR-4)-dependent mechanism and affects the TLR-4 signaling pathway, causing insulin resistance, inflammation, and hepatic steatosis. and hepatic steatosis (X. Yang et al., 2019). In addition, LPS promotes the release of tumor necrosis factor-α (TNF-α) from macrophages in the liver, which plays a vital role in the interaction between lipid accumulation and inflammatory response (Macaluso, Maida, & Petta, 2015). Furthermore, a experiments investigating liver damage induced by LPS in mic found that the rate of hepatic steatosis was accelerated after 24 weeks in mice fed LPS compared to controls (Fukunishi et al., 2014). All restuls mentioned above suggested that the increase in LPS content positively correlates with the degree of validation of liver disease.

Moreover, short-chain fatty acids (SCFAs) are also essential metabolites of GM. Plant-derived polysaccharides are first partially catabolized by enzymes in the host and then fermented by microorganisms in the colon to produce SCFAs. Among these SCFAs, acetic acid, propionic acid, and butyric acid are the main products of carbohydrate fermentation by GM, and the gut microbiota produces about 50-100 mmol/L SCFAs per day (Leung, Rivera, Furness, & Angus, 2016). In addition to plant-derived polysaccharides, which are used as the raw material for the fermentation of SCFAs, GM can also produce branch SCFAs through amino acid fermentation (Nyangale, Mottram, & Gibson, 2012). Most of these SCFAs are consumed by the gut. Still, some are absorbed by the intestinal epidermis and transported to the liver through the portal vein, where they participate in lipid metabolism. Some researches showed that supplementation with acetic or propionic acid reduces hepatic fat synthesis and fatty acid uptake and protects mice from high-fat diet (HFD) induced weight gain, steatosis, and insulin resistance (Koh, De Vadder, Kovatcheva-Datchary, & Bäckhed, 2016). Butyrate also plays a vital role in the gut-liver axis, which is responsible for essential SCFAs that maintain the stability of the intestinal barrier (Yaku et al., 2012; Ziegler, Kerimi, Poquet, & Williamson, 2016). When butyrate levels are reduced, it weakens the intestinal tight junctions, increasing the intestinal barrier's permeability (Cresci et al., 2017). In addition to their involvement in synthesizing sugars and fats in the liver, a relationship exists between SCFAs and metabolic disorders. For example, an increase in butyrate prevents

liver steatosis induced by a high-fat diet degeneration (Endo, Niioka, Kobayashi, Tanaka, & Watanabe, 2013). Regarding inflammation, butyric acid and propionic acid have favorable anti-inflammatory effects, they protect the integrity of the intestinal barrier mainly through their impact on the activation of intestinal epithelial cells, inducing the secretion of interleukin-18 (IL- 18). They also promote antibody production by regulating B-cells' energy metabolism. They also promote the expansion of regulatory T-cells from the head of differentiation (M. Kim, Qie, Park, & Kim, 2016).

Nowadays, the liver-bile acid axis is also coming into the research picture. Primary bile acids are synthesized from cholesterol in the liver, secreted by hepatocytes, and enter the duodenum through the bile duct. Bile can be used to maintain the integrity of the intestinal barrier to prevent intestinal bacterial translocation, and bile also has antimicrobial activity (Poeta, Pierri, & Vajro, 2017). In the intestinal tract, the GM located in the small intestine and colon processes the primary bile secreted from the bile ducts for decoupling and dehydroxylation, converting it into primary secondary bile acids. Secondary bile acids aid in the digestion and absorption of nutrients such as fats, cholesterol, and fat-soluble vitamins ingested by the body. Of these 95% of secondary bile acids are reabsorbed at the end of the ileum and circulate to the liver through the portal vein in a cycle known as the hepato-biliary axis. Bile acids act as endogenous ligands for the nucleofarnesoid X receptor (FXR) and bind to the FXR to control lipogenesis in the liver. When GM produce excess secondary bile acids, it inhibits gluconeogenesis and glycogenolysis in the liver and increases insulin sensitivity in adipose tissue and skeletal muscle with FXR binding (Shu et al., 2021; Taoka et al., 2016). In addition to acting as a ligand for FXR, secondary bile acids are also endogenous ligands for the membrane receptor GPCR 5 (TGR5), which, after activation by secondary bile acids, maintains glucose homeostasis in vivo by inducing enteroendocrine cells (EECs) to regulate glucagon-like peptide 1 (GLP-1) secretion (H. Kim & Fang, 2018). Thus, secondary bile acids can alleviate NAFLD through FXR and TGR5.

3.3 Gut microbiome Dysbiosis

With the deeper study of human GM, many liver diseases associated with dysbiosis of the GM were found (Fig 4). Dysbiosis of GM mainly refers to changes in the composition and function of the GM. Dysbiosis of GM is mainly caused by several factors such as family genetics, physical condition, lifestyle and environmental factors, among which environmental factors are the most important.

For the development of ALD there are currently three major factors that we know of, including alcohol content, genetics and environmental factors. With further research, dysregulation of the GM as another important factor, is also found. One study found accelerated alcohol-induced ALD pathogenesis by transplanting the GM of ALD patients into mice (Llopis et al., 2016). Through the transplantation of the flora, the GM in the mice became dysregulated and there was an increase in the permeability of the intestinal barrier, leading to the entry of microbial metabolites into the liver through the portal vein. Dysbiosis of the GM not only induces the production of ALD individually, but also contributes to the development of ALD, which can lead to the progression of ALD toward hepatic inflammation and cirrhosis when fungal overgrowth and translocation of fungal products occurs (A.-M. Yang et al., 2017).

Dysbiosis of the GM has a notable influence on the induction and development of liver disease, and although there is a vast range of bacteria in the gut, intestinal dysbiosis is predominantly connected with the four major bacterial phyla in the gut, including *firmicutes*, *bacteroidetes*, *actinobacteria*, and *proteobacteria* (Mokhtari, Gibson, & Hekmatdoost, 2017), with as many as 90% of microorganisms estimated to belong to the thick-walled and anaplasmodial phyla. Changes in these phyla can have an impact on NAFLD. In one study, a comparison was made between a healthy group and a NAFLD group, in which the healthy group had a 20% increase in *Mycobacterium* anisopliae while the thick-walled phylum decreased by 24% (B. Wang et al., 2016). In addition, the species found to have a reduced thick-walled bacterial phylum were mainly those producing SCFA, which may contain an important message that the dysbiosis of the GM disrupts the intestinal barrier integrity and innate mucosal immune homeostasis. In addition to this, the pathogenesis of NASH patients has been associated with changes in Anaplasma phylum and Aspergillus spp (Consolandi et al., 2015). Mouzaki et al. found that the diagnosis of NASH was inversely correlated with the proportion of Anaplasma phylum abundance in the intestinal tract of the patients and also found that the diagnosis of NASH was accompanied by a higher level of alcohol-producing bacteria and endogenous ethanol levels (Mouzaki et al., 2016). Zhu L et al. found that in addition to an increase in the abundance of Anaplasma phylum in NASH patients, a concomitant a significant increase in the abundance of Anaplasma phage, and a decrease in the abundance of Thick-walled bacteria phage (Zhu et al., 2013).

Furthermore, small intestinal bacterial overgrowth (SIBO) is also associated with NAFLD. When small intestinal bacterial overgrowth first leads to the loss of several endogenous defense mechanisms such as gastric acid secretion, intestinal motility, intact ileo-caecal valve, immunoglobulins in intestinal secretion, and pancreatic and biliary secretion (Quigley, 2014). In recent studies, it has been shown that SIBO is prevalent in NAFLD patients and plays a key role in the pathogenesis of NAFLD. In a clinical study that examined 23 NASH patients and 23 healthy participants, elevated concentrations of SIBO and TNF-a were found in NASH patients. However, no increased intestinal permeability or higher endotoxin levels were found (Fitriakusumah et al., 2019).

Liver cirrhosis is a serious chronic liver disease, is associated with GM dysbiosis. In patients with cirrhosis, the GM has a change from normal components to a colony dominated by pro-inflammatory bacteria, and these pro-inflammatory bacteria will produce a large number of LPS to break down the intestinal barrier and enter the liver, which accelerates liver damage and increases the systemic inflammatory response, as well as inducing other complications such as portal hypertension, variceal bleeding and ascites (Wigg et al., 2001). In addition, when the GM of the body is in a state of pro-inflammatory bacteria, not only cirrhosis, a liver disease, can be induced, but also hepatocellular carcinoma (HCC) can develop. It has been found that the α-abundance of bacterial flora in the feces of patients with HCC undergoes a significant reduction in comparison with that of the healthy population, and after 16S bacterial RNA sequencing, it was shown that, using patients with cirrhosis without HCC as a control, the abundance of *Bacteroides simulans*, *Rumatococcus*, *Enterococcus*, *Phage*, and *Schephelococcus* was increased in the feces of patients with HCC,

and that *Bifidobacterium* and *Lampsococcus* were decreased in the feces of patients with HCC. Since the reduction of anti-inflammatory microorganisms such as Bifidobacterium and Lancet promote hepatocellular carcinoma (Acharya, Sahingur, & Bajaj, 2017).

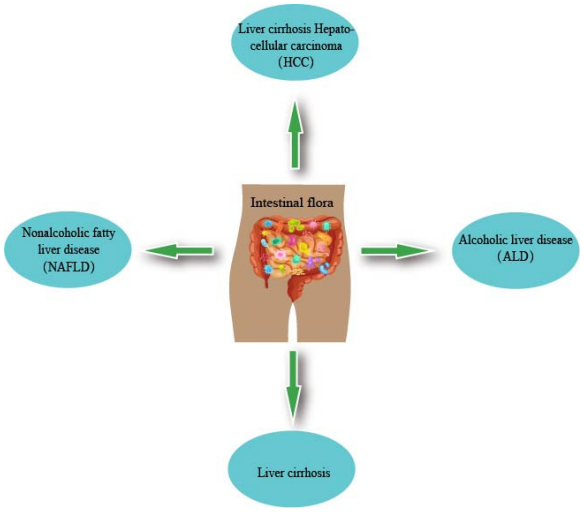

Fig 4 Effects of dysbiosis of GM on the liver

4. The effect of particulate matter on liver through gut microbiome

Previous studies have shown that human exposure to PM can affect GM and also can cause liver damage, but no link was made between them. It was not until a few years ago that studies proposed the concept of the gut-liver axis to investigate the interrelationship between PM-GM-liver. Animal experiments have shown that airborne PM enters the circulatory system through the lungs, thereby interfering with the GM and disrupting the balance between oxidative stress and antioxidants in mice, which in turn causes a significant increase in the levels of 4-HNE, MDA, and 8-OHdG in the liver of mice (Mutlu et al., 2018). Moreover, it was found that the abundance of GM in the PM2.5-exposed group increased in the thick-walled and actinomycetes phylum and decreased in the bacillus phylum at the phylum level, and it was also demonstrated that the abundance of thick-walled and actinomycetes phylum was positively correlated with the hepatic levels of 4-HNE, and MDA (N. Wang et al., 2019). Table 3 shows instances of research in which PM and its components have led to abnormalities in the intestinal flora and, consequently, hepatic fat buildup.

NO_2 an inorganic component of PM2.5, can potentially produce GM dysbiosis and lead to liver injury. Weizhuo Yi et al. observed that NO_2 exposure from PM2.5 reduced the number of *Actinomycetales* and *Coriobacteriales* in the human gut. Among them, *Coriobacteriales* plays a significant mediating function in NO_2-induced liver injury, since it is an important producer of short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate (Hongli Li et al., 2024). A fall in the quantity of *Coriobacteriales* leads to lower SCFA levels in the gut, which affects the integrity of the intestinal barrier and increases the translocation of endotoxins (e.g., lipopolysaccharide, LPS) into the liver via the portal circulation (Di Vincenzo, Del Gaudio, Petito, Lopetuso, & Scaldaferri, 2024). Additionally, SCFAs are known to decrease pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6); hence, their loss results in increasing levels of these cytokines, ultimately promoting liver inflammation (Yi et al., 2021).

Cadmium (Cd), a metallic component of PM2.5, not only directly produces liver injury but also exacerbates hepatic damage by affecting the GM (Di Ciaula et al., 2020). Studies have revealed that exposure to modest doses of Cd in mice leads to a considerable decrease in the relative abundance of *Firmicutes* and a contemporaneous increase in *Bacteroidetes* at the phylum level. Within the *Firmicutes* family, considerable reductions are noted in *Paraprevotellaceae*, *Helicobacteraceae*, *Ruminococcaceae*, and *Streptococcaceae*, alterations that are connected with the advancement of liver disorders. In addition to structural and compositional abnormalities of the GM, Cd also promotes metabolic problems that further aggravate hepatic damage. According to a study by Zhang S et al., metagenomic investigation of Cd-exposed mice found a considerable rise in the abundance of genera such as *Alloprevotella*, *Prevotella*, and *Anaeroplasma*, alongside a drop in *U29–B03* and *Turicibacter* (S. Zhang, Jin, Zeng, Liu, & Fu, 2015). Furthermore, metabolomics profiling indicated a reduction in conjugated primary bile acids (e.g., cholic acid glucuronide) and an increase in detoxifying products of secondary bile acids (e.g., deoxycholic acid-3-glucuronide). These data demonstrate that Cd exposure leads to bile acid dysregulation, defined by reduced conjugation of main bile acids and elevated synthesis of detoxified secondary bile acids, which may contribute to liver damage and undermine intestinal barrier integrity (Gu, Xu, & Li, 2025).

PM contains not only of inorganic and metallic components but also a large number of organic molecules, among which PAHs are essential contributors. Exposure to benzo[a]pyrene (BaP), a typical high-molecular-weight PAHs (HMW-PAHs), has been found to change GM composition and trigger hepatic lipid metabolic abnormalities. In mice exposed to BaP, the relative abundance of *Odoribacter*, *Bifidobacterium*, and *Actinomyces* rose. Notably, *Odoribacter* exhibited a strong positive correlation with hepatic triglyceride (TG) levels and the expression of lipid metabolism–related genes such as PPAR-γ and GPAT ($r = 0.79$, 0.91, and 0.91, respectively), possibly due to its capacity to produce butyrate, which may promote hepatic lipid accumulation via host lipid metabolic pathways. *Bifidobacterium*, a well-known probiotic, plays a key function in maintaining intestinal barrier integrity and boosting host health. Its increased abundance may constitute a compensatory response aimed at minimizing BaP-induced hepatic damage. In contrast, Actinomyces, whose abundance is known to rise in circumstances such as nonalcoholic fatty liver disease (NAFLD) and alcoholic fatty liver disease (AFLD), may serve as a microbiological marker of hepatic pathology (Jia et al., 2020; M. Yang et al., 2024). In addition to BaP, low-molecular-weight PAHs (LMW-PAHs) also produce hepatotoxic effects via the gut–liver axis. For instance, Xiu Yu et al. observed that phenanthrene (PHE) treatment in mice significantly increased the abundance of pro-inflammatory gut bacteria such as *Helicobacter*, *Bacillus*, and *Bacteroides*, while decreased the quantity of beneficial microbes like *Allobaculum*. These inflammatory strains enhance the expression of cytokines including IL-6, IL-22, and TNF-α, thereby inducing liver inflammation and lipid metabolic problems through gut-derived immune activation (X. Yu, Lv, Guan, Zhang, & Sun, 2021).

5. LAB as an intervention to prevent or respond to PM-induced liver injury

LAB has excellent immunomodulatory properties as a natural immunomodulator, and its role in improving gastrointestinal, oral and vaginal disorders is well known(Margiotta et al., 2021; Z. Zhang, Lv, Pan, & Zhang, 2018; Zheng, Guo, Wang, Zhou, & Ling, 2021). In terms of intestinal colonization, Jotham Suez et al. demonstrated in a study utilizing pseudo-germ-free mice that LAB had good colonization capacity in the gut (Suez et al., 2018). Regarding PM-induced oxidative stress, studies have shown that *Lactobacillus acidophilus TW01* can significantly inhibit reactive oxygen species (ROS) production triggered by $PM_{2.5}$, promote the repair of Caco-2 intestinal epithelial cells, and enhance intestinal barrier function, thereby effectively mitigating damage to the gut–liver axis (Luo & Chen, 2023). Furthermore, *Lactobacillus rhamnosus GG* has been observed to lower hepatic levels of inflammatory cytokines such as IL-6 and TNF-α, while altering bile acid and lipid metabolism in response to environmental pollutants including PM and microplastics (C. Yu et al., 2024). In addition, compared with other probiotic strains, LAB has exhibited better binding capacity to several components of PM, hence lowering the absorption of harmful chemicals by the host. In an in vitro binding experiment, M. Yousefi et al. evaluated the binding affinities of nine probiotic bacteria with four polycyclic aromatic hydrocarbons (PAHs), namely benzo[a]pyrene (BaP), benz[a]anthracene (BaA), chrysene (Chr), and benzo[b]fluoranthene (BbF). The results demonstrated that *Lactobacillus acidophilus LA-5* had the highest binding capability among the investigated strains, whilst *Bifidobacterium lactis BB-12* showed the smallest affinity (Yousefi et al., 2019). Meanwhile, new research findings suggest that LAB can improve liver injury. *Lactobacilli* have been proven to play a beneficial role in liver diseases, including NAFLD, ALD, cirrhosis, drug-induced liver injury (DILI) cholestatic liver disease and viral hepatitis(Leitner, Brits, Xu, Patil, & Sun, 2024; Huizhen Li et al., 2020; Monteiro et al., 2019; Ritze et al., 2014; Wong et al., 2013; Wu et al., 2022). Therefore, administration of Lactobacillus is an effective strategy to ameliorate PM-induced liver injury.We will discuss the mechanism of LAB intervention in PM-induced liver injury based on the available studies.

5.1 LAB Physical Adsorption PM

Physisorption is the main mechanism by which LAB reduces PM in vivo. Currently, experiments on the physical adsorption of LAB with PM mainly focus on PAHs and lead (Pb).The adsorption of PAHs and Pb by LAB is mainly related to polysaccharides and proteins on the cell wall of LAB.The physical adsorption of peptidoglycan on LAB with PAHs mainly relies on electrostatic and hydrophobic interactions.1) The anionic groups (e.g., carboxyl groups) in the peptidoglycan and the charge distributions of the surface of PAHs attract each other, forming electrostatic interactions; 2) the peptidoglycan surface contains a large number of hydrophobic regions, which can adsorb PAHs through hydrophobic interactions. attraction, forming electrostatic interactions(Sangsila, Faucet-Marquis, Pfohl-Leszkowicz, & Itsaranuwat, 2016; Yousefi et al., 2019), which can adsorb PAHs through hydrophobic interactions.The physical adsorption of peptidoglycan on LAB with Pb mainly relies on the proteins on the cell wall; 1) soluble Pb can enter and transport to the cytoplasm through membrane proteins and be isolated in specific regions, reducing its toxicity; 2) Pb binds to the proteins on the LAB cell wall which in turn results in the presence of Pb in the form of harmless and metal

chelates(Sun et al., 2007; Wang XiaoWei et al., 2014).The physisorption of LAB avoids secondary contamination by toxic secondary metabolites generated during PM degradation (Fig 5) (Lili, Junyan, Hongfei, Baoqing, & Bolin, 2018).

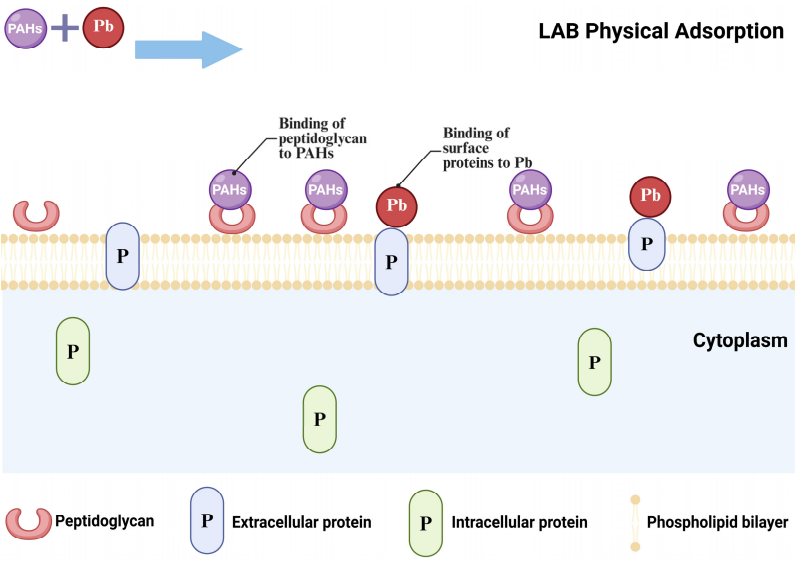

Fig 5 Physical adsorption effect of LAB.

5.2 Interaction of lactic acid bacteria with GM

In addition to its physical adsorption effect, the biological role of LAB is also crucial.LAB can regulate PM-induced intestinal flora disorders by entering the intestinal interior. First, for PM-induced intestinal pathogenic bacteria, LAB can reduce the impact of intestinal pathogenic bacteria on the organism through competitive exclusion, which is because the collagen and fibronectin on the surface of lactic acid bacteria will produce competitive exclusion with the adhesion proteins of the pathogenic bacteria(Szajewska & Kołodziej, 2015), however, this adhesion of lactic acid bacteria is strain-specific.In addition to reducing the impact of pathogenic bacteria on the health of the organism through the competitive exclusion, LAB can also reduce the impact of pathogenic bacteria on the health of the organism through the production of Lactobacilli, which is the most important biological role of LAB. In addition to reducing the health effects of pathogenic bacteria in the gut through competitive exclusion, LAB can also produce bacteriocins, which, by binding to lipid II, can create pores in the bacterial membrane of pathogenic bacteria, leading to the loss of internal metabolites of the pathogenic bacteria, and ultimately leading to cell death(Dickman, Mitchell, Figueiredo, Hansen, & Tabor, 2019).

SCFAs, such as lactic acid, butyric acid, formic acid, and acetic acid, are well-known metabolites generated by yeast and other microbes that can reduce both internal and extracellular pH, hence reducing the growth of pathogenic bacteria (Markowiak-Kopeć & Śliżewska, 2020). In addition, organic acids have been demonstrated to decrease protein expression in filamentous fungi (Ström, Schnürer, & Melin, 2005). The antibacterial action requires SCFAs permeating the pathogen's cell membrane in their undissociated state (R-COOH). Once inside the near-neutral cytoplasmic environment of the pathogen, they rapidly dissolve into H^+ and $R\text{-}COO^-$ ions, leading to intracellular acidification. This acid stress disrupts critical enzymatic activity

and metabolic processes, slows protein synthesis, and eventually leads in growth arrest or cell death (Fig 6) (Chang, Nagarajan, & Gan, 2024; Den Besten et al., 2013; Strain, Stanton, & Ross, 2022).

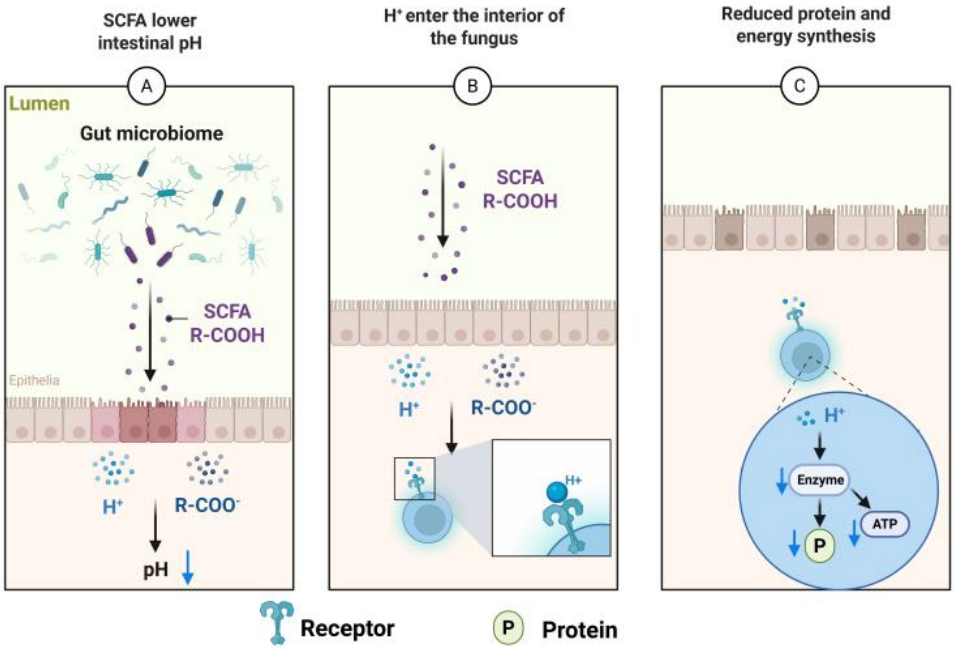

Fig 6 The destructive effect of SCFAs on pathogenic bacteria.

In addition, SCFAs can increase energy metabolism, inflammatory response, and bile acid metabolism in the body. Firstly, SCFAs can bind to the free fatty acid receptor 3 (FFAR3) on the liver. Upon activation, FFAR3 activates the AMP-activated protein kinase (AMPK) pathway. When the AMPK pathway is engaged, it limits fatty acid production [such as by decreasing the activity of acetyl-CoA carboxylase (ACC) and fatty acid synthase (FAS)], while also increasing fatty acid β-oxidation and controlling lipid homeostasis; Furthermore, activated FFAR3 can promote PPAR-α expression through neural or metabolic control, consequently stimulating the expression of lipid metabolism-related genes, such as carnitine palmitoyltransferase 1 (CPT1) and peroxisomal acyl-CoA oxidase 1 (ACOX1), to enhance fatty acid breakdown (Ang & Ding, 2016; Kimura et al., 2013; Tolhurst et al., 2012). Secondly, SCFAs (acetic acid, propionic acid, and butyric acid) can activate G protein-coupled receptor 43 (GPR43) in the liver. Activated GPR43 can block the NF-κB signalling pathway, lowering the expression of pro-inflammatory molecules (TNF-α, IL-6, and IL-1β); Additionally, GPR43 activation promotes the inhibition of inflammasome 3 (NLRP3), hence reducing the maturation and release of IL-1β (X.-f. Liu et al., 2023; Mann, Lam, & Uhlig, 2024; Yao et al., 2022). Finally, SCFAs increase the release of fibroblast growth factor 19 (FGF19) by intestinal cells. After entering the liver via the portal vein, FGF19 inhibits the expression of cholesterol 7α-hydroxylase (CYP7A1), a key enzyme in bile acid synthesis, thereby reducing bile acid production; Additionally, SCFAs can regulate the farnesoid X receptor (FXR) signalling pathway, influencing hepatic cells' perception and metabolism of bile acids (Fig 7) (Collins, Stine, Bisanz, Okafor, & Patterson, 2023; T. Li & Chiang, 2014; Sayin et al., 2013).

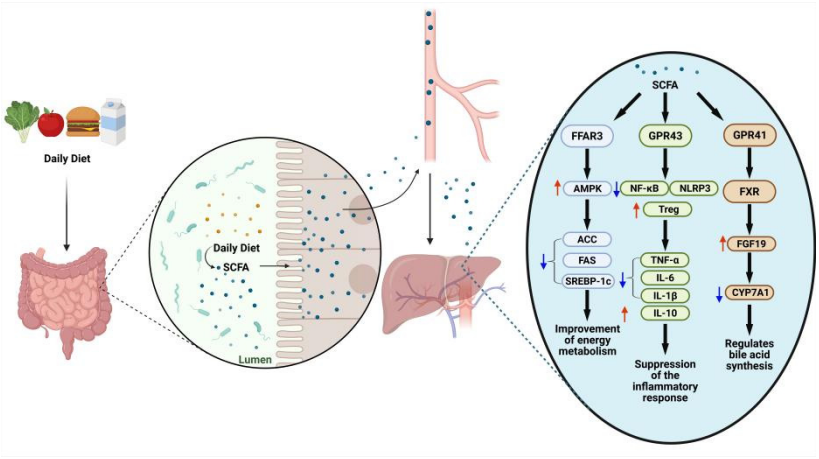

Fig 7 SCFAs improve energy metabolism, inflammatory response, and bile acid metabolism in the body.

LAB are not only antagonistic to pathogenic bacteria, but also have a symbiotic relationship with probiotics in the gut. For example, Lactobacillus can break down sugars in the gut into lactic acid, which can be metabolized by lactic acid utilizing bacteria(Jian Xu & Gordon, 2003). In addition to this. Polysaccharides synthesized by LAB (oligosaccharides fructose and galactosaccharides), these polysaccharides can be used as prebiotics, which can be used for the growth and metabolism of beneficial bacteria in the intestinal tract(Gibson et al., 2017).

5.3 LAB metabolites

LAB metabolites are important components of LAB in maintaining the health of the organism. Organic acids (lactate, butyrate, formate, and acetate) are well known LAB metabolites that have the ability to lower intra- and extracellular pH and thus inhibit the growth of pathogenic bacteria(Mao, Zhang, & Xu, 2020). In addition, organic acids inhibit protein expression in filamentous fungi(Ström et al., 2005). Lactate, as a major component of organic acids, increased lactate content contributes to the antimicrobial activity of LAB against Salmonella typhimurium, and protonated lactate (non-dissociated lactate) possesses both antimicrobial and immunomodulatory activities(De Keersmaecker et al., 2006; Tachedjian, Aldunate, Bradshaw, & Cone, 2017), and butyrate is also an important component of organic acids, which promotes intestinal epithelial barrier function, including the promotion of IEC proliferation, stabilization of hypoxia-inducible factor (HIF) to orchestrate barrier protection, and the inhibition of histone deacetylases (HDACs), and activation of G protein-coupled receptors (GPCRs) in IEC and immune cells for anti-inflammatory effects(Kelly et al., 2015). In addition to organic acids, secreted proteins are also important metabolites of LAB, and these proteins enhance the intestinal epithelial barrier function, which can effectively prevent harmful substances (LPS) from entering the liver through the intestinal barrier(Han et al., 2019).

4. Summary and prospect

In summary, PM can not only alter the composition of the GM, which in turn can cause intestinal inflammation, but it can also transport inflammation-triggering bacteria to the liver through the portal vein or damaged intestinal barriers causing liver damage. An unfavorable intestinal environment increases the risk of liver disease.

However, there are few studies on the mechanisms by which PM affects liver inflammation through the GM at present, and the study of the "gut-liver axis" has just begun in recent years. The research on it mainly focuses on NAFLD and ALD, which is mainly based on animal studies, and there is not much data on clinical studies, and some of the clinical studies have inconsistent results with animal experiments. Therefore, more experiments are needed to improve the credibility of animal experiments in the study of the enterohepatic axis and to provide a strong basis for the effects of PM on the enterohepatic axis in humans. In addition, the effects of PM on the "gut-liver axis" can be modulated by LAB,such as physisorption, interaction with GM, and LAB metabolites (e.g., SCFAs, lactate, and butyrate), which can provide a cost-effective way to prevent the effects of PM exposure on intestinal and hepatic inflammation.

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Declaration of competing interest

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Data availability

No data was used for the research described in the article.

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