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Gut Microbiota-short-chain Fatty Acid Axis: Novel Perspective for Traditional Chinese Medicine Intervention in Acute Pancreatitis

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Abstract: ***In recent years, the role of gut microbiota and its key metabolites, short-chain fatty acids (SCFAs), in acute pancreatitis (AP) has gained significant attention. Traditional Chinese medicine (TCM), as an important traditional medical system, demonstrates unique potential in regulating gut microbiota balance and treating AP. This review explores recent research progress on TCM intervention in AP through modulation of the gut microbiota-SCFAs axis, to provide novel insights and strategies for the prevention and treatment of AP.***

Keywords: Traditional Chinese medicine, Short-chain fatty acids, Gut microbiota, Acute pancreatitis**.**

1. Introduction

Acute pancreatitis (AP), as a common digestive emergency worldwide, exhibits significant regional variations in incidence, ranging from 4.9 to 73.4 per 100,000 population [1]. Over the past two decades, the incidence of AP in China has increased from 0.19% to 0.71% [2], and its onset is primarily associated with factors such as biliary stones, alcohol abuse, and hyperlipidemia [3, 4]. Clinically, mild AP is more common with a relatively low mortality rate; however, the mortality rate in severe cases can be as high as 30%-40% [5]. The core pathological mechanism involves the abnormal activation of pancreatic enzymes leading to pancreatic autodigestion, accompanied by an inflammatory cascade and oxidative stress.

The gut microbiota is a complex symbiotic microbial community residing in the human intestine, which produces metabolites such as short-chain fatty acids (SCFAs) through the fermentation of dietary fiber. These metabolites play a key role in maintaining intestinal barrier function, inhibiting inflammation, regulating the immune system, promoting energy metabolism, and preventing digestive system diseases such as colorectal cancer, acting as crucial regulators of intestinal and overall health [6]. Studies have confirmed that an imbalance in the composition or function of the gut microbiota is closely associated with the pathological processes of various digestive system diseases, including AP [7]. Traditional Chinese medicine (TCM), as a traditional medical system inherited for thousands of years in China, leverages the core advantages of a holistic view and treatment based on syndrome differentiation. It demonstrates unique potential with multi-target, multi-pathway synergistic effects in regulating gut microbiota balance, improving intestinal microecological disorders, and clinically treating AP [8].

Based on this, this review focuses on the gut microbiota-SCFAs axis, systematically integrating relevant experimental evidence on TCM treatment for AP. It analyzes the common and specific mechanisms by which TCM regulates this axis, clarifying the core regulatory chain where microbiota remodeling leads to increased SCFAs, thereby mediating intestinal barrier repair and inflammation inhibition. This provides a theoretical basis for multi-target TCM interventions in AP and lays the foundation for subsequent development of precision treatment strategies and clinical translation [9].

2. Gut Microbiota-SCFAs Axis

The gut microbiota-SCFAs axis is a research hotspot in the field of microbiomics in recent years, revealing a complex regulatory network formed between the gut microbiota and the host through SCFAs. The human gut microbiota is a highly complex and diverse microbial ecosystem, primarily composed of five major bacterial phyla: Firmicutes, Bacteroidetes, Actinobacteria, Proteobacteria, and Verrucomicrobia [10]. Each phylum contains multiple genera with specific functions, which collaborate to maintain the stability and functionality of the intestinal environment. Among these, Firmicutes and Bacteroidetes account for approximately 90% of the total microbiota. They play a key role in breaking down complex carbohydrates and can produce various metabolites, including SCFAs [11]. SCFAs have become core molecules connecting the structure of the gut microbial community with host health homeostasis, underpinned by multi-level regulatory pathways and potential intervention targets.

2.1 Synthesis Process of SCFAs

SCFAs primarily include acetate, propionate, and butyrate, along with small amounts of valerate and isovalerate. In the intestines of healthy adults, the typical ratio of acetate, propionate, and butyrate is 3:1:1 [12], and these three are the focus of this article. The synthesis of SCFAs relies on a complex metabolic network to achieve efficient production.

Acetate is mainly produced by bacteria such as Bacteroidetes and Firmicutes through the fermentation of dietary fiber or resistant starch [13]. Bifidobacteria can convert lactate into acetate via cross-feeding [14], and the fermentation of branched-chain amino acids also contributes a small amount to its synthesis [15, 16]. Propionate is primarily generated through carbohydrate and amino acid metabolism. In carbohydrate metabolism, the core pathway is the vitamin

B12-dependent succinate pathway [17, 18], dominated by Bacteroidetes and Firmicutes. Additionally, the propanediol pathway, involving bacteria such as those in the family Lachnospiraceae, also contributes [19, 20]. Butyrate synthesis mainly relies on the fermentation of carbohydrates and amino acids [21]. In carbohydrate metabolism, acetyl-CoA produced during glycolysis is converted to butyrate via two enzymatic pathways. This process is widespread in the butyryl-CoA: acetate CoA-transferase pathway found in the families Ruminococcaceae and Lachnospiraceae within the phylum Firmicutes [22]. Furthermore, lactate and succinate can be converted into butyrate through cross-feeding [23, 24], and this conversion efficiency is enhanced under low pH conditions [24].

Together, these metabolic pathways constitute the complex network of SCFA synthesis, enabling their efficient production.

2.2 Functions of SCFAs

SCFAs are core metabolites derived from the fermentation of dietary fiber by the gut microbiota, playing three synergistic roles in intestinal health: strengthening intestinal barrier integrity to build a physical barrier, regulating inflammatory responses to balance immune status, and maintaining energy balance to stabilize metabolic homeostasis. From a mechanistic perspective, these effects are associated with factors such as the activation of free fatty acid receptors by SCFAs, the regulation of epigenetic modifications, and their mediation of the immune-metabolic axis [6, 25, 26].

Acetate, with the highest concentration in the intestine, participates in peripheral fat metabolism and immune regulation. It not only mediates anti-inflammatory effects by binding to G protein-coupled receptor (GPR) 43 but also maintains immune homeostasis through epigenetic pathways [27-29]. Propionate is primarily metabolized in the liver. It synergizes with butyrate to induce the differentiation of regulatory T cells (Tregs) by inhibiting histone deacetylase (HDAC) activity, thereby suppressing intestinal inflammation. Additionally, it can independently regulate metabolic and immune signaling through high-affinity binding to the GPR41 receptor [30]. Butyrate is the main energy source for intestinal epithelial cells. It significantly enhances intestinal barrier function and exerts anti-inflammatory effects by inhibiting HDAC and activating peroxisome proliferator-activated receptor γ (PPARγ) [31, 32]. Its specific functions include: (1) exerting anti-inflammatory effects by blocking the activation of nuclear factor κB (NF-κB), downregulating the secretion of pro-inflammatory cytokines such as interleukin (IL)-6 and IL-12, while promoting the production of the anti-inflammatory cytokine IL-10; (2) improving intestinal barrier integrity by upregulating the expression of tight junction proteins Claudin-1, ZO-1, and Occludin [33] ; and (3) maintaining the structural and functional homeostasis of the intestinal epithelium by regulating the dynamic balance between proliferation and apoptosis of crypt epithelial cells [34]. It is noteworthy that the biological effects of butyrate in the intestine are significantly dose-dependent [35]; low concentrations promote intestinal mucosal integrity, whereas high concentrations can induce cell cycle arrest or apoptosis [36]. Furthermore, butyrate possesses pleiotropic functions, including anti-cancer effects [37]. For example, butyrate selectively induces apoptosis in colorectal cancer cells by activating the activator protein 1 (AP-1) signaling pathway [38], and enhances the anti-cancer effects of dietary fiber through GPR109A-mediated tumor suppression [39] (Table 1).

Table 1: Functions of SCFAs and their impact on gut microbiota

SCFAs	Main Functions	Mechanism of Action	Specific Effects on Microbiota
Acetate	(1)Participation in peripheral lipid synthesis (2)Anti-inflammation (3)Maintenance of immune homeostasis	(1)Mediates anti-inflammatory effects by binding to GPR43 (2)Influences intestinal Treg function (3)Epigenetic pathways involved in immune regulation	(1)Direct effects: Lowers intestinal pH, inhibiting the colonization of harmful bacteria such as Escherichia coli and Salmonella; promotes the proliferation of beneficial bacteria like Bifidobacterium and Lactobacillus (2)Indirect effects: Enhances immune tolerance and inhibits inflammatory responses by activating GPR43
Propionate	(1)Involved in regulating gluconeogenesis and cholesterol metabolism in the liver (2)Anti-inflammation (3)Maintenance of immune homeostasis	(1)Inhibits HDAC, promoting acetylation of the FOXP3 gene (2)High-affinity binding to GPR41 receptor (3)Synergizes with butyrate to induce Tregs	(1) Direct effects: Lowers intestinal pH, inhibiting harmful bacteria such as Escherichia coli; promotes the proliferation of beneficial bacteria like Bifidobacterium (2)Indirect effects: Regulates metabolic-immune signaling via GPR41, influencing microbiota structure
Butyrate	(1) Main energy source for intestinal epithelial cells (2)Enhances intestinal barrier function (3)Maintains epithelial homeostasis (4) Anti-inflammatory, anti-cancer	(1)Inhibits HDAC, activates PPARγ (2)Blocks NF-κB, downregulates pro-inflammatory cytokines (IL-6/IL-12), upregulates IL-10 (3)Upregulates tight junction proteins (Claudin-1, ZO-1, Occludin) (4)Activates AP-1 to promote cancer cell apoptosis; mediates anti-cancer effects via GPR109A	(1) Direct effects: Reshapes microbiota. Lowers pH to inhibit the proliferation of pathogenic Gram-negative bacteria such as Escherichia coli and Salmonella; promotes the colonization of acid-producing bacteria like Bifidobacterium and Lactobacillus (2)Indirect effects: Upregulates tight junction proteins to reduce bacterial translocation; activates PPARγ to promote defensin secretion and inhibit the overgrowth of Proteobacteria; inhibits HDAC to induce Treg differentiation, alleviating inflammatory dysbiosis

2.3 Regulation of Gut Microbiota by SCFAs

The gut microbiota and SCFAs form a bidirectional regulatory network, and their interaction mechanism is becoming a core direction in microecological medical research. On one hand, the composition and abundance of the gut microbiota directly determine the yield and types of SCFAs, as previously discussed. On the other hand, SCFAs influence the structure and function of the microbiota through various signaling pathways. First, SCFAs can directly reshape the microbiota ecology. By lowering the intestinal pH, SCFAs specifically inhibit the colonization of harmful bacteria such as Escherichia coli and Salmonella, while promoting the proliferation of beneficial bacteria like Bifidobacterium and

Lactobacillus [40]. Second, SCFAs can indirectly influence the microbiota structure through host-microbiota interactions. For instance, SCFAs promote the activity of Tregs by activating G protein-coupled receptors such as GPR43, enhancing immune tolerance and reducing autoimmune reactions [41]. Alternatively, they can affect the production of inflammatory factors by modulating the metabolites of the gut microbiota [42]. Additionally, SCFAs can simultaneously maintain multiple intestinal barrier functions [43] (Table 1).

3. Regulatory Effects of the Gut Microbiota-SCFAs Axis in AP

Gut microbiota dysbiosis is closely associated with the occurrence and severity of AP. Multiple studies have shown that the diversity of the gut microbiota is significantly reduced in both AP patients and animal models [44, 45], mainly characterized by an increase in pathogenic bacteria and a decrease in beneficial bacteria. At the phylum level, the abundance of Bacteroidetes and Proteobacteria is increased in the intestines of AP patients, while the abundance of Firmicutes and Actinobacteria is decreased; at the genus level, the main changes are characterized by an increased abundance of Enterobacteriaceae, Enterococcus, and Escherichia - Shigella, and a decreased abundance of Bifidobacterium [46]. Imbalance in the gut microbiota can lead to reduced SCFA production, triggering pathological changes such as intestinal barrier dysfunction, immune dysregulation, and metabolic disorders, thereby exacerbating the process of pancreatic injury. Furthermore, impaired intestinal mucosal defense function can lead to the translocation of microbiota and their metabolites, further triggering local or systemic inflammatory responses, promoting the formation of pancreatic necrosis and secondary severe infections, which become important driving factors for the worsening of AP [47].

Currently, experimental research on SCFAs in the treatment of AP centers on butyrate, whose protective mechanisms encompass intestinal barrier repair, local inflammation inhibition, and multi-organ system protection.

Studies have shown that butyrate treatment can alleviate intestinal mucosal injury in rats with severe acute pancreatitis (SAP), reduce epithelial cell apoptosis, and protect the intestinal barrier by upregulating the tight junction proteins ZO-1 and Occludin [48]. Oral administration of Clostridium butyricum and its main metabolite, butyrate, also improves intestinal barrier function in SAP rats. This is manifested by the upregulated expression of tight junction proteins ZO-1, Claudin-1, and Occludin; inhibition of tumor necrosis factor-α (TNF-α) and matrix metalloproteinase 9 (MMP9) activity; decreased plasma levels of diamine oxidase (DAO) and lipopolysaccharide (LPS); and reduced intestinal permeability. Concurrently, it increases the abundance of Lactobacillus while decreasing the abundance of Proteobacteria [33].

Butyrate regulates the inflammatory microenvironment through a dual mechanism involving the inhibition of pro-inflammatory signals and the activation of anti-inflammatory pathways [49]. For instance, butyrate significantly reduces the expression levels of the pro-inflammatory cytokines TNF-α, IL-6, and C-C motif chemokine ligand 2 in the pancreas and colon of AP mice, and inhibits the activation of the NOD-like receptor family pyrin domain-containing protein 3 (NLRP3) inflammasome. This mechanism involves the interaction of HDAC1, the transcription factor AP-1, and signal transducer and activator of transcription 1 (STAT1). In the pancreas, butyrate significantly increases acetylation levels at sites such as histone H3 lysine 9 (H3K9) and H3K14 by inhibiting HDAC1 activity, thereby suppressing the transcription of pro-inflammatory genes. In the colon, butyrate activates the receptor GPR109A, inhibits the phosphorylation of NF-κB and AP-1, downregulates NLRP3 inflammasome activity, and reduces serum DAO activity. Furthermore, when gut microbiota dysbiosis leads to decreased SCFA levels, it promotes excessive monocyte activation, thereby exacerbating the severity of AP. This is also a key mechanism by which carboxymethyl cellulose aggravates the condition in AP mice [50].

It is noteworthy that SCFAs not only alleviate local intestinal damage but also reduce systemic inflammation through the "gut-lung axis" and "gut-kidney axis," lowering the risk of AP complicated by acute lung injury and kidney injury [46].

4. Effects of Traditional Chinese Medicine on the Gut Microbiota-SCFAs Axis in AP

Although the specific disease name "AP" does not exist in ancient Chinese medical texts, ancient physicians, through detailed analysis of clinical symptoms, had already formed a profound understanding of its typical clinical manifestations. Using the dialectical thinking pattern of "inferring internal disorders from external observations," they constructed corresponding pathological models and diagnostic-treatment systems. Records such as "Juexin Pain, with abdominal distension and chest fullness, severe heart pain, this is Stomach Heart Pain" in Miraculous Pivot (Lingshu) – Juebin (Treatise on Reversal Syndromes), and descriptions like "Pain below the heart, hard to touch" and "Hard, full, and untouchable pain from below the heart down to the lower abdomen" in Treatise on Cold Damage (Shanghan Lun), are all related to the clinical features of AP. Based on the location and clinical characteristics of AP, modern TCM scholars classify it under the category of "Abdominal Pain" (Futong), encompassing disease names such as "Spleen Heart Pain" (Pinxintong), "Stomach Heart Pain" (Weixintong), and "Pancreatic Distention" (Yidan) [51]. Its basic pathogenesis is "blockage of fu-qi" (fuqi butong), with the disease location primarily in the spleen and closely associated with the intestines.

Targeting the core pathogenesis of AP, TCM compound formulas such as Qingyi Decoction (Qingyi Tang) and Qingyi Huoxue Granules (Qingyi Huoxue Keli), monomers like berberine, as well as electroacupuncture therapy, have all been clinically proven to have definite efficacy. Modern pharmacological research further indicates that these interventions can significantly optimize the function of the gut microbiota-SCFAs axis by regulating the intestinal microecological balance.

4.1 Qingyi Decoction (Qingyi Tang)

As a classic formula in TCM for treating AP, Qingyi

Decoction possesses the effects of "clearing heat and removing toxicity, soothing the liver and promoting gallbladder function, and unblocking the fu-organs and discharging turbidity." Modern pharmacological research has confirmed its ability to precisely regulate the gut microbiota-SCFAs axis, a mechanism that provides significant support for its role in improving SAP and associated acute lung injury [8].

First, in the upstream regulatory link of the gut microbiota-SCFAs axis, Qingyi Decoction can reverse the gut microbiota imbalance that occurs during SAP. Research shows that the abundance of pathogenic bacteria such as Escherichia and Enterococcus in the intestines of SAP mice is significantly increased. These bacteria not only disrupt the integrity of the intestinal mucosa but may also cause distal lung tissue damage through the gut-lung axis. Intervention with Qingyi Decoction can significantly reduce the relative abundance of these pathogenic bacteria, while specifically increasing the proportion of SCFA-producing bacteria such as Bacteroides and Roseburia. This optimization of the microbiota structure directly promotes the activation of the gut microbiota-SCFAs axis, highly consistent with the TCM core principle of "unblocking the fu-organs and discharging turbidity" (restoring intestinal microecological balance by clearing intestinal turbidity). Second, at the core regulatory level of the gut microbiota-SCFAs axis, Qingyi Decoction achieves a simultaneous increase in SCFAs across multiple sites by remodeling the microbiota structure. With the increased abundance of SCFA-producing bacteria, the levels of SCFAs in the feces, intestinal tissue, serum, and lung tissue of SAP mice are all significantly elevated. This cross-organ distribution pattern from the intestine to serum and then to the lung not only reflects the axis's ability to regulate the systemic pathological process of SAP but also explains why Qingyi Decoction can simultaneously ameliorate intestinal injury and associated acute lung injury in SAP. The remote transmission function of SCFAs further extends the regulatory effect of the gut microbiota to the lung tissue, creating a synergistic effect of intestinal protection and lung injury alleviation. Finally, in the downstream effector link of the gut microbiota-SCFAs axis, SCFAs further exert their effect in improving SAP by activating the AMP-activated protein kinase (AMPK)/NF-κB/NLRP3 signaling pathway. On one hand, SCFA-mediated AMPK activation inhibits the nuclear translocation of NF-κB and the activation of the NLRP3 inflammasome, significantly reducing the release of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α, thereby blocking the cascade amplification of the systemic inflammatory response in SAP. This aligns with the TCM effect of clearing heat and removing toxicity. On the other hand, SCFAs also synergistically repair the intestinal barrier through this pathway. They not only promote the expression of ZO-1 and Occludin to enhance intestinal mucosal integrity, thereby reducing bacterial and toxin translocation, but also repair the mitochondrial function of intestinal epithelial cells and promote intestinal epithelial regeneration. This consolidates the intestinal barrier both structurally and functionally, further strengthening the regulatory effect of the gut microbiota-SCFAs axis [8].

4.2 Chaihuang Qingyi Huoxue Granules (Chaihuang Qingyi Huoxue Keli)

As an effective TCM formula for treating acute pancreatitis, Chaihuang Qingyi Huoxue Granules possess comprehensive effects including purging heat and unblocking the fu-organs, clearing heat and removing toxicity, promoting qi flow and alleviating pain, activating blood and resolving stasis, and strengthening the spleen and nourishing the stomach. In improving the pathological process of SAP, these effects achieve multi-link synergy through precise regulation of the gut microbiota-SCFAs axis [52].

First, in the upstream regulatory link of the gut microbiota-SCFAs axis, Chaihuang Qingyi Huoxue Granules can reverse the intestinal microecological disturbance in SAP rats. Under SAP conditions, the relative abundance of Firmicutes and Bacteroidetes in the intestine is significantly reduced, leading to an imbalance in microbiota distribution; intervention with Chaihuang Qingyi Huoxue Granules effectively reverses this trend, restoring the ratio of these two dominant phyla to near-normal levels. Simultaneously, this formula significantly inhibits the abnormal proliferation of opportunistic pathogens such as Escherichia-Shigella and Enterobacter, reducing bacterial translocation and endotoxin release caused by these pathogenic bacteria. This reduces the risk of a secondary hit in SAP at the source, aligning with its TCM effects of purging heat and unblocking the fu-organs, and clearing heat and removing toxicity. Second, at the core regulatory link of the gut microbiota-SCFAs axis, Chaihuang Qingyi Huoxue Granules significantly enhance the production capacity of intestinal metabolites by improving the living environment for SCFA-producing bacteria. This formula specifically increases the abundance of SCFA-producing genera such as Lachnospiraceae_NK4A136_group, Ruminococcus_1, and Prevotellaceae_UCG-001. The enrichment of these bacterial groups directly promotes intestinal metabolic remodeling, leading to significantly elevated SCFA levels in the intestines of SAP rats after drug intervention, providing core material support for subsequent repair of the intestinal mucosal barrier. Finally, in the downstream effector link of the gut microbiota-SCFAs axis, Chaihuang Qingyi Huoxue Granules, mediated by SCFAs, achieve synergy between intestinal mucosal barrier repair and systemic inflammation inhibition. On one hand, SCFAs promote the upregulated expression of ZO-1 and Occludin, repairing the structural integrity of the intestinal mucosal mechanical barrier. This corresponds to the formula's effects of strengthening the spleen and nourishing the stomach, and activating blood and resolving stasis. On the other hand, the optimized microbiota and increased SCFAs collectively reduce the entry of intestinal endotoxins into the bloodstream, significantly lowering serum endotoxin levels in SAP rats. This, in turn, blocks the cascade amplification of the systemic inflammatory response and alleviates pathological damage to the pancreas and intestines [52].

4.3 Berberine

Berberine, a core active component of traditional medicinal plants such as Coptis chinensis and Phellodendron amurense, is a natural isoquinoline alkaloid. Its multi-target, multi-pathway pharmacological properties have garnered significant attention in the treatment of metabolic and inflammatory diseases. A clinical study involving 78 SAP patients confirmed that berberine can alleviate the clinical

symptoms of SAP through precise regulation of the gut microbiota-SCFAs axis [53].

First, in the upstream regulatory link of the gut microbiota-SCFAs axis concerning microbiota modulation, berberine can reshape the disturbed intestinal microecology in SAP patients. Under SAP conditions, pathogenic bacteria such as Escherichia coli overproliferate in the intestine, while the abundance of SCFA-producing probiotics like Bifidobacterium decreases, disrupting the microbial balance. Following berberine intervention, this imbalance is significantly reversed. Not only is the count of the pathogenic bacterium E. coli substantially reduced, with a decrease significantly greater than that in the conventional treatment group, but the count of the probiotic Bifidobacterium is also significantly increased, with an increase far exceeding that of the conventional treatment group. This ultimately leads to a marked improvement in the Bifidobacterium/E. coli (B/E) ratio. This precise modulation, inhibiting pathogens while promoting beneficial bacteria, lays a solid microbial foundation for the activation of the gut microbiota-SCFAs axis, reducing the risk of bacterial translocation and LPS release at the source. Second, at the core regulatory link of the gut microbiota-SCFAs axis, berberine significantly enhances the production and accumulation of SCFAs by optimizing the microbiota structure. Clinical data show that the levels of major SCFAs, including acetate, propionate, and butyrate, in the feces of patients in the berberine intervention group are significantly increased and are markedly higher than those in the conventional treatment group. Finally, in the downstream effector link of the gut microbiota-SCFAs axis, SCFAs translate the microbiota regulatory effects into downstream biological effects. The study confirmed that serum concentrations of inflammatory cytokines in patients from the berberine intervention group were significantly lower than those in the conventional treatment group, a change directly associated with elevated SCFA levels. It is hypothesized that SCFAs may block the cascade amplification of the systemic inflammatory response in SAP by inhibiting the activation of inflammatory signaling pathways, while also reducing intestinal mucosal permeability and decreasing endotoxemia [53].

The results of this clinical study fully demonstrate that berberine, by inhibiting the proliferation of pathogenic bacteria, elevating SCFA levels, and mediating anti-inflammatory effects and intestinal barrier protection, acts as an effective active TCM ingredient for improving SAP via the gut microbiota-SCFAs axis. This provides new ideas and evidence support for the clinical treatment of SAP. However, direct evidence from animal experiments on berberine's intervention effect on the gut microbiota-SCFAs axis in AP treatment is still lacking.

4.4 Electroacupuncture Therapy

In the intervention system of TCM for treating AP, pharmacological therapy is not the only approach. Clinical strategies are progressively evolving towards a diversified model of "drug-non-drug synergistic regulation." Among these, electroacupuncture (EA) at the Zusanli (ST36) acupoint, as a classic non-pharmacological therapy, has its mechanism of action in treating AP elucidated through the precise regulation of the gut microbiota-SCFAs axis [54].

First, in the upstream regulatory link of the gut microbiota-SCFAs axis concerning microbiota modulation, EA can specifically reshape the disturbed intestinal microecology in SAP rats. Under SAP conditions, the abundance balance between Firmicutes and Bacteroidetes is disrupted, and pathogenic bacteria overproliferate. EA intervention effectively reverses this trend, not only restoring the relative abundance of Firmicutes and Bacteroidetes and significantly optimizing the Firmicutes/Bacteroidetes (F/B) ratio compared to the model group, but also inhibiting the proliferation of pathogenic bacteria and reducing their risk of invading the intestinal mucosa. This creates conditions for the activation of the gut microbiota-SCFAs axis at the source. Second, at the core regulatory link of the gut microbiota-SCFAs axis, EA significantly enhances the production and accumulation of SCFAs by optimizing the microbiota structure. Following EA intervention, the levels of major SCFAs, such as acetate and butyrate, in the intestinal contents of SAP rats are significantly elevated. Notably, the concentration of butyrate shows a marked upward trend compared to the model group. The enrichment of butyrate is a core indicator of the functional activation of the gut microbiota-SCFAs axis. Correlation analysis of microbial metabolism further confirmed that the abundance of SCFA-producing bacteria, specifically *Lachnospiraceae_UCG-006* and Romboutsia, was significantly positively correlated with butyrate levels, clarifying the regulatory logic that EA elevates SCFA levels by promoting the colonization of acid-producing bacteria. Finally, in the downstream synergistic effect link of the gut microbiota-SCFAs axis, EA achieves the dual effects of intestinal barrier repair and systemic inflammation inhibition. On one hand, the regulatory effects mediated by SCFAs drive the upregulated expression of ZO-1 and Occludin, repairing the structural integrity of the intestinal mucosal mechanical barrier. This leads to a significant reduction in serum endotoxin levels compared to the model group, diminishing the secondary hit caused by bacterial translocation. On the other hand, EA further regulates the inflammatory response through this axis, both by reducing the release of pro-inflammatory cytokines such as TNF-α and IL-6 to block the systemic inflammatory cascade, and by increasing the expression of the antimicrobial peptide LL-37 to strengthen the immune defense function of the intestinal mucosa. These actions ultimately synergistically alleviate the pathological progression of SAP.

In summary, within the research field of TCM intervention in AP, it has been confirmed that various approaches—including the TCM compound formulas Qingyi Decoction and Chaihuang Qingyi Huoxue Granules, the monomer berberine, and the non-pharmacological therapy of electroacupuncture — exert therapeutic effects through the precise regulation of the gut microbiota-SCFAs axis. These therapeutic methods all share a common pathway: starting with microbiota remodeling as the initial step, having the elevation of SCFA levels as the core factor, and achieving subsequent effects such as repairing the intestinal mucosal barrier and inhibiting inflammatory responses. This forms a complete chain of evidence connecting mechanism to efficacy. The aforementioned studies not only provide modern molecular

mechanistic support for the effects of classic TCM formulas, monomeric components, and non-pharmacological therapies, revealing previously unexplained mechanisms of the "multi-component, multi-target" actions of TCM, but also experimentally confirm the core value of the gut microbiota-SCFAs axis in TCM treatment of AP. This lays a theoretical foundation for the subsequent development of AP treatment strategies targeting the gut microbiota.

Beyond the aforementioned therapeutic methods that explicitly involve the regulation of the gut microbiota-SCFAs axis, there are more monomeric components and compound formulas in the field of TCM that have demonstrated potential in regulating gut microbiota and restoring intestinal function in the treatment of AP. Anthraquinones from rhubarb (such as emodin, rhein, and aloe-emodin) can regulate the balance of gut microbiota by upregulating the abundance of probiotics like Lactobacillus and Bifidobacterium, downregulating the proliferation of opportunistic pathogens like Escherichia coli, inhibiting intestinal bacterial translocation, and ameliorating abnormalities in short-chain fatty acid metabolism. This, in turn, protects the integrity of the intestinal mucosal barrier, reduces inflammatory damage to the pancreas and surrounding tissues, and lowers the likelihood of acute pancreatitis progressing to a severe state [55]. In the realm of TCM compound formulas, Dachengqi Decoction (Dachengqi Tang), a representative prescription of the "purgation method" (tongli gongxia), can promote intestinal microecological balance and restore gastrointestinal function by reducing the relative abundance of Escherichia-Shigella and Erysipelatoclostridium, while increasing the relative abundance of beneficial genera such as Lactobacillus, Romboutsia, and Blautia. This improves intestinal barrier function, creating favorable conditions for the recovery of intestinal function in AP patients [56]. Enema treatment with Qingchang Xiaodan Fang (Qingchang Xiaodan Fang) for SAP can more effectively improve the diversity of the patients' gut microbiota and maintain intestinal microecological balance, as evidenced by significant increases in the Chao index and Shannon index of the gut microbiota [57] It is noteworthy that although the efficacy of these TCM monomers and compound formulas in regulating gut microbiota and restoring intestinal function has been experimentally validated, research on their mechanisms of action still requires further improvement. Existing studies have largely focused on changes in gut microbiota composition (such as microbial abundance and diversity indices) and improvements in intestinal function indicators (such as intestinal motility rate and intestinal permeability). They have not yet directly analyzed the changes in SCFA levels in intestinal contents, serum, or feces during the treatment process. This limitation leaves two key questions unanswered: first, whether these TCM interventions, after regulating the gut microbiota, can promote the synthesis and release of SCFAs similar to Qingyi Decoction and berberine; and second, whether their therapeutic effects on AP (such as alleviating inflammation and repairing the intestinal mucosa) can be attributed to SCFAs as a core mediator. Therefore, these results are currently insufficient to construct a complete causal chain of "TCM intervention – gut microbiota regulation – SCFA changes – therapeutic effect in AP." To ensure the rigor and sufficiency of evidence for the conclusions of this paper, the aforementioned TCM monomers and compound formulas are temporarily not included in the core discussion regarding the "gut microbiota-SCFAs axis." Future research needs to design more precise experimental protocols to detect the dynamic changes of SCFAs following relevant TCM interventions, clarify their association with therapeutic effects, and further refine the molecular mechanism network of TCM in treating AP.

Table 2: Study on the therapeutic effect of traditional Chinese medicine on AP by regulating the gut microbiota-SCFAs axis

TCM Therapy	Experimental Model	Gut Microbiota	SCFAs Content	Pathological Damage	Mechanism of Action
Qingyi Decoction [8] (Rhubarb, Bupleurum, Costus Root, White Peony Root, Glauber's Salt, Corydalis Rhizome, Gardenia, Scutellaria)	C57BL/6 mice (SAP induced by retrograde infusion of 3.5% sodium taurocholate into the biliopancreatic duct)	Pathogenic bacteria: Escherichia coli ↓, Enterococcus ↓ SCFA-producing bacteria: Bacteroides ↑, Roseburia ↑, Akkermansia ↑	Acetate ↑ Propionate ↑ Butyrate ↑	Pancreatic and ileal pathological scores ↓	Anti-inflammatory effects: Serum α-amylase ↓, IL-1β ↓, IL-6 ↓, TNF-α ↓ Intestinal barrier: ZO-1 ↑, Occludin ↑, Serum D-lactate ↓, Serum LPS ↓, Serum DAO ↓ Molecular pathway: pAMPK ↑, NF-κB ↓, NLRP3
Chaihuang Qingyi Huoxue Granules [52] (Bupleurum, Magnolia Bark, Red Peony Root, Rhubarb, Peach Kernel, Salvia Root, Licorice, Corydalis Rhizome, Astragalus, Scutellaria, Immature Bitter Orange, Gardenia, White Peony Root, Dandelion)	Sprague-Dawley rats (SAP induced by retrograde infusion of 3.5% sodium taurocholate into the biliopancreatic duct)	Pathogenic bacteria: Proteobacteria ↓, Escherichia-Shigella ↓, Enterococcus ↓, Enterobacter ↓ SCFA-producing bacteria: Firmicutes ↑, Bacteroides Ruminococcus ↑, Lachnospiraceae_NK4A136_group ↑, Parabacteroides ↑, Lactobacillus ↑, Prevotellaceae_UCG-001↑	Acetate ↑ Propionate ↑ Butyrate ↑	Pancreatic and colonic、 pathological scores ↓	Anti-inflammatory effects: Serum amylase ↓, Serum lipase ↓, Serum LPS ↓ Intestinal barrier: ZO-1 ↑, Occludin ↑
Berberine [53]	SAP patients	Pathogenic bacteria: Escherichia coli ↓ SCFA-producing bacteria: Bifidobacterium ↑	Acetate ↑ Propionate ↑ Butyrate ↑		Anti-inflammatory effects: Serum IL-6 ↓, IL-8 ↓, TNF-α ↓
Electroacupuncture at Zusanli (ST36) [54]	Sprague-Dawley rats (SAP induced by intraperitoneal injection of L-ornithine)	Pathogenic bacteria: Ruminococcus ↓, Muribaculaceae ↓ SCFA-producing bacteria: Firmicutes ↑, Lactobacillus ↑, Bifidobacterium ↑, *Lachnospiraceae_UCG-006* ↑, Romboutsia ↑	Acetate ↑ Propionate ↑ Butyrate ↑	Pancreatic and intestinal pathological scores ↓	Anti-inflammatory effects: Serum amylase ↓, lipase ↓, TNF-α ↓, IL-6 ↓ Intestinal barrier: Occludin ↑, ZO-1 ↑, antimicrobial peptide LL-37 ↑

5. Summary

In summary, the core regulatory role of the gut microbiota-SCFAs axis in AP has been clearly confirmed. SCFAs, through mechanisms such as regulating immune homeostasis, strengthening intestinal barrier function, and inhibiting inflammatory cascades, have emerged as key protective mediators in the pathological process of AP. Traditional Chinese medicine, leveraging its holistic regulatory characteristics involving multiple components and multiple targets, demonstrates unique advantages in modulating the gut microbiota-SCFAs axis, thereby providing diversified intervention strategies for AP treatment (Table 2).

However, current research still has significant limitations: First, the mechanistic explanations often remain at the macro-level correlation between microbiota, SCFAs, and phenotype, lacking molecular-level resolution from TCM components to microbial metabolic enzymes, SCFA synthesis, and host targets. Second, intervention protocols lack standardized criteria, and the impact of interindividual variability in the gut microbiota on therapeutic efficacy has not been adequately addressed. Third, evidence for clinical translation is weak, mostly consisting of animal experiments or small-sample clinical observations, lacking validation data from large-sample, long-term follow-up studies, and lacking an efficacy prediction and evaluation system based on the gut microbiota-SCFAs axis.

In view of this, future research should: (1) utilize multi-omics technologies to deeply explore the molecular mechanisms by which TCM regulates the gut microbiota-SCFAs axis, clarifying the specific roles of key bacterial strains, SCFA subtypes, and downstream signaling pathways; (2) promote research that integrates standardization with individualization, establishing precise TCM intervention protocols based on gut microbiota typing; (3) conduct large-sample clinical studies to verify its efficacy and safety in AP patients; and (4) strengthen translational applications by developing TCM preparations targeting SCFA-producing bacteria, providing a solid theoretical foundation and clinical basis for TCM's targeted regulation of the intestinal microecology in treating AP. Furthermore, the TCM principle of treatment based on syndrome differentiation aligns closely with the individualized regulatory characteristics of the intestinal microecology. The profound integration of these two aspects may represent a new breakthrough in the precision treatment of AP.

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