Treatment with mannose oligosaccharides reverses the intestinal injury in the acetylsalicylic acid-treated rat model

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Abstract

Mannose oligosaccharide (MOS) has been shown to promote animal growth, maintain intestinal health, and activate the intestinal immune system. However, the question of whether MOS can stimulate the immune system and alleviate acetylsalicylic acid (ASA)-induced gut damage remains unresolved. The purpose of this study was to investigate the impact of MOS pretreatment on the immunological and anti-inflammatory capabilities of rats with ASA-induced intestinal injury. Thirty-six male Sprague-Dawley rats were divided into 6 groups and were fed with 0 (negative control), 100, 300, 600, and 800 mg/kg·Body weight (BW) of MOS for 3 weeks. From day 8, rats were fed with 200 mg/kg BW of ASA for 14 days to induce intestinal injury. The growth performance, viscera index, serum and intestinal immunity, intestinal inflammation and morphology of ASA-induced intestinal injury rats with or without MOS administration were investigated. In MOS deficient rats, oral treatment of ASA causes severe intestine damage and immunological dysfunction. In a rat model, 600 mg/kg BW MOS can lower the expression of inflammatory markers and effectively increase liver index, serum interleukin-2 (IL-2), lysozyme contents, intestinal secretory immunoglobulin A (sIgA) and mucus volume, intestinal villus height, crypt depth and villus height/crypt depth in comparison to the ASA group. These results imply that providing rats with MOS at the appropriate dosage can significantly improve their immune system and successfully shield the intestines from ASA damage. MOS is therefore expected to be a promising gut immunopotentiator for enhancing intestinal health in animals.

Keywords: mannose oligosaccharide, acetylsalicylic acid, intestinal injury, serum immunity
**Introduction**

Changes in temperature, environment, and diet can cause stress in animals, and in some extreme cases, intestinal dysfunction, commonly manifested as diarrhea, results in decreased food intake and increased mortality (De Oliveira et al. 2019, Pluske et al. 2019). As a result, mitigating intestinal damage serves as an effective means to enhance nutrient absorption and safeguard the welfare of animal husbandry (Chen et al. 2021). The gut is considered the body’s largest immune organ and plays a crucial role in digesting and nutrient absorption (Elderman et al. 2017). A healthy digestive system has an intact intestinal villi structure, which may improve the body’s capacity to digest and absorb nutrients (Landin et al. 2012). In addition, as the initial line of defense against foreign substances, gut mucosal immunity can assist the animal’s immune system (Zou et al. 2021). Intestinal injuries and disorders can be brought on by modifications to the breeding habitat, the diet that the animals are provided, and the conditions of transit. Inflammation and abnormalities in the digestive tract follow, resulting in diarrhea, a slower metabolic rate, stunted growth, and eventually death (Yi et al. 2017, Chen et al. 2021).

The establishment of a successful intestinal injury model is the base for investigating intestinal problems in the animal model (Cipriani et al. 2013, Lai et al. 2015). Acetylsalicylic acid (ASA) is a non-steroidal anti-inflammatory drug (NSAID) that can harm the mucosa of the stomach, causing erosion and ulcers (Sugimoto et al. 2000). In the meanwhile, ASA primarily damages the stomachs, duodenum, and posterior intestinal tract of animals, as well as the intestinal mucosa, intestinal tight junctions, and immune system. It is an effective drug for producing gastrointestinal inflammatory injury (Suyama et al. 2018, Mortensen et al. 2019). In our previous study (Chen et al. 2020), we demonstrated that giving 200 mg/kg Body weight (BW) ASA for 14 consecutive days can successfully induce intestinal injury model in the Sprague Dawley (SD) rat model. We found the body weight gain, liver index, serum interleukin-2 (IL-2) content and lysozyme (LZM) activity, intestinal mucosa secretory immunoglobulin A (sIgA) content, intestinal villus height (VH), crypt depth (CD) and their ratio were all decreased significantly. According to recent research, intestinal immune malfunction may contribute to the emergence of NSAIDS-induced intestinal mucosal injury (Kim et al. 2019, Watanabe et al. 2020, Cai et al. 2021). Consequently, one way to treat intestinal inflammation is to find natural additives that can strengthen the immune system of the body and maintain intestinal health without having harmful side effects.

Mannose oligosaccharide (MOS) synthesized from the cell wall of *Saccharomyces cerevisiae* has been shown to promote the proliferation of beneficial bacteria in the intestine, increase animal food intake, and improve growth performance (Halas and Nochta, 2012, Cheng et al. 2019, Mohammad et al. 2019). In addition, MOS also has immunological features such as phagocytosis, superoxide anion generation, and lysozyme activity (Mohammad et al. 2019). It has been extensively utilized in food, medicine, and animal nutrition because of its ability to activate B lymphocytes and natural killer cells (Sun et al. 2019). Other studies found that MOS supplementation significantly improved broiler intestinal morphology, and increased the VH and CD ratios in young broilers (Iji et al. 2001) and weaned piglets (Shen et al. 2009). Mannan compounds can directly enhance the immune system of pigs, stimulate the intestinal mucosa locally, raise IgA secretion in different parts of the mucosa, and consequently initiate the host’s systemic immune response (Halas and Nochta 2012). Davies et al. (2004) discovered that supplementation of phosphomannan in a dose of 2 g/kg to the diet for 21 days altered the amount of jejunum intrinsic T cells. MOS, in its capacity as a functional oligosaccharide, being regarded as a potential substitute immunopotentiator for enhancing the intestinal health, growth, and development of animals (Hutsko et al. 2016, Yan et al. 2019, Wang et al. 2021).

MOS may enhance intestinal health, promote intestinal growth, and stimulate the intestinal immune system, according to these results. Nonetheless, the potential of immune system stimulation to mitigate the intestinal harm induced by ASA remains unknown. The present investigation examined the impact of MOS pretreatment on the anti-inflammatory and immunological capacities of rodents that had intestinal damage induced by ASA. Our findings may pave the way for a novel method of enhancing the intestinal health of animals.

**Materials and Methods**

**Chemicals**

MOS (PW120) was obtained from Angel Yeast Co. Ltd. (Yichang, China), while ASA was acquired from Henan Fengmu Biotechnology Co. Ltd (Zhengzhou, China).

The ELISA kit for LZM, IL-2, and sIgA was purchased from Jiancheng Biologic Project Company (Nanjing, China). The RNA Isolation kit was procured from Shengong Bio Inc. (Shanghai, China). The Reverse Transcriptase kit and SYBR qPCR kit were obtained from Takara Bio Inc. (Shiga, Japan).
The qTOWER 3G real-time PCR system were obtained from Analytik Jena (Thuringia, Germany). All the other chemicals were analytical grade.

**Experimental animals**

36 six-week-old male Sprague-Dawley (SD) rats weighing 150-180 g were obtained from Henan Experimental Animal Center (Henan, China). The rats in the present study were taken care of in complete compliance with the National Institutes of Health’s guidelines for the Care and Use of Laboratory Animals (NIH, 1996). The protocols were accepted by the animal care review committee of the Henan University of Technology with the approval number HAUTETHI-2017-0485.

**Experimental design and feeding condition**

36 healthy SPF grade SD male rats (body weight: 150-180 g) were randomly assigned to one of six groups (n=6 per group): Control group, ASA group, 100 mg/kg BW MOS group (MOS 100 group), 300 mg/kg BW MOS group (MOS 300 group), 600 mg/kg BW MOS group (MOS 600 group) and 800 mg/kg BW MOS group (MOS 800 group). The rats were allowed to eat and drink freely in a temperature range of 20-25°C and a humidity range of 40-60%. After 1 week of adaptation, the experiment lasted for 3 weeks. The food intake and body weight of the rats were recorded daily. The control group was given normal saline intragastrically for 21 days. The rats in the MOS and ASA groups received varied dosages of MOS and normal saline intragastrically for 7 days as their assigned group dosage. An intragastric injection of 200 mg/kg BW ASA was administered for 14 days. On the 22nd day, the rats were fasted but had unrestricted access to water. On the 23rd day, the rats were sedated with ether after being weighed, and blood was collected from their eyeballs. The rats were dissected after being euthanized via neck dislocation. Rat spleens, livers, and thymuses were dissected, cleansed in sterile normal saline, dried, and weighed. After separating the intestines, jejunum tissue was taken for immunological index examination. A portion of the intestinal tissue was kept in 10% formalin buffer for paraffin sectioning, which utilized to examine intestinal morphology and assess VH and CD.

**Growth performance and viscera index**

The body weight gain (BWG), average daily gain (ADG), average daily feed intake (ADEF), feed conversion rate (FCR), food consumption, and body weight were recorded at the beginning and end of the study to assess the rat’s overall growth. The liver, spleen, and thymus were dissected at the end of the experiment, and the carcass was cleaned and dried with normal saline before being weighed and recorded using the equation below:

\[
\text{spleen index (\%)} = \left( \frac{\text{spleen mass (g)}}{\text{body mass (g)}} \right) \times 100
\]

\[
\text{liver index (\%)} = \left( \frac{\text{liver mass (g)}}{\text{body mass (g)}} \right) \times 100
\]

\[
\text{thymus index (\%)} = \left( \frac{\text{thymus mass (g)}}{\text{body mass (g)}} \right) \times 100
\]

**Immunity ability**

**Serum immunoassay**

Rat blood was coagulated at room temperature and centrifuged at 2000 rpm for 20 min to prepare the serum. After that, the serum was carefully extracted and the levels of LZM and IL-2 were determined using an ELISA kit (Jiancheng Biologic Project Company, Nanjing, China) following the manufacturer’s instructions.

**Determination of slgA content**

Jejunum tissue (5 cm) near the duodenum was sliced open on the gauze, and the intestinal mucosa was scraped with glass slides and placed in a centrifuge tube, which was stored at -80 °C. The presence of slgA in the intestinal mucosa was determined using the ELISA kit (Jiancheng Biologic Project Company, Nanjing, China) in accordance with the manufacturer’s instructions.

**Determination of intestinal mucus**

The method to determine the intestinal mucus volume was following the report of Suyama et al. (2018). In short, a portion (approximately 5 cm in length) of the intestine (10 cm from the pylorus) was chosen for homogenization, and 1 mL phosphate buffer solution (PBS) was added to form a suspension. Centrifuge at 15000 rpm for 15 minutes at 4°C. After incubating with periodate (0.1%, 100 µL) at 37°C for 2 hours, 100 µL Schiff reagent was added and incubated at room temperature for 30 minutes. The absorbance (OD) value, representing the amount of mucus, was determined at 555 nm utilizing the Spark 10M Multimode Microplate Reader (TECAN, Mannedorf, Switzerland).

**Intestinal morphology**

The tissue from rats’ jejunum tissue was removed after dissection, fixed for 24 h in 4% paraformaldehyde, dehydrated using a concentration gradient of ethanol, translucid and embedded in paraffin. It was then cut into 5 µm thin slices using a microtome, stained with hematoxylin and eosin (HE), dehydrated, sealed, and exa-
mined under a microscope to observe the histopathology. The morphology of intestinal villi in jejunum tissue from each group was observed and analyzed using an inverted light microscope (Echo Laboratories, CA, USA). In addition, the VH and CD were measured, and the ratio of VH/CD was calculated.

**qRT-PCR analysis**

An animal total RNA Isolation kit (Shenggong, Shanghai) was used to extract RNA from jejunum tissue following the manufacturer’s instructions. The Reverse Transcriptase Kit (Takara, Japan) was utilized for cDNA synthesis, and the temperature settings were 15 min at 42℃, followed by 5 min at 95℃ for deactivation.

On a qTOWER 3G real-time PCR system (Analytik Jena, Germany), the SYBR qPCR Kit (Takara, Japan) was used to run qRT-PCR under the following conditions: 94℃ for 30 s, followed by 30 cycles of 72℃ for 60 s, followed by 60℃ for 30 s. Table 1 lists the PCR primer sets for gene identification. Using the $2^{-\Delta\Delta CT}$ approach, the relative mRNA expression levels of proinflammatory cytokine genes (TNF-α, IL-6, and IL-1β) were adjusted based on the β-actin expression.

**Statistical analysis**

The data were presented as mean ± SEM, and the statistical analysis was carried out using SAS 9.1 statistical software and one-way ANOVA. The significant difference was defined as $p<0.05$.

**Results**

**Effects of MOS on the growth performance of rats**

Table 2 shows the daily gain and feed conversion rate (FCR) of rats in all groups. The daily gain and FCR in the ASA group were 15.61% and 15% lower than in the control group, respectively. The groups administered with MOS at doses of 100, 300, 600, and 800 mg/kg BW showed better growth performance compared to the ASA group, with daily increases of 13.40%, 15.66%, 11.89%, and 15.09%, respectively. The feed conversion efficiency rose by 11.76% in all groups compared to the ASA group.

**Effects of MOS on viscera index of rats**

Table 3 presents that the thymus index and spleen index in the ASA group were lower than in the control group, decreasing by 11.76 % and 13.04 %, respectively ($p>0.05$). Organ indicators in the MOS groups showed improvement to different extents as compared to the ASA group. The liver index of the MOS 600 and MOS 800 groups showed a substantial increase compared to the ASA group ($p<0.05$), with increments of 17.03% and 27.30%, respectively. Furthermore, there was no
Treatment with mannose oligosaccharides reverses the intestinal injury ... significant difference in the organ index between the MOS group and the control group.

Effects of MOS on inflammatory cytokine IL-2 and antimicrobial enzyme lysozyme

Table 4 shows that the serum IL-2 and LZM content of rats in the ASA group decreased by 21.76% and 28.6%, respectively, as compared to the control group. The serum IL-2 concentration in MOS groups from 300 to, 600 and 800 mg/kg BW was increased by 42.61%, 35.51%, and 21.90% (p<0.05), respectively, as compared to the ASA group. The serum LZM content in MOS groups from 600 to 800 mg/kg BW increased by 31.64% and 43.01%, respectively (p<0.05). Meanwhile, levels of serum IL-2 and LZM levels were consistent between the two highest dose groups of MOS 600 and MOS 800, suggesting that administering 600 and 800 mg/kg BW MOS orally may provide the maximum protective effect in our animal model.

Effects of MOS on intestinal sIgA levels and mucus volume in rats

Table 5 shows that the intestinal mucosal sIgA content in MOS groups from 100 to, 300, 600, and 800 mg/kg BW was increased by 21.75%, 24.55%, 100.34% and 162.82%, respectively. In the meantime, sIgA levels were considerably greater in the MOS 600 and 800 groups compared to the ASA and control groups (p<0.05). Furthermore, MOS
300, 600, and 800 considerably raised intestinal mucosal volume by 21.25%, 21.13%, and 16.53% (p<0.05) in comparison to the ASA group.

Effects of MOS on intestinal morphology of rats

Figure 1 depicts a microscopic image of the morphological structure of intestinal villi in rats. From the figure, it is evident that the intestinal villi in the blank control group exhibit an intact and well-defined structure. In contrast, the ASA group displays disrupted villi morphology with obvious signs of inflammatory damage. However, with MOS treatment, significant improvements in both villus morphology and inflammatory status are observed. As shown in Table 6, the intestinal VH of MOS groups from 100, 300, 600, and 800 increased by 14.07%, 32.99%, 46.45%, and 57.60% compared to the ASA group, respectively. Intestinal mucosal CD increased by 16.04%, 24.89%, and 32.44% in the MOS 300, 600, and 800 groups, respectively, compared to the ASA group. Similar to the intestinal VH/CD ratio, which was 17.63%, 15.84%, 17.59%, and 16.86% greater than the ASA group, the VH/CD ratio in the MOS group was also higher.

Effects of MOS on inflammatory cytokine gene expression in the intestine

Our data show that TNF-α, IL-6, and IL-1β mRNA expression levels were considerably higher following ASA treatment compared to the control group (p<0.05) (Fig. 2), suggesting that ASA may activate the upstream of an inflammatory pathway. Additionally, a decrease in the levels of TNF-α, IL-6, and IL-1β mRNA expression
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was observed with an increase in MOS levels, suggesting that MOS can mitigate intestinal inflammation by obstructing the inflammatory pathway. When comparing the MOS group’s mRNA expression levels of TNF-α, IL-6, and IL-1β to the ASA group’s vehicle control at 600 mg/kg, there was a significant down regulation of 44.7%, 38.6%, and 46.2% (p<0.05), respectively. Furthermore, when MOS supplementation levels of 600 mg/kg were compared to MOS supplementation levels of 800 mg/kg, there was no further reduction in mRNA expression levels of those inflammatory markers.

Discussion

No studies have been reported on the effects of MOS on ASA-induced enteritis, despite research on its effects on constipation, obesity, and growth performance in rats. Our results suggest that mannan-oligosaccharide can protect the intestinal barrier and potentially prevent enteritis.

MOS supplementation may partially promote growth in rats with ASA-induced enteritis, as mentioned before. Our findings align with those reported by Jahanian and Ashnagar (2015), who observed no significant impact of mannan supplementation on the performance of laying hens. Additionally, studies indicate that supplementing the diet of Caspian trout (Salmo trutta caspius) with oligosaccharides alone or in combination with other prebiotics has minimal impact on growth performance. However, a combination of oligosaccharides and probiotics can notably enhance feed conversion rate and daily gain (Mohammad et al. 2019). Therefore, MOS supplementation may not have effectively enhanced rodent growth since ASA medication damaged the intestinal barrier, impacted the intestinal flora, and decreased beneficial bacteria. Furthermore, short-term MOS administration did not restore normal probiotic levels, which impacted MOS metabolism in the colon. In future research, it is crucial to extend the duration of MOS treatment and explore the interaction between MOS and the intestinal flora. This approach will facilitate a deeper comprehension of MOS’s mechanism in mitigating intestinal injury among rats.

The thymus, spleen and liver are significant immune organs, and their indexes can be utilized to evaluate the overall immune health of the body (Attia et al. 2017). The thymus is a primary immunological organ that generates T lymphocytes and secretes thymic hormones (Thapa and Farber. 2019). The spleen is a peripheral immunological organ that serves as the hub for both humoral and cellular immunity (Li et al. 2019). The liver is a vital metabolic and immune organ in the human body, performing a variety of functions including synthesis, metabolism, excretion, detoxification, and immunity (Tarasenko and McGuire. 2017). The immunological organ index correlates positively with the body’s immune state. According to a study performed by Veronika et al. (2012), the MOS can improve disease resistance and stress resistance by increasing...
blood glucose transportation into immune organs and assisting immune function maturation, promoting plas- 
ma cell production, lengthening immunological memo- 
ry time, and significantly improving disease resistance 
and stress resistance. The study found that supplement-
ating with MOS at a dosage of 600-800 mg/kg BW 
proved the spleen and thymus index and significantly 
enhanced the viscosa index in rats with ASA-induced 
enteritis. The results were comparable to those of Wu 
(2018) who fed growing rabbits with konjac mannan 
oligosaccharides. An increase in the immune organ 
index suggests an enhancement in the body’s immuno-
logical activity. Various factors such as different forms 
of mannan oligosaccharides, dietary composition, ani-
mal species, developmental stage, and feeding condi-
tions might affect the growth and development of ani-
mal immune organs (Chen et al. 2021).

MOS has an immune-enhancing function and 
can boost the body’s immunity (Attia et al. 2017). 
IL-2 plays a crucial role as an inflammatory cytokine 
in the immune system’s response to combat microbial 
infections. It can initiate a series of immune responses 
by stimulating T cells and inducing the release of T cells 
associated cytokines such as TNF and IFN-γ (Ma et al. 
2021). LZM has antibacterial activity by efficiently 
hydrolyzing the mucopolysaccharide of pathogenic 
bacteria and breaking down bacterial cell walls to create 
peptioglycan (Saito et al. 2019). Our results were simi-
lar to a previous report by Vlies et al. (2012), who 
demonstrated that indigestible oligosaccharides can 
efficiently drive T cells to perform immunological func-
tions, significantly increasing the level of IL-2 in the 
blood, and boosting immune response. Meanwhile, 
Geraylou et al. (2012) discovered that an appropriate 
amount of oligosaccharide can raise the concentration 
of LZM in serum, and increased LZM activity can mini-
mize the degree of damage caused to the body by inflam-
matory agents.

Intestinal mucus serves as the body’s first line 
of defense against pathogens, successfully blocking 
the pathogen’s invasion and enhancing the intestine’s 
immune system by secreting the immunological protein 
sIgA. Following intestinal mucosal injury caused by 
5-fluorouracil, the animals had weight-loss, exfoliation 
of their intestinal villus, aggregation of inflammatory 
cells, and a significant drop in blood sIgA levels (Wang 
et al. 2019). In addition, increased intestine sIgA secre-
tion could improve the antioxidant and immunological 
capacities of piglets (Zhu et al. 2017). Our results 
showed that in rats with ASA-induced enteritis, intesti-
nal mucus volume, and sIgA level were significantly 
increased by MOS administration. MOS can reverse 
the decrease of sIgA content and mucus volume induced 
by intestinal mucosal injury, as well as reduce intestinal 
damage induced by bacteria, antigens, and other sources 
(Chen et al. 2021). In the meanwhile, MOS can increase 
disease resistance and decrease infection rates by limit-
ting the rate at which potentially harmful microbes 
attach to intestinal epithelial cells (Torrecillas et al. 
2011, Zhu et al. 2014). MOS could also contribute to an 
increase in the number of acidic mucus-secreting cells 
per unit area of the intestinal mucosa and the number of 
eosinophils and LZM activity (Torrecillas et al. 
2011), which increased harmful pathogenic bacteria 
breakdown and elimination from the body. As a result, 
increasing mucus secretion on the mucosal surface 
of the host is an effective way to prevent pathogenic 
bacteria colonization and destroy pathogenic bacteria 
(Zhu et al. 2014).

Animal growth performance is strongly associated 
with the structure of the intestinal morphology. Intact 
intestinal structure and functional integrity are crucial 
for facilitating the body’s digestion and absorption 
of nutrients (Tiwari et al. 2020). Our results showed that 
the intestinal VH of MOS groups from 100, 300, 600, 
and 800 increased significantly compared to the ASA 
group. Increased villus height suggests an increase 
in the contact area between the digestive tract and 
nutrients, as well as an increase in the ratio of intestinal 
villus height to crypt depth, implying improved intesti-
nal digestion and absorption capability (Shalaei et al. 
2014, Mohammadsadeghi et al. 2019). Furthermore, 
the study demonstrated that including mannan oligosac-
charides enhanced the intestinal morphology of rats 
with intestinal injury and significantly increased the 
VH/CD ratio. Supplementing rats with 600 and 
800 mg/kg BW MOS can restore the intestinal villus 
morphology to normal levels, suggesting an enhanced 
digestion and absorption ability in the intestinal 
system. This was consistent with the findings of 
Mourao et al. (2006), who showed that both intestinal 
VH and VH/CD ratios were significantly changed, even 
though there was no effect on weight gain and daily 
food consumption in the fat rabbits. The positive 
impacts of MOS on intestinal structure are mainly 
linked to controlling the resident microorganisms, 
decreasing pathogen presence and its harmful effects 
on intestinal cells, and promoting the growth of benefi-
cial bacteria (Agazzi et al. 2020). However, some inves-
tigations found that intestinal morphology was unaf-
fected by MOS therapy; the reasons for this could be 
associated with the MOS supply, the type of animal 
used, or the research design (Giannenas et al. 2016, 
Anjos et al. 2019).

There is a direct correlation between inflammatory 
biomarkers and the occurrence of enteritis. Activated 
immune cells like monocytes and macrophages secrete 
inflammatory cytokines, initiating a series of inflamma-
tory reactions (Yu et al. 2017). Local tissues and blood vessels release a high number of pro-inflammatory substances, including TNF-α, IL-1, and IL-6, during the initiation and progression of enteritis (Yu et al. 2017). An imbalance of anti-inflammatory mediators can contribute to disease progression and irreversible consequences by supporting chronic inflammatory responses. Inflammatory cytokines such as TNF-α, IL-1, and IL-6 can lead to an increase in vascular dilatation and permeability. The acute-phase protein, complement, which is released by neutrophils and mononuclear cells, can aggravate the inflammatory response and cause lasting damage to the vascular bed and its surrounding tissues (Yin et al. 2014). The study showed that rats administered ASA developed severe enteritis, leading to an inflammatory reaction. MOS, particularly at elevated concentrations, significantly decreased the production of proinflammatory cytokines and alleviated inflammation in the intestines.

Conclusions

Our findings show that 600 mg/kg BW MOS treatment can successfully reduce ASA-induced intestinal damage and improve the immunity of intestinal injury rats by modulating serum cytokines, immune cells, proinflammatory cytokines, and intestinal morphology. An effective dose of 600 mg/kg BW of MOS is best for repairing ASA-induced intestinal damage in rats. Additional studies are required to determine the effects of MOS on intestinal flora, its metabolites, and its influence on other intestinal diseases.

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