The Role of Curcumin in Bacterial Translocation and Inflammatory Response in an Experimental Intestinal Obstruction Models in Rats*

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ABSTRACT

Introduction: Intestinal obstruction is associated with impaired intestinal barrier function and the translocation of the enteric bacteria to the systemic circulation, thus leading to a distortion in the balance of the anti-inflammatory cytokines. The aim of this study was to evaluate the effects of curcumin on the bacterial translocation and inflammatory response induced by a mechanical bowel obstruction in a rat model.

Materials and Methods: Thirty Wistar albino rats weighing 200-250 g were randomized into three groups of 10 rats each. Group 1 (sham) underwent only ileocecal junction dissections; Group 2 (intestinal obstruction) underwent complete ileal ligations; and Group 3 (intestinal obstruction + curcumin) underwent complete ileal ligations and received intraperitoneal administration of curcumin at a dose of 100 mg/kg. Twenty-four hours later, the rats were sacrificed by drawing blood from the heart for the biochemical analyses. The peritoneal swab culture, liver, mesenteric lymph nodes (MLNs), spleen, and ileum were collected for the microbiological and histopathological analyses.

Results: Curcumin reduced the secretion of the inflammatory cytokines, the damage and bacterial translocation; prevented the formation of inflammatory changes in the intestine, liver, spleen, and mesenteric lymph nodes; and also significantly prevented the formation of intestinal damage subsequent to the intestinal obstruction (p< 0.05).

Conclusion: This experimental study showed that curcumin may have protective effects against bacterial translocation and intestinal oxidative damage in mechanical intestinal obstructions. Further experimental studies are needed to explain the exact mechanism of this beneficial effect.

Key words: Intestinal obstruction, Bacterial translocation, Inflammatory cytokines, Curcumin

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* Editor’s Note: Self-assessment questions for this article are found on page 38.
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ÖZET

Ratlarda DeneySEL Obstrüksiyon Modelinde Bakteriyel Translokasyon ve İNflamatuvar YanIT Üzerinde Kurkuminin Rolü

GİRİŞ: Intestinal obstrüksiyonda başırsak bariyer fonksiyonunun bozulması ve enterik bakterilerin sistemik dolaşma geçmesiyle antiinflamatuvar sitokinlerin dengesi bozulur. Bu çalışmanın amacı, ratlarda deneySEL obstrüksiyon modelinde bakteriyel translokasyon ve inflamatuar yanıt üzerinde kurkuminin etkisini değerlendirilmektir.

MALERIAL VE METOD: 200-250 g ağırlığında 30 adet wistar-albino türü rat her bir grupta 10 adet olmak üzere üç gruba ayrıldı: Grup 1 (sham), yalnızca ileocekal bısgık diseksiyonu; Grup 2 (intestinal obstrüksiyon), komplet ileal ligasyon; Grup 3 (intestinal obstrüksiyon + kurkumin), komplet ileal ligasyon ve 100 mg/kg dozunda intraperitoneal kurkumin uygulandı. Ratlar 24 saat sonra biyokimyasal analizler için intrakardiyak kan alınarak sakrifiye edildi. Periton sürüntüsü, mezenterik lenf nodu, karaciğer ve ileal doku örnekleri mikrobiyolojik, biyokimyasal ve histopatolojik olarak incelendi.

BULGULAR: Kurkumin inflamatuar sitokinlerin salgılanmasını ve bakteriyel translokasyonu azalttı, başırsak, karaciğer, dalak ve mezenterik lenf düğümlerinde inflamatuar değişikliklerin oluşumunun engelledi. Aynı zamanda başırsak tıkanıklığında başırsak hasarı oluşmasını önemli derecede önledi (p< 0.05).

SONUÇ: Bu deneySEL çalışma kurkuminin mekanik başırsak tıkanıklığında oluşan bakteriyel translokasyon veintestinal oksidatif hasara karşı koruyucu etkisi olduğunu göstermektedir. Ayrıca, bu yararı etkinin mekanizmasını tam olarak açığa koyamak için daha çok deneySEL çalışmalara ihtiyaç duyulmaktadır.

ANAHAT KELİMELER: İntestinal obstrüksiyon; bakteriyel translokasyon; inflamatuar sitokinler; kurkumin

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INTRODUCTION

Despite the different treatment modalities developed in recent years, intestinal obstructions (IO) remain one of the prominent causes of abdominal emergencies, which continue to be a problem in surgical practice[1]. Under normal circumstances, the intestinal mucosa is a major barrier preventing the bacteria colonizing the gut from invading systemic organs and tissues[1-3]. In case of an IO, this barrier is disrupted. In consequence, the bacteria within the lumen rapidly multiply and pass across the intestinal wall into the bloodstream to migrate to the mesenteric lymph nodes (MLN) and sterile systemic organs like the liver and spleen, leading to bacterial translocation[4].

Bacterial translocation is suggested to be an important factor contributing to the development of sepsis[4-7]. Sepsis and multisystem organ failure (MOF) are major complications leading to increased postoperative morbidity and mortality in patients with IO[8,9].

The origin of curcumin -a polyphenol- is the root of the turmeric plant Curcuma longa, and curcumin is the substance that gives curry its characteristic yellow-orange color and spicy flavor[10]. Various anti-neoplastic, cytoprotective, anti-inflammatory, and antioxidant characteristics of curcumin have already been demonstrated in several experimental and clinical studies. Furthermore, both preventive and therapeuetic anti-inflammatory effects of curcumin treatment have been shown in a number of animal models[11,12].

To date, there is no study in the literature focusing on the effect of curcumin on bacterial translocation subsequent to IO in rats. In this experimental study, we aimed to investigate the effect of curcumin in reducing the severity of the inflammatory response and bacterial translocation induced by a mechanical IO.

MATERIALS and METHODS

Chemicals

In order to prepare the study solution, the curcuminoid manufactured by Sigma (C7727, St. Louis, MO) was dissolved in dimethyl sulfoxide at a dose of 1 mg/mL and stored in brown glass vials at 4°C.

Animals

Thirty Wistar albino rats, each weighing 200-250 g, were included in the study at the Dicle University Health Sciences Application and Research Center. The study was designed in compliance with the “Animal Welfare Act and the Guide for the Care and Use of Laboratory Animals” guidelines approved by the Dicle University School of Medicine, Animal Care and Use Local Ethics Committee. Before the initiation of the study, the rats were kept in cages and nourished with standard food and water in an air-conditioned room at a constant temperature of 22 ± 2°C, where 12-hour light/dark cycles were applied. On the night
before the surgery, the animals were fasted but had free access to water. The rats were divided into three groups (n= 10 each): Group 1 (Sham, S) underwent only ileocecal junction dissections. Group 2 (intestinal obstruction, IO) underwent ileocecal junction dissections with ileal ligations (1 cm proximal to the cecum, with 3-0 silk sutures and no additional medication). Group 3 (intestinal obstruction + curcumin, IO + C) rats underwent ileocecal junction dissections with ileal ligation and received intraperitoneal administration of 100 mg/kg of curcumin.

**Surgical Procedure**

Anesthesia was performed using 50 mg/kg of ketamine hydrochloride (Ketalar®, Parker Davis, Eczacibasi, Istanbul, Turkey) and 10 mg/kg xylazine (Rompun®, Bayer AG, Leverkusen, Germany) administered intramuscularly to all rats. Under sterile conditions, the laparotomy was started with a midline incision and the ileocecal junction was dissected. After the administration of 2 mL saline into the peritoneal area, the abdominal wall was closed in a single layer in Group S. In Groups IO and IO + C, following the laparotomy through the midline incision, the ileocecal junction was revealed, and the distal ileum was ligated using 3-0 silk sutures 1 cm proximal to the cecum. Thus, the passage was obstructed without hindering the blood circulation. Subsequently, 2 mL of saline was administered into the peritoneal area and the abdominal wall was closed in a single layer. Twenty-four hours later, the rats were anesthetized and sacrificed by drawing blood from the heart for the biochemical analyses. A thoracoabdominal midline incision was made immediately under completely sterile conditions. After the abdomen was revealed, peritoneal swab cultures were obtained for the microbiological analysis, and a 1 mL blood sample was drawn from the inferior vena cava. Samples from the liver, MLNs, spleen, and ileum were obtained for the microbiological and histopathological examinations. The blood was centrifuged and the derived serum was stored at -80°C until the analyses were performed. For the histopathological evaluation, the tissues were placed in 10% formaldehyde solution in plastic containers after they were rinsed with saline to remove the tissue and blood residues.

**Biochemical Analyses**

The collected blood samples were tested for the total oxidant activity (TOA), total antioxidant capacity (TAC), paraoxonase (PON), tumor necrosis factor-alpha (TNF-α), interleukin (IL)-6, IL-1β, and C-reactive protein (CRP).

**Measurement of TOA**

The TOA of the supernatant fractions was tested using a novel automated measurement method developed by Erel[13]. The method is based on the oxidation of the ferrous iron-dianisidine complex into ferric ion by the oxidants contained in the sample. This oxidation is enhanced by the glycerol molecules, which are copious in the reaction medium. In an acidic medium, the ferric ion results in a colored complex with xylene orange. The color intensity measured through spectrophotometry correlates with the total amount of oxidants in the sample. Hydrogen peroxide is used for the calibration of the assay, and the results are expressed in μmol H₂O₂ Eq/L.

**Measurement of TAC**

The TAC value in the supernatant fractions was also assessed with the help of a novel automated measurement method developed by Erel[14]. This method is based on the formation of the most potent biological free radical: the hydroxyl radical. During the assay, the ferrous ion solution in Reagent 1 is mixed with the hydrogen peroxide in Reagent 2. The resulting sequential radicals, including the brown dianisidinyl radical cation generated by the hydroxyl radical, are also potent ones. Through this method, the antioxidative effect of the sample is measured against the potent free radical reactions initiated by the produced hydroxyl radical. The results are expressed in mmol Trolox Eq/L.

**Measurement of PON**

For the measurement of the serum PON levels, the modified spectrophotometric Eckerson method was used[15]. The initial paraoxon (0.0-diethyl–0-p-nitrophenylphosphate; Sigma Chemical Co. London, UK) hydrolysis rates were measured based on the liberated p-nitrophenol at 405 nm at a temperature of 37°C. The obtained results are expressed in U/L[14].

**Measurement of TNF-α, IL-6, IL-1β, CRP**

The enzyme-amplified sensitivity immunoassay method was used for the quantification of TNF-α, IL-6 and IL-1β (DiaSource; Nivelles, Belgium). For the measurement of the serum high sensitivity (Hs)-CRP levels (DRG; NJ, USA), the enzyme-linked immunosorbent assay method was used.

**Microbiological Assay**

Blood samples drawn from the heart were cultured both aerobically and anaerobically on BacTec™ Peds (Becton-Dickinson Diagnostic Inc., Sparks, MD, USA). The BD-Phoenix 100 TM system was used for the
microbiological identification. The peritoneal swabs and positive cultures were inoculated into blood agar, eosin methylene blue (EMB) agar, chocolate agar and Sabouraud-dextrose agar. The MLNs, spleen and liver were excised and stored within sterile brain-heart infusion media in sterile glass vials. The vials were re-weighed, and tissue homogenates were prepared in 2 mL brain-heart infusion with the help of a sterile mortar and pestle. A 0.1 mL volume of each homogenate was inoculated in blood agar, EMB agar, chocolate agar, and Sabouraud-dextrose agar. The agar plates were incubated at 37°C and were examined at the 24 hour and 48 hour time points. In order to calculate the incidence of bacterial translocation, the number of the rats with positive bacterial culture was divided by the total number of rats included in the study.

**Histopathological Assessment**

The ileal segment and liver tissues were fixed in 10% formalin solution and embedded in paraffin blocks, from which 4 μm cross-sections were obtained. The tissues were stained with hematoxylin-eosin according to the standard protocols. The ileal segment and liver samples were examined in terms of the degree of the inflammatory cell infiltrate. The ileal segments were also examined using light microscopy (Nikon ECLIPSE 80i) for the ileal mucosal injury score by an expert pathologist blinded to the study groups. In light of the literature, the changes were graded as follows: Grade 0, no change; Grade 1, mild changes; Grade 2, moderate changes; and Grade 3, severe changes[16].

**Statistical Analysis**

Statistical analyses were carried out using the SPSS for Windows 11.5 (SPSS Inc., Chicago, IL, USA) software package. The data were expressed as mean ± SD (standard deviation) for the biochemical values. The groups were compared through the nonparametric Kruskal-Wallis test. Mann-Whitney U test was used for the binary comparisons of the continuous variables, and the chi-square test was employed for the categorical variables. A p value below 0.05 was considered as significant.

**RESULTS**

All animals survived the study period. The biochemical results are summarized in Table 1. IO showed a statistically significant relationship with oxidative stress. Although varying serum PON and total oxidant status (TOS) levels were observed between the groups, no such difference was found in terms of the total antioxidant status (TAS) levels: both in the S group and the IO + C group, the PON activity was higher than in the IO group. The TOS levels were increased in the IO group compared to the S group, while the administration of C in the IO + C group significantly prevented the increase in TOS levels.

The inflammatory cytokines TNF-α, IL-6, IL-1β, and CRP were increased subsequent to the IO. In the IO + C group, treatment with C significantly decreased all these cytokines in comparison to the IO group.

The histopathological grading of the liver and ileum are summarized in Table 2. In the IO group, the inflammation scores of the ileum (p< 0.001) and liver (p< 0.001) were found to be higher than in the S group. The score indicating the ileal mucosal damage was also higher in the IO group (p< 0.001). In the IO + C group, the liver inflammation scores were found to be higher in comparison to the S group, while the inflammation scores of the ileum and liver were lower.

| Table 1. Biochemical results of the groups |
|-----------------------------|-----------------------------|-----------------------------|
| Groups | S (n= 10) | IO (n= 10) | IO + C (n= 10) |
|-----------------------------|-----------------------------|-----------------------------|
| PON (U/L) | 35.54 ± 8.52 | 18.68 ± 4.26<sup>a</sup> | 26.48 ± 4.02<sup>b</sup> |
| TAS (mmol Trolox Eq/L) | 0.72 ± 0.059 | 0.71 ± 0.09 | 0.83 ± 0.06 |
| TOS (μmol H₂O₂ Eq/L) | 12.14 ± 1.21 | 33.52 ± 10.58<sup>a</sup> | 16.15 ± 3.73<sup>a,b</sup> |
| TNF-α (pg/mL) | 1.93 ± 0.86 | 7.59 ± 1.72<sup>a</sup> | 3.95 ± 2.86<sup>b</sup> |
| IL-6 (pg/mL) | 31.25 ± 8.45 | 65.83 ± 20.44<sup>a</sup> | 33.87 ± 9.08<sup>c</sup> |
| IL-1β (pg/mL) | 0.47 ± 0.11 | 1.62 ± 0.59<sup>a</sup> | 0.72 ± 0.16<sup>c</sup> |
| CRP (mg/L) | 30.46 ± 4.64 | 165.27 ± 41.06<sup>a</sup> | 54.37 ± 6.83<sup>a,b</sup> |

PON: Paraoxonase, TAS: Total antioxidant status, TOS: Total oxidant status, TNF: Tumor necrosis factor, IL: Interleukin, CRP: C-reactive protein.

Data are given as Mean ± SD.

<sup>a</sup> Significantly different when compared with S group (p< 0.001),

<sup>b</sup> Significantly different when compared with IO group (p< 0.001),

<sup>c</sup> Significantly different when compared with IO group (p= 0.01).
than in the IO group. The height of the intestinal villi was shorter in the IO group. However, the administration of C in the IO + C group avoided the shortening of the intestinal villi and demonstrated a protective effect on the intestinal mucosal damage score (p < 0.001) (Figures 1, 2).

The culture results are summarized in Table 3, expressed as the number of rats with positive bacterial culture divided by the total number of rats. No difference was observed among the groups regarding the peritoneal cultures. The blood (p = 0.002), liver (p < 0.001), spleen (p = 0.007), and MLN (p = 0.007) cultures gave statistically significant positive results in the IO group in comparison to the S group. However, the ratio of the positive cultures was lower in the IO + C group that received treatment with C. The ratios of the positive blood, liver, spleen, and MLN cultures were found to be significantly higher in the IO group compared to the IO + C group (p = 0.023). *Escherichia coli* (88.6%), *Proteus mirabilis* (7.6%) and *Klebsiella* spp. (3.8%) were the leading species among the isolated microorganisms.

<table>
<thead>
<tr>
<th>Groups</th>
<th>S (n= 10)</th>
<th>IO (n= 10)</th>
<th>IO + C (n= 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver inflammation score</td>
<td>0.11 ± 0.31</td>
<td>1.34 ± 0.48&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.40 ± 0.52&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ileum inflammation score</td>
<td>1.00 ± 0.00</td>
<td>2.59 ± 0.51&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.40 ± 0.97&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ileal mucosal damage score</td>
<td>0.00 ± 0.00</td>
<td>1.39 ± 0.50&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.80 ± 0.42&lt;sup&gt;b,d&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Data are given as Mean ± SD.

<sup>a</sup> Significantly different when compared with S group (p< 0.001),
<sup>b</sup> Significantly different when compared with S group (p< 0.01),
<sup>c</sup> Significantly different when compared with IO group (p< 0.01),
<sup>d</sup> Significantly different when compared with IO group (p≤ 0.05).

Figure 1. A. Sham group. Mild mucosal inflammation (HE stain, x200); B. IO group. Moderate to severe inflammation and edema in the mucosa with subtotal villous atrophy (HE stain, x200); C. IO + C group. Mild to moderate inflammation and edema in mucosa of the villi, some of which show slightly blunted ends (HE stain, x200).

Figure 2. A. Sham group. Mild vascular congestion in the liver parenchyma (HE stain, x200); B. IO group. Moderate portal inflammation in the liver (HE stain, x200); C. IO + C group. Minimal portal inflammation in the liver (HE stain, x200).
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DISCUSSION

Mechanical intestinal obstruction (MIO) is one of the most common causes of acute abdominal problems. The impairment in the normal activity of the intestinal tract by MIO may lead to important results like bacterial overgrowth in the gut lumen, structural changes in the layers of the bowel wall, detrimental effects on the blood circulation, and reduced competence of the mucosal immune function. The intestinal mucosa is a major barrier preventing the systemic spread of the colonizing bacteria from the gut[17]. Bacterial translocation is defined as the phenomenon by which live bacteria and/or their products cross the intestinal barrier into the bloodstream to reach the MLNs and sterile systemic organs like the liver and spleen[4]. Factors facilitating the occurrence of bacterial translocation can be listed as ionizing radiation, endotoxins, trauma, nutritional stress, peritoneal inflammation, kidney failure, changes in the microflora, obstructions, hemorrhagic shock, cellular immune disorders, IgA defects, phagocytic cell defects, total parenteral nutrition, antibiotics, and immunosuppression[18]. Bacterial translocation is suggested to be an important factor contributing to the development of sepsis[4,5]. Sepsis and MOF are major complications leading to increased postoperative morbidity and mortality in patients with IO[3]. In the present study, the bacterial translocation caused by the IO was demonstrated through the bacterial growth in the MLN, liver, spleen, and blood as well as the intestine.

TNF-α is known to be an important mediator in MOF and the systemic inflammatory response syndrome. Clinically observed high serum levels are interpreted in favor of a probable infection. Increased amounts of the cytokines TNF-α and IL-6 in the circulation have also been detected in local injuries. The absorption of the endotoxins following the IO leads to an increase in the TNF-α levels. This increase in the secretion of TNF-α causes a necrotizing effect on the intestinal and vascular endothelium[19]. Cytokines like IL-1β and IL-6 lead to proinflammatory and inflammatory changes and the rapid immune response, enabling the elimination of the pathogens[20]. IL is known to be an effective inhibitor of a number of molecules leading to oxidative injury caused by the generation of free radicals such as lipoxygenase, cyclooxygenase, xanthine oxidase, xanthine dehydrogenase, nitric oxide synthase, and TNF-α[21,22]. CRP is a classical acute phase reactant that can increase up to 1000-fold in cases of infection, ischemia, trauma, burns, and inflammatory events. CRP is an indicator of the severity of tissue damage and/or inflammation, and its levels increase in parallel to the severity of the condition[23]. In their study on bacterial translocation following IO, Cevikel et al.[24] reported that the CRP levels are related to the severity of the bacterial translocation and can therefore be beneficial in monitoring the severity of bacterial translocation that occurs due to an IO. In our study, we observed that the levels of inflammatory cytokines and CRP significantly increased after the IO and can thus be used as parameters to monitor the progress of the bacterial translocation.

PON is a glycoprotein with antioxidant properties against peroxidative damage[25]. Further, studies have shown that the measurement of the TAS and TOA values is beneficial in the determination of the oxidant and antioxidant parameters[26]. In the present study, it was observed that the PON values diminished and the TOA values increased following an IO, and these changes were observed to be statistically significant. On the other hand, no significant difference was observed in the TAS values. This finding may be interpreted to indicate that the oxidative stress increases following an IO and the response brought about through antioxidant mechanisms in parallel to this increase is not adequate.

### Table 3. Microbiological culture results of groups

<table>
<thead>
<tr>
<th>Groups</th>
<th>S (n= 10)</th>
<th>IO (n= 10)</th>
<th>IO + C (n= 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood culture (c/d)</td>
<td>0/10</td>
<td>8/10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1/10&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Liver culture (d/e)</td>
<td>0/10</td>
<td>9/10&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1/10&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Spleen culture (d/e)</td>
<td>1/10</td>
<td>8/10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1/10&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>MLN culture (d/e)</td>
<td>2/10</td>
<td>9/10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2/10&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Peritoneal culture (d/e)</td>
<td>2/10</td>
<td>4/10</td>
<td>1/10</td>
</tr>
</tbody>
</table>

MLN: Mesenteric lymph nodes.
Data are given as (d/e) = positive culture/total rats of group.
<sup>a</sup> Significantly different when compared with S group (p< 0.01),
<sup>b</sup> Significantly different when compared with S group (p< 0.001),
<sup>c</sup> Significantly different when compared with IO group (p< 0.05).
The increased intraluminal pressure after the IO leads to a parallel increase in the capillary hydrostatic pressure and tissue edema, as well as an impact on the arterial blood circulation\(^{27}\). The inflammation that develops in the intestinal tissue plays an important role in the physiopathology of IO\(^{28}\). Akcay et al.\(^{16}\) reported that following an IO, the inflammatory infiltration, edema and hyperemia show a significant increase in the small intestinal tissue, with a disturbance in the structure and diminished lengths of the intestinal villi. While edema alone is reported in the intestine after a simple IO, edema, bleeding and necrosis are observed in those in which strangulation develops\(^{29}\). In our study, hepatic inflammation was investigated in addition to the ileal changes subsequent to the IO.

In conclusion, a significant increase in hepatic and ileal inflammation and moderate degenerative epithelial changes in the ileal mucosa, accompanied by subtotal villous atrophy, severe edema and inflammation, were observed following the IO. These changes were also found to be statistically significant.

The results obtained from the present study demonstrate that the intraperitoneal administration of curcumin maintains antioxidant defenses and reduces the intestinal mucosal injury, oxidative damage in the ileum, and bacterial translocation in patients with MIO. However, further studies of clinical and experimental models are required to evaluate the antioxidant and anti-inflammatory properties of curcumin.

REFERENCES


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