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THE IMPACT OF SELENIUM SUPPLEMENTATION ON THE MECHANISMS OF THE INFLAMMATORY PROCESS IN PATIENTS WITH ULCERATIVE COLITIS: A SYSTEMATIC REVIEW

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ABSTRACT

Introduction and purpose: Ulcerative colitis (UC) is one of the subtypes of inflammatory bowel disease (IBD), alongside Crohn's disease. UC is associated with an inflammatory process occurring in the mucosa and submucosa of the large intestine. Various factors interact in the pathogenesis, determining the development and progression of UC. These include genetic and environmental factors, as well as changes in the immune system. The role of oxidative stress is also emphasized. Serum selenium (Se) levels in patients with UC were lower than in healthy individuals. Furthermore, the level of this microelement was inversely correlated with disease activity. In this paper, we review the physiological roles of Se and its impact on the course of UC. Given the multifaceted role of this microelement in the human body, we primarily focus on the influence of Se as a dietary component on immune response mechanisms and inflammation.

Materials and methods: A systematic literature search was conducted using the electronic databases, including PubMed and Google Scholar. Various combinations of the following keywords were used in the search „ulcerative colitis”, „selenium”, „oxidative stress” and „ulcerative colitis pathophysiology”. The search was limited to publications from the last 5 years. Those directly related to the topic under discussion were then selected.

Conclusions: Studies confirm the beneficial effect of Se on the course of UC. They present the mechanisms underlying the anti-inflammatory action of Se, from molecular processes at the gene expression level to clinical evidence demonstrating improved quality of life. The effects of Se supplementation appear promising, but further research in this area is needed.

KEYWORDS

Ulcerative Colitis, Selenium, Oxidative Stress

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Introduction

Ulcerative colitis (UC) is a chronic inflammatory disease that effects the rectum and has the potential to spread continuously to the colon. The inflammatory process is limited to the mucous membrane of the large intestine. (Abraham & Cho, 2009) The clinical course is characterized by periods of exacerbation and remission. The predominant symptom in most patients is rectal bleeding. Symptoms generally reflect the severity of the disease, and as the disease progresses, there is an increased frequency of loose stools. Patients also complain of cramping abdominal pain, usually located in the lower left quadrant, and report relief after a bowel movement. In 2023 the global incidence of UC was estimated at 5 million cases, with a steady increase in this number. (Le Berre et al., 2023) The incidence of the disease is similar in both sexes, although according to some studies, men are slightly more affected. The peak age of onset for UC is between the second and fourth decades of life; however, the incidence of inflammatory bowel disease with late-onset is on the rise. (Jeurig et al., 2016; Kontola et al., 2023) Although the exact cause of UC remains unknown, it is suggested that a combination of various factors, such as genetic predisposition, impaired immune response, and environmental factors, are involved in its pathogenesis. (Kaur & Goggolidou, 2020) The role of dietary factors and micronutrients, especially their deficiencies, should not be overlooked. This concept is supported by studies showing reduced Se levels in patients with UC. (Liu et al., 2024) UC is a global health challenge, and its chronic course and troublesome symptoms worsen patients' quality of life. Currently, the goal of treatment is to induce remission and prevent relapses. Depending on the extent and severity of the disease, treatment options include: 5-aminosalicylic acid derivatives, corticosteroids, immunosuppressive drugs (e.g. azathioprine, cyclosporine, or tacrolimus), as well as biological drugs (e.g., infliximab, adalimumab, vedolizumab) and JAK kinase inhibitors (tofacitinib). In severe cases, where drug therapy is ineffective or complications arise, surgical treatment may be necessary. (Gajendran et al., 2019)

Despite the availability of the presented therapeutic options and ongoing research in this area, some patients still fail to achieve significant clinical improvement. This suggests a need to explore effective supportive strategies. In this review, we attempt to summarize the role of oxidative stress in the pathophysiology of UC and the importance of the health-promoting properties of selenium, focusing mainly on its antioxidant and anti-inflammatory properties. The primary objective of this study is to demonstrate the potential Se supplementation to support the treatment of UC, taking into account its mechanisms of action on inflammation.

Description of The State of Knowledge**The role of oxidative stress in the pathogenesis of UC**

The exact pathogenesis of UC remains unclear. It is believed that oxidative stress plays a crucial role among the various mechanisms that contribute to the development of this disease. Oxidative stress is a phenomenon in which the level of reactive oxygen species (ROS) is increased, which has a destructive effect on the regulation of cellular metabolism and contributes to the damage of cellular components. (Lushchak, 2011) Studies in animal models with dextran sulfate sodium (DSS)-induced colitis show increased ROS production. A corresponding decrease in antioxidant levels has been observed. (Zhu & Li, 2012) In addition to increased ROS production, the mechanisms of oxidative stress include impaired antioxidant defense, damage to biomolecules, recruitment of immune cells and activation of inflammatory pathways. Figure 1. (Muro et al., 2024) Oxidative stress destabilizes the epithelial barrier of the gastrointestinal tract, facilitating bacterial invasion, which triggers an immune response. (Vona et al., 2021) The interaction between immune cell recruitment and oxidative stress appears to be highly complex. This interaction may initiate a vicious cycle, where oxidative stress induces inflammation, subsequently leading to increased ROS production. When discussing pathogenesis, it is also worth mentioning two transcription factors – NF- κ B (nuclear factor- κ B) and Nrf2 (nuclear factor (erythroid-derived 2)-like 2) - which regulate the response to oxidative stress and inflammation. (Wardyn et al., 2015) Studies have shown that NF- κ B is abnormally activated in patients with

inflammatory bowel disease, and prolonged NF- κ B signaling may exacerbate inflammation in patients with UC. (Andresen et al., 2005) It turns out that ROS may play a role in this by activating the NF- κ B pathway. (Morgan & Liu, 2011) This leads to increased expression of various pro-inflammatory cytokines. In the course of UC, a predominance of an immune response dependent mainly on Th-2 and Th-17 lymphocytes is observed, which is associated with the production of specific cytokines, including IL-5, IL-6, IL-13, IL-17, IL-21, IL-22, and TNF- α . (Nakase et al., 2022) Among the Th cell population, which also plays a role in the pathogenesis of UC, regulatory T cells (Treg cells) stand out, whose role is to suppress the inflammatory response. (Gomez-Bris et al., 2023) At the other end of the spectrum from NF- κ B is the aforementioned Nrf2 factor, whose role is to inhibit excessive ROS production and suppress the inflammatory response in the intestinal mucosa. (Gerstgrasser et al., 2017) Nrf2 controls the expression of several enzymatic antioxidants, such as GPx (glutathione peroxidase) and SOD (superoxide dismutase), thereby influencing the maintenance of cellular homeostasis. The harmony between the induction of NF- κ B and Nrf2 signaling pathways plays a role in controlling oxidative stress in inflammatory bowel disease. (Muro et al., 2024) The absence of Nrf2 may enhance NF- κ B activity, leading to increased production of proinflammatory cytokines. Interestingly, NF- κ B can function both as an activator and an inhibitor of genes regulated by Nrf2. (Wardyn et al., 2015) These findings may help explain the growing interest in substances with antioxidant potential as supportive therapy in the treatment of UC.

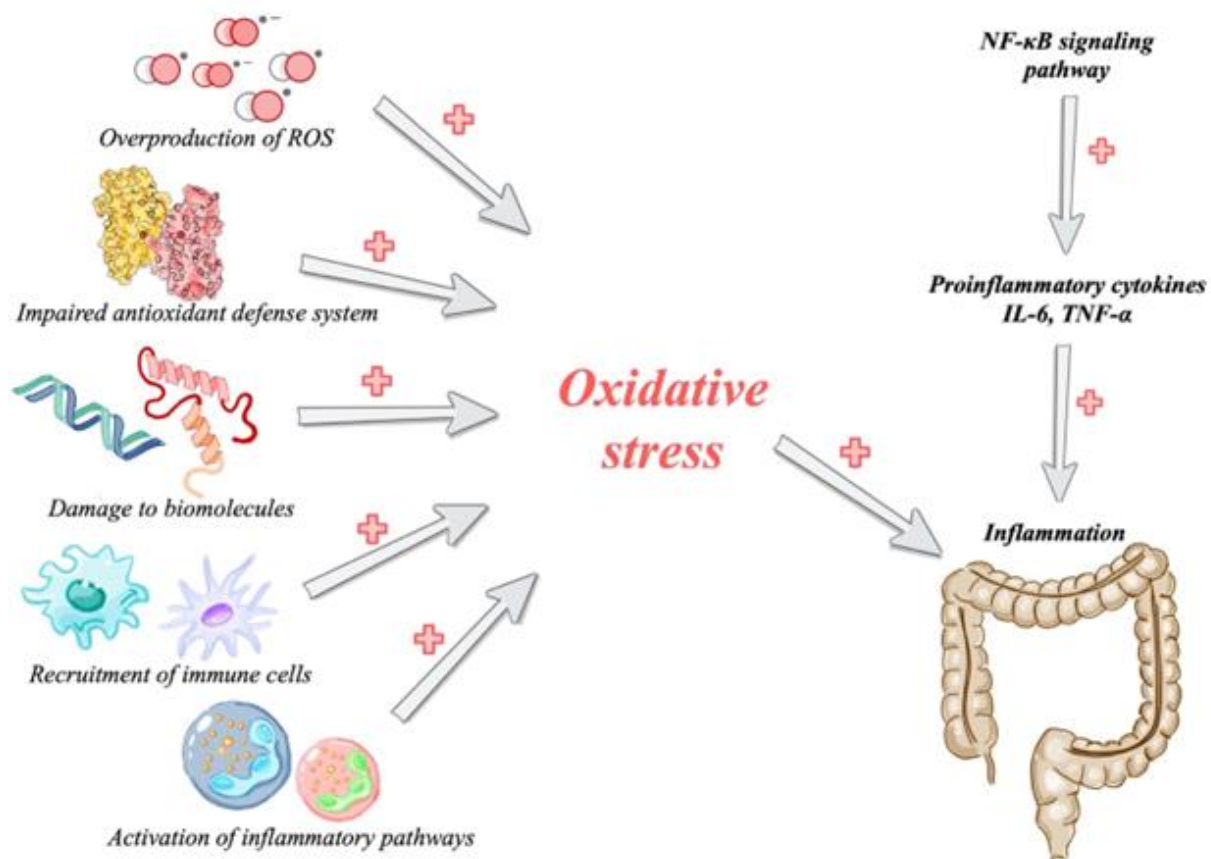


Fig. 1. Components of the oxidative stress mechanism. [Based on Muro et al. (Muro et al., 2024)]

Selenium as a trace element in the human body

Selenium is one of the essential micronutrients in the human body. It must be supplied to the body through food. The Se content in individual products varies and depends on the concentration of this micronutrient in the soil and water. Products rich in Se include offal, especially kidneys, and seafood: shellfish and fish. The vegetables that contain the highest amounts of Se are garlic, mushrooms, and dry legume seeds. Selenium yeast is a safe and natural source of Se, and the vitamins it contains, mainly B and E, additionally support its absorption. The bioavailability of this element depends on its chemical form. The most easily absorbed forms are selenites (IV) and (VI), selenomethionine, selenocysteine, and amino compounds of

selenium. The average requirement (EAR) for both women and men is 45 µg, while the recommended intake (RDA) for both group is 55 µg. However, it is important to note that the margin between the amount required for proper physiological function and the toxic dose of Se is narrow. The toxic effect of Se ranges from 400 to 700 µg/day. (Jarosz et al., 2020; Kieliszek et al., 2012)

Se performs various functions in the human body. When combined with amino acids, it forms selenoproteins. These are responsible for neutralizing reactive oxygen species that are produced during the oxidative burst when neutrophils and monocytes destroy pathogens. In this way, selenoproteins protect the body's cells from damage. (Gärtner et al., 2002; Spallholz et al., 1990) As a component of glutathione peroxidase, it enables the reduction of peroxides and the deactivation of oxygen radicals, thereby reducing oxidative stress and the risk of cancer and autoimmune diseases. Selenoprotein P and thioredoxins also have antioxidant properties. In addition, Se is a component of many enzymes and proteins involved in the structure of cell membranes, thereby performing enzymatic and stabilizing functions. This microelement also participates in the detoxification process by forming inactive complexes, which are then removed from the body. In this way, heavy metals such as cadmium, lead, and mercury can be eliminated.

Se also plays a crucial role in the proper functioning of the immune system. As a component of glutathione peroxidase and thioredoxin reductase, it protects leukocytes from damage by free radicals. (Arthur et al., 2003) What is more, studies have shown that Se deficiency impairs lymphocyte function and antibody production, while supplementation stimulates the proliferation of these cells. In addition, Se influences the expression of receptors for cytokines that stimulate the activation and differentiation of CD4⁺ lymphocytes. (Rayman, 2000) Se also limits the production of pro-inflammatory prostaglandins and leukotrienes, which directly reduces inflammation and the fibrosis process. Given its important role in the proper functioning of the immune system, Se supplementation may modify the course of many autoimmune diseases. One of these diseases is UC.

Selenium supplementation and the expression of SIRT1 and PGC-1 α genes.

SIRT1 (Silent information regulator 1) is a NAD(+)-dependent deacetylase which, through the deacetylation of histones and non-histone proteins, is responsible, among other things, for preventing oxidative stress and inflammation. (Singh & Ubaid, 2020) The results proved the role of the SIRT1 gene in colitis, when its level significantly decreased in intestinal parietal cells during the acute phase of colitis in mice. (Singh et al., 2010) A correlation between SIRT1 and PGC-1 α was observed. SIRT1 performs its biological functions through its ability to deacetylate certain transcription factors or coactivators such as PGC-1 α or NF- κ B. NF- κ B activity is thus inhibited, which reduces the transcription of pro-inflammatory genes. (Hwang et al., 2013) Deacetylated PGC-1 α , in its active form, protects against inflammation of the large intestine and plays a role in maintaining the integrity of the intestinal epithelium. (Cunningham et al., 2016) A similar concept is considered in a study conducted by M. Khazdouz et al. It evaluated the effect of selenium supplementation on the expression of SIRT1 and PGC-1 α genes involved in the inflammatory response in patients with mild to moderate UC. In a double-blind randomized clinical trial, a group of 50 patients with UC received 1 capsule of 200µg selenomethionine daily, while another 50 patients received a placebo for 10 weeks. The expression levels of SIRT1 and PGC-1 α genes were examined in peripheral blood mononuclear cells using real-time polymerase chain reaction. SIRT1 gene expression was significantly increased in the Se group compared to the placebo group ($p < 0.001$). In contrast, the increase in PGC-1 α gene expression was not statistically significant. This suggests that Se contributes to a decrease in the inflammatory response through the activation of SIRT1. However, the authors point to the need for further research to determine the exact role of Se in this process. (Khazdouz et al., 2020)

Selenium and ferroptosis

Ferroptosis is a type of cell death, different from apoptosis, that results from the accumulation of iron and lipid peroxidation. A characteristic feature of ferroptosis is oxidative damage caused by reactive oxygen species. Ferroptosis plays a role in the pathogenesis of various diseases, including cancer and inflammatory disorders. Recently, it has also been discovered that ferroptosis is one of the mechanisms of intestinal epithelial cell death in UC, however, the underlying relationship has not yet been fully elucidated. Nrf2 and Gpx4 are among the transcription factors that regulate the expression of antioxidant response elements. A growing number of studies show that the Nrf2/Gpx4 signaling pathway can effectively inhibit ferroptosis. In ulcerative colitis, on the other hand, the activity of this pathway is inhibited. In a study by Jie Shi et al. using in vivo and in vitro models, it was shown that selenium alleviates DSS-induced colitis and inhibits ferroptosis in intestinal epithelial cells by activating the Nrf2/Gpx4 pathway, which may represent a new option for the treatment of ulcerative colitis. (Shi et al., 2024)

The role of selenoprotein S in regulating the immune response in ulcerative colitis.

As already mentioned, Se plays a therapeutic role mainly due to its presence in selenoproteins as an amino acid-selenocysteine, which is responsible for modifying processes in inflammatory states. (Rayman, 2000) Selenoproteins are therefore proteins that mediate the beneficial effects of Se on the immune system. (Hatfield et al., 2014) Selenoprotein S (SelS) is present in intestinal epithelial cells and intestinal macrophages. (Speckmann et al., 2014) A study by Yujie Yao et al. showed that SelS deficiency in colonic epithelial cells contributed to the release of reactive oxygen species, cytokines, necroptosis, and intestinal barrier integrity disorders. In turn, a decrease in the level of this protein in macrophages activated M1 macrophage polarization, exacerbating damage to the colonic epithelium. (Yao et al., 2024) It is worth noting the differences in the function of both types of macrophages in inflammatory processes. M1 macrophages exacerbate damage in inflammatory bowel disease, while M2 macrophages lead to tissue repair and suppress inflammatory processes. (Du et al., 2021) There is evidence of a link between the conversion of macrophages from the M1 to the M2 phenotype and adequate Se availability. (Speckmann & Steinbrenner, 2014) Se supplementation in mice treated with DSS reduced M1 markers and upregulated M2 markers in the large intestine. (Kudva et al., 2015) In the aforementioned study, Yujie Yao et al. also evaluated the effect of Se supplementation on alleviating colon damage in a mouse model of induced UC. Intervention through exogenous supplementation of this micronutrient improved clinical symptoms (including food intake, body weight, and stool consistency), increased SelS protein levels, and reduced the induction of M1 markers. These findings underscore the protective role of Se in inflammatory bowel injury, clarifying the mechanism of action and the involvement of SelS in regulating the intestinal immune response. (Yao et al., 2024)

Sodium selenate alleviates sodium dextran – induced chronic colitis.

In a scientific study by Li-Xuan Sang et al., the effect of sodium selenate on the severity of chronic colitis in mice induced by oral administration of DSS was investigated. The effect of sodium selenate administration was assessed using the following indicators: comparison of survival, clinical symptoms (weight loss, diarrhea, rectal bleeding), disease activity index (DAI), colon length, and colon histology in mice that received sodium selenate and those that did not. Survival was similar in both groups, while clinical symptoms and DAI were less severe in the group of mice that received sodium selenate. In addition, this group had a significantly longer colon and lower macroscopic inflammation index. Histological examination measured the percentage and number of immune cells in the lamina propria lymphocytes (LPL) of the colon, the level of mRNA expression of inflammatory markers in colonic tissue, and the concentration of Th1, Th17, and Treg cytokines in LPL. Se significantly alleviated the symptoms of colitis and inflammatory changes in the histopathological picture by increasing the percentage of neutrophils and CD4⁺ CD25⁺ T lymphocytes, while reducing the percentage of $\gamma\delta$ T, CD4⁺, CD4⁺CD44⁺, and CD4⁺ CD69⁺ T cells in LPL. Se also reduced the expression of IL-6, IFN- γ , IL-17A, IL-21, T-bet, and ROR γ t, which are pro-inflammatory factors, while increasing the expression of IL-10 and Foxp3, which have anti-inflammatory properties. The results of this study indicate the protective potential of Se against chronic colitis. The likely cause of this effect is thought to be an increase in the number of CD4⁺ CD25⁺ Tregs, which contribute to the inhibition proinflammatory cytokines secretion and Th1, Th17, and $\gamma\delta$ T cell populations. (Sang et al., 2017)

Selenium supplementation and disease activity – evidence from a clinical trial

The literature does not provide many clinical studies on the therapeutic benefits of Se without combination with other antioxidants in patients with UC. In recent years, Maryam et al. Presented a randomized, double-blind clinical trial showing the therapeutic effects of Se supplementation on disease activity, quality of life, and inflammatory markers in 100 patients with UC. Participants with mild to moderate UC were randomly divided into two groups. One group received a daily oral capsule containing 200 mcg of selenomethionine (n=50), and the other group received a placebo capsule (n=50) for 10 weeks. After 10 weeks, the first group showed reduced disease activity and improved quality of life. These parameters were assessed using the SCCAI disease activity index and the IBDQ-9 questionnaire. In this group, the average concentration of interleukin-17 (a pro-inflammatory cytokine) also decreased, while the level of interleukin-10 did not differ significantly between the two groups. The results of this study show that Se supplementation as an adjunct to medical treatment has a positive effect on patients with UC by improving symptoms and quality of life. However, the authors note certain limitations of the study related to the lack of data on its long-term effects. They also emphasize the need for further research involving invasive diagnostic methods, such as endoscopy, to elucidate the precise mechanisms underlying the effects of Se supplementation in patients with UC. (Khazdouz et al., 2023)

Conclusions

UC is a serious health problem with a steady increase in the number of cases. The course of the disease is very burdensome and significantly impairs the quality of life of patients. The development of UC is driven by multifactorial processes, among which, immune response dysregulation with a visible influence of oxidative stress plays a prominent role. The high prevalence of the disease in developed countries also suggests a role for environmental factors, including dietary components. Given the occurrence of clinical cases unresponsive to current treatments, there is a need for further investigation into dietary interventions, with particular emphasis on the supplementation of various micronutrients. Understanding the role of oxidative stress in the pathogenesis of this disease underpins studies investigating various antioxidants, where their effects on symptom relief are evaluated. This also provides a source for new therapeutic strategies targeting this pathway. To date, results suggest potential benefits of Se in alleviating inflammation and oxidative stress in patients with UC. Further research is needed to better elucidate the pathological role of oxidative stress in UC and to identify effective interventions leveraging the antioxidant properties of Se. Future clinical trials could include a longer period of intervention in the form of Se supplementation. This would allow for the determination of an appropriate supplementation period and dosage. A thorough definition of the role of Se in UC may qualify this micronutrient as a potential adjunctive therapy for this disease entity.

Author's contribution:

Conceptualization: PG, PK

Methodology: PG, KB

Software: PK, JK

Check: JK, PK, KB, PG, HN

Formal analysis: KB, HN

Investigation: JK, KB

Resources: PK, PG

Data curation: KB, HN

Writing - rough preparation: JK, PG

Writing - review and editing: PK, HN

Visualization: KB, PG

Supervision: JK, HN

Project administration: PK, JK

Receiving funding: not applicable

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In preparing this work, the authors used Grammarly and DeepL as an application to improve language and readability. After using these tools, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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