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THE IMPACT OF DIET ON RHEUMATOID ARTHRITIS (RA) TREATMENT AND ACTIVITY

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ABSTRACT

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by joint inflammation, destructive changes, and numerous systemic complications. Treatment is based on disease-modifying antirheumatic drugs (DMARDs) and biological therapies. In recent years, increasing attention has been paid to the impact of diet on the risk of developing RA and disease activity, as well as the role of diet as an adjunctive therapy in the treatment of RA. Many studies indicate that an appropriately selected diet can modulate the inflammatory response, affect the gut microbiota and the overall clinical condition of patients with RA. We analyse publications from 2018–2025, including observational studies, randomized controlled trials (RCTs) and systematic reviews on the role of diet in RA. We discuss the effect of selected types of anti-inflammatory diets and omega-3 fatty acids, fiber, polyphenols, and vitamin D in patients with RA. We highlight also the role of the gut microbiota in RA.

Aim of this study: The objective of this study is to summarise the latest reports on how diet and its components impact RA activity and treatment.

Materials and methods: A literature review was conducted using the professional PubMed database. Articles published between 2018 and 2025 were included. The searches included combinations of the following keywords: "rheumatoid arthritis," "diet," "anti-inflammatory diet," "omega-3 fatty acids," "fiber" and "polyphenols".

KEYWORDS

Rheumatoid Arthritis, Diet, Anti-Inflammatory Diet, Omega-3 Fatty Acids, Fiber, Polyphenols

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Introduction

Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease that causes progressive inflammation of the synovial membrane and destruction of cartilage and bone. It is characterized by symmetrical inflammation of small and large joints, mainly in the hands and feet, pain, joint destruction, and the risk of disability. There are also extra-articular symptoms, such as fever, anemia, and osteoporosis. RA can cause damage to organs, including the kidneys, lungs, stomach, liver and increase the risk of cardiovascular disease. The etiology of RA is not fully understood. It occurs twice as often in women [1]. A characteristic symptom of this disease is morning stiffness. The disease is characterized by periods of exacerbation and remission. The DAS28-CRP and DAS28-ESR scales are used to assess disease activity. It takes into account the number of swollen joints, the number of painful joints, CRP or OB, and the patient's overall assessment of disease activity according to the VAS scale. Treatment is based on disease-modifying antirheumatic drugs (DMARDs), and nonsteroidal anti-inflammatory drugs are used for pain relief [2].

Genetic and environmental factors, including diet, play a significant role in the development of RA. Currently, there is considerable interest in the role of diet as an adjunctive therapy in the treatment of RA. Diet affects inflammatory processes and the gut microbiome [3]. The review describes the impact of different types of diets and their individual components on the treatment and activity of RA.

Rheumatoid Arthritis Epidemiology

The prevalence of RA varies depending on the geographical region. Globally, it is approximately 1%. RA is more prevalent in Western countries, where diets are dominated by large amounts of red meat, saturated fats, and carbohydrates. These products have pro-inflammatory effects and may contribute to the development of RA [4].

Gut microbiota

Research has shown that gut microbiota plays a significant role in the human body. Modifying its composition in an appropriate manner can protect against the development of many diseases and promote their treatment, including the treatment of RA.

Reduced microbiota diversity and changes in the prevalence of specific strains have been observed in patients with RA compared to the general population. A high proportion of Prevotella copri has been found in the microbiome of patients with early RA and in the pre-symptomatic phase. Prevotella copri is a known bacterial strain with pro-inflammatory properties. A diet rich in salt and sugar increases the proportion of this bacterium in the gut microbiome, thereby increasing inflammation in the body. This condition negatively affects the treatment of RA, which is an inflammatory disease. Researchers have found that Prevotella copri also has a large share in the microbiome of people with a genetically increased risk of developing RA. In people treated for RA, this relationship was not noticeable [5,6,7].

A reduction in the proportion of bacteria producing short-chain fatty acids (SCFA), which have anti-inflammatory properties, has been demonstrated in the microbiome of people with RA. However, the results are not entirely consistent across populations, which may be due to differences in diet, ethnicity, and sequencing methodology [8,9].

There is growing evidence that intestinal dysbiosis may contribute to the loss of immune tolerance and promote inflammatory processes associated with RA [10]. Proposed mechanisms linking the microbiota to the pathogenesis of RA include modulation of T cell balance (increase in pro-inflammatory Th17 cells, decrease in regulatory Treg cells), increased intestinal permeability allowing the translocation of microorganisms and their products, such as lipopolysaccharides and peptidoglycan, into the circulation, molecular mimicry, autoantibody induction, and the influence of microbiota metabolites (SCFA, tryptophan metabolites) on immune cell function. Experimental and clinical studies provide evidence supporting each of these mechanisms, although their relative importance in different phases of the disease is still under investigation [11].

Several studies have shown correlations between specific microbiological profiles and disease activity indices (DAS28), proinflammatory cytokine concentrations, and the presence of autoantibodies (RF, ACPA). Some analyses suggest that the metabolic potential of the microbiome may correlate better with disease progression than its composition. However, more detailed studies are needed [12].

Medications used in RA therapy, such as methotrexate and biological therapy, affect the composition of the gut microbiota. In some patients, DMARD treatment partially reverses dysbiosis. At the same time, there are large differences in the microbiological response between patients, which may be associated with differences in treatment efficacy [13].

Research into the use of probiotic supplementation and faecal microbiota transplantation (FMT) as adjunctive therapies for patients with rheumatoid arthritis (RA) is ongoing. While clinical data suggest that certain probiotics may have a mild beneficial effect on symptoms and inflammatory parameters, there are currently no large, conclusive randomised trials determining the efficacy and safety of FMT in RA. Diet and microbiota metabolites appear to have the potential to modify disease progression, but more extensive studies are needed [14].

Fiber-rich diet

Epidemiological data suggest that higher fiber intake (especially fiber from cereal products) correlates with a lower risk of RA and lower inflammation markers in part of the population. Short-term interventions in RA patients have shown an improvement in the gut metabolite profile (higher SCFA levels) and favorable changes in inflammation biomarkers, although clinical data, such as a reduction in DAS28, are limited and inconsistent.

Fiber fermented by the gut microbiota leads to the formation of short-chain fatty acids (SCFAs) (acetate, propionate, butyrate), which affect the function of immune cells. They regulate Treg lymphocyte function, reduce the production of pro-inflammatory cytokines, and improve the integrity of the intestinal barrier. In intervention studies, an increase in SCFA concentration after fiber supplementation was associated with a reduction in certain inflammatory markers [16].

A diet rich in fiber changes the composition and functions of the microbiota, increasing the abundance of species that produce beneficial metabolites. Due to the fact that dysbiosis of the gut microbiome has been observed in patients with RA, dietary interventions have the potential to restore balance in the composition of the gut microbiota. Improving the intestinal barrier reduces the translocation of bacterial products and systemic inflammatory stimulation, which theoretically may suppress the activity of rheumatic disease. Studies show

correlations between markers of intestinal permeability, changes in the microbiota, and the severity of inflammation [17].

Short-term interventions, such as supplementation with ready-made fiber preparations for 2–4 weeks in patients with RA, have shown an improvement in SCFA production and a reduction in certain pro-inflammatory cytokines, indicating a beneficial effect on inflammatory mechanisms. However, the clinical effects — DAS28 measurements, pain assessment, function — are often small, and the studies have limited sample sizes and varied methodologies. These results indicate a promising mechanism, but there is a lack of clear evidence to support the widespread inclusion of fiber supplementation as a therapeutic standard in RA [16].

On the other hand there are also reports indicating that, under certain conditions, a high fiber intake may lead to adverse pro-inflammatory effects in experimental models. Experimental studies in animal models have shown that a high fiber intake in the presence of Prevotella copri colonization can lead to the overproduction of certain metabolites (e.g., succinate, fumarate) and, consequently, to an exacerbation of the pro-inflammatory response and worsening of arthritis. This indicates that the effect of fiber is not universally beneficial — it depends on the composition of the host microbiota and bacterial metabolism. This suggests the need for individualized dietary recommendations [15,18].

Trace elements

Zinc and cadmium play an important role in the development and treatment of RA. It has been observed that people with RA have low blood zinc levels and high cadmium levels. Exposure to cadmium-containing dust is one of the risk factors for developing RA. Cadmium is found in tobacco smoke and smoking is one of the factors that increase the risk of developing RA. Cadmium interferes with the action of zinc. Zinc deficiency causes greater susceptibility to infections, including invasion by Porphyromonas gingivalis. This bacterium promotes the production of citrullinated autoantigens, which play a key role in the development of RA [19]. However, not all studies have shown a link between zinc levels and the development of RA [20]. Detailed research on this issue is needed.

The effect of vitamin B6 on the development of RA has also been studied. It has anti-inflammatory properties. The results of the studies are inconsistent. Some of them showed that high levels of vitamin B6 reduce the risk of developing RA [21]. Other studies show that vitamin B6 is a risk factor for the development of RA, and its role varies depending on the stage of the disease and other factors [20]. Further research is still needed to determine the exact role of vitamin B6 in RA.

Anti-inflammatory diets

Anti-inflammatory diets include the Mediterranean diet, vegan diet, and elimination diets (such as the ketogenic, vegan, or gluten-free diets).

Some studies have shown that RA patients following a generally anti-inflammatory diet experienced less pain and their BMI decreased. However, no reduction in inflammatory parameters such as CRP and ESR was observed [22].

Mediterranean diet

The Mediterranean diet (MD) consists of large amounts of vegetables, fruits, legumes, nuts, olive oil, and fish. It is associated with beneficial effects on inflammatory and chronic diseases. Epidemiological and clinical studies on the role of the Mediterranean diet in RA are inconclusive.

Potential mechanisms of the Mediterranean diet's effect on RA include: reduction of inflammation thanks to polyunsaturated fatty acids (omega-3), polyphenols (olive oil, vegetables, fruit), fiber, better control of body weight and metabolic profile, and reduced intake of pro-inflammatory saturated fats and processed foods. Genetic studies suggest that the MD may modify the inflammatory response in RA [23].

Several cross-sectional and cohort studies have shown that higher adherence to MD may be correlated with lower disease activity (lower DAS28), less perceived impact of the disease, and better functioning in some populations. However, some studies have not found a significant correlation [24].

A cohort analysis (UK Biobank) with meta-analysis showed that higher adherence to the Mediterranean diet was associated with a reduced risk of developing RA, suggesting a protective effect of the MD against RA incidence [25].

The EIRA study showed a lower risk of developing RA when following a Mediterranean diet only among men and in the form of seropositive RA. No significant association was found in the female population [26].

The 2023 MADEIRA study determined the impact of a 12-week intervention involving the introduction of a personalized Mediterranean diet and physical activity plan on the health of women with RA compared to standard care. The beneficial effects of a MD-based lifestyle in women with RA were demonstrated. A reduction in disease activity (DAS28) was observed 12 weeks after the start of the study. [27]

A randomized study from 2025 combining a Mediterranean diet with an exercise program in women with RA showed improvement in pain, muscle strength, function, and a decrease in CRP in the group using the Mediterranean diet and exercise compared to exercise alone. This suggests that the MD may be a beneficial addition to a rehabilitation program [28].

The heterogeneity of the studies (different indicators for assessing adherence to the Mediterranean diet, short observation period, small sample sizes) limits the possibility of formulating clear recommendations regarding the use of the Mediterranean diet in RA [25].

Ketogenic diet

The ketogenic diet (KD) is a low-carbohydrate, high-fat nutritional strategy that leads to increased concentrations of ketone bodies, mainly β-hydroxybutyrate (BHB). Currently, this diet is gaining interest as a potential modifier of inflammatory processes in autoimmune diseases [29].

β-hydroxybutyrate (BHB) is a signal that regulates inflammation—it inhibits the NLRP3 pathway, affects the activation of immune cells, and modulates the production of proinflammatory cytokines. Preclinical studies in animals have shown that BHB can alleviate the expected changes associated with arthritis and reduce tissue damage. However, the effect depends on the disease model, dose, and exposure time [30].

Animal models of inflammation (arthropathy models) have repeatedly shown that KD reduces allodynia and inflammatory markers, suggesting a potential analgesic and anti-inflammatory effect [31].

Reviews indicate that fasting and low-carbohydrate diets (including ketogenic diet) may reduce inflammatory symptoms in RA and other rheumatic diseases, although most data come from short-term studies or small trials [29].

Reports describing very-low-calorie ketogenic diets (VLCKD) in rheumatic diseases are mostly case reports. They show improvement in pain symptoms and inflammatory markers in overweight/obese patients with joint diseases, but it is difficult to separate the effect of weight loss from the metabolic effects of ketones [32].

The ketogenic diet can reduce insulin resistance, lower leptin levels, and modify the adipokine profile — mechanisms involved in the modulation of systemic inflammation, which potentially influence the development and course of RA. Changes in the gut microbiota observed with restrictive low-carbohydrate diets can have a dual effect: on the one hand, they can have a beneficial effect on inflammation; on the other hand, fiber restriction in inadequately balanced KD may negate these benefits [33].

The observed benefits are likely due to a combination of effects: the direct action of BHB, weight loss, and improved metabolic parameters [34]. Studies involving larger clinical groups and longer follow-up periods are needed.

Vegan diet

Studies have shown that a vegan diet has a beneficial effect on RA activity due to its high antioxidant content and low saturated fat content [35]. Other analyses have shown a reduction in subjective pain perception in patients following a vegan diet, but no significant improvement in disease activity [36]. More detailed research is needed in this area, as well as standardization of the vegan diet studied in terms of its vitamin and omega-3 content.

Gluten-free diet

Gluten is a mixture of plant proteins (gliadins, glutenins) found naturally in wheat, rye, barley, and other grains. Celiac disease is an autoimmune disease associated with intestinal inflammation resulting from exposure to gluten. A link between celiac disease and other autoimmune diseases, including rheumatoid arthritis, has been confirmed. In patients with celiac disease, exposure to gluten activates the immune system and autoimmunity, leading to inflammation and increased intestinal permeability. One study showed that a gluten-free vegan diet followed for one year by people with RA reduced disease activity. However, there is no clear evidence that gluten exacerbates RA. More research on this topic is needed [33,37].

Omega-3 fatty acids

Omega-3 fatty acids (EPA, DHA) act as precursors of inflammatory mediators and can weaken and modulate the autoimmune inflammatory response [38]. EPA and DHA reduce the production of pro-inflammatory eicosanoids, e.g., PGE2, LTB4, by competing for cyclooxygenase and lipoxygenase enzymes [38]. Omega-3 fatty acids are precursors of resolvins, protectins, and maresins, which support the termination of the inflammatory response and tissue repair [39]. They also modulate the function of antigen-presenting cells, reduce the production of TNF-α, IL-1β, IL-6, and affect the Th17/Treg balance in in vitro and in vivo models [38].

Observational data and prospective analyses suggest that higher EPA/DHA intake may be associated with a lower risk of developing certain autoimmune diseases. In the VITAL clinical trial and secondary analyses, a signal suggesting a reduction in the incidence of autoimmune diseases with omega-3 supplementation was observed, although the results for RA alone are mixed and often do not reach statistical significance. The evidence for the protective effect of omega-3 in preventing RA is inconclusive [40].

Meta-analyses have shown that omega-3 supplementation can reduce pain severity and morning stiffness duration in patients with RA. These changes tend to be moderate, and the effect depends on the dose and duration of supplementation. A reduction in the number of painful and swollen joints and an improvement in disease activity indices (DAS28) have been demonstrated after supplementation, although the effects vary between studies. Systematic reviews indicate statistically significant but clinically moderate benefits.

Some studies have shown a reduction in the need for non-steroidal anti-inflammatory drugs (NSAIDs) in patients taking omega-3 fatty acids, which is important in reducing the side effects of painkillers [41]. A meta-analysis of randomized controlled trials (RCTs) showed improvement in eight markers associated with disease activity. After omega-3 intake, leukotriene B4 levels decreased and a significant improvement in blood triacylglycerol concentrations was observed. However, the authors point to the moderate quality of the evidence and the heterogeneity of the studies [42].

A review of the literature showed that omega-3 fatty acids can prevent or alleviate experimental arthritis in mice and bring clinical benefits in RA, such as a reduction in the number of swollen and painful joints [38].

Reviews of the effects of dietary interventions with or without omega-3 fatty acids have confirmed that combined dietary changes and supplementation are beneficial in terms of disease activity, although it is difficult to separate the specific effect of omega-3 from broader dietary changes [43].

An improvement in symptoms and a reduction in inflammation markers were observed, confirming the clinical use of omega-3 as an adjunctive therapy alongside standard DMARDs [44].

The heterogeneity of studies (different doses, sources of omega-3, duration, endpoints) makes it difficult to standardize recommendations. Most studies on the role of omega-3 acids in RA focus on short-term symptomatic effects. There is a lack of large, long-term studies evaluating the impact on RA progression and long-term outcomes. Differences in populations, concomitant DMARD and biologic therapy, and standards of care influence the detectability of the effect of omega-3 fatty acids [41].

ADIRA- Anti- inflammatory Diet in Rheumatoid Arthritis

The ADIRA diet assumes high consumption of omega-3 fatty acids, but instead of supplementation, it introduces large amounts of fish [45]. High EPA and DHA content has only been demonstrated in the case of unfried fish consumption. RA patients who consumed unfried fish at least twice a week had a significantly lower DAS28-CRP disease activity index compared to patients who did not consume fish or ate it less than once a month. It has been shown that the higher the proportion of fish in the diet of RA patients, the greater the decrease in DAS28-CRP [46].

Taking fish oil supplements also lowers DAS28-CRP. The dose is important. In patients taking high doses of fish oil (EPA + DHA 5.5 g/day), the time to remission was significantly shorter compared to patients supplementing with low doses (EPA + DHA 0.4 g/day). However, general differences in the lifestyles of the study participants cannot be ruled out [46].

Vitamin D3

Vitamin D3 is known not only for its role in calcium and bone homeostasis, but also as a regulator of the immune system—it affects the function of antigen-presenting cells, T and B lymphocytes, and cytokine secretion. It has many properties, including anti-inflammatory ones. Its active metabolite is 1,25-dihydroxyvitamin D (1,25(OH)2D). The hypothesis that low vitamin D levels may increase the risk of

developing autoimmune diseases, including RA, and that supplementation may modify the course of the disease, has been the subject of numerous studies [47].

The active form of vitamin D modulates the immune response, reducing antigen presentation, shifting the T cell profile towards greater regulation (increased Treg), reducing the pro-inflammatory effect (Th17), and inhibiting the production of selected pro-inflammatory cytokines (e.g., IL-17, TNF-α). Local conversion of 25(OH)D to its active form in immune tissues suggests the importance of 25(OH)D availability for modulating the immune response in the joint microenvironment [47].

It has been observed that people with RA often have vitamin D deficiency or suboptimal 25(OH)D levels. Studies focusing specifically on early RA have shown that lower vitamin D concentrations correlate with higher disease activity, higher inflammation indices, and poorer radiological prognosis. However, the results are inconsistent, and in some studies the correlation has not been confirmed. The differences may be due to demographic and geographic factors, the definition of deficiency, and the timing of measurement [48].

Meta-analyses and randomized clinical trials do not provide clear results. Some meta-analyses have shown that vitamin D supplementation improves parameters such as pain intensity, number of tender joints, and DAS28, especially at higher doses or longer durations of therapy. Other studies indicate a limited or no significant effect of vitamin D on RA. The latest studies from 2024–2025 indicate a beneficial effect of supplementation (e.g., 4000 IU/d for 6 months) on DAS28 and VAS in selected patients, but the authors emphasize the need for further research to determine the optimal dose and target groups [49].

Subgroup analyses suggest that patients with documented 25(OH)D deficiency prior to supplementation and those with early RA or higher disease activity may derive greater clinical benefit. In addition, higher doses and longer duration of therapy are more often associated with observed improvement, although they also increase the need for safety and calcium level monitoring [48].

In clinical practice, it seems reasonable to routinely test 25(OH)D levels in patients with RA and to correct deficiencies according to local guidelines, as well as to monitor calcium levels at high doses. Randomized clinical trials with clear inclusion criteria (e.g., confirmed vitamin D deficiency), standardized doses, and longer radiological follow-up are needed to assess the impact on RA progression [50].

Polyphenols

Polyphenols are water-soluble compounds of plant origin. They have antioxidant properties. They are found in greater quantities in green tea than in black tea. Green tea is known for its immune-modulating properties through its effect on pro-inflammatory cytokines. This suggests that it is a better choice for people with RA [51].

As polyphenols are poorly absorbed and broken down by bacteria in the gut microbiome, research is needed on their interaction with the composition of the gut microbiome in RA. The metabolites produced as a result of decomposition are also responsible for the biological activity of polyphenols in the diet. However, more detailed research is needed on their role in the mechanism of RA [52].

Conclusions

The collected data indicate the potential benefits of anti-inflammatory diets and products on RA activity and treatment, but their effect is not universal in all patients.

Several population-based and cohort studies suggest an inverse relationship between fiber intake and the risk of developing RA, as well as an association between higher fiber intake and lower markers of inflammation. However, in patients with a specific microbiological profile (presence of Prevotella copri), a high intake of certain types of fiber could cause adverse effects. Further randomized studies are needed to evaluate the long-term clinical outcomes of fiber use in patients with RA and to formulate specific therapeutic recommendations [53].

A large proportion of observational studies have shown that the Mediterranean diet is associated with a lower risk of RA and better clinical outcomes in people with RA. Some studies have not shown a significant association with disease activity. Further RCTs and well-designed cohort studies are needed to confirm the strength and mechanisms of the effects of the Mediterranean diet in RA [25].

Research points to the immunomodulatory mechanisms of the ketogenic diet (BHB, changes in microbiota, reduction of adipokines and insulin resistance) and the potential clinical effect of reducing disease activity, although evidence from randomized, large controlled trials is still limited. Larger, well-designed clinical trials and long-term follow-up are needed to determine the efficacy and safety of the ketogenic diet in patients with RA. In clinical practice, consideration of a ketogenic diet in patients with RA should be

individualized, supervised (monitoring of metabolism, lipid profile, nutritional status), and ensure adequate intake of fiber and minerals.

Clinical studies have shown that high intake or supplementation of omega-3 fatty acids (EPA, DHA) reduces the number of painful and swollen joints, reduces inflammation markers, and the need for NSAIDs. Available evidence indicates that omega-3 fatty acids have a solid biological basis for anti-inflammatory action and that they can clinically reduce pain, morning stiffness, and the number of painful/swollen joints in patients with RA. In clinical practice, omega-3 fatty acids supplementation may be considered as an adjunct to drug therapy to reduce symptoms and the need for NSAIDs, taking into account possible drug interactions and the quality of the preparation. Further well-designed randomized controlled trials (RCTs) with long follow-up periods and standardization are needed to assess the impact on long-term outcomes, radiographic progression, and the risk of comorbidities [54].

Vitamin D3 has anti-inflammatory properties. Studies have shown that people with RA and those at increased risk of developing RA have low levels of this vitamin. Some meta-analyses indicate an improvement in DAS28 and a reduction in pain after supplementation, while others show no significant changes, which may be due to heterogeneity in doses and periods of administration. Well-designed, large randomized controlled trials with clearly defined criteria (especially measurement of baseline deficiency) and determination of the optimal dose and duration of therapy are needed [55]. The results of studies on the role of vitamin D in the prevention and treatment of RA are unclear. It is very difficult to measure the total dose of vitamin D that a patient has received as a result of exposure to sunlight [33].

The collected data Indicate that dietary modifications, IIing Increased Intake of omega-3 fatty acids, polyphenols, and vitamin D supplementation, may contribute to reducing inflammation and improving symptoms in some patients with RA. However, the results of the studies are not conclusive. This is due to differences in the composition of the diets of the study participants, short observation periods, small sample sizes, and the lack of uniform endpoints (DAS28, CRP, ESR, VAS). Well-designed, large randomized controlled trials with clearly defined entry criteria, longer follow-up periods, and larger study groups are needed to clearly determine whether any diet or food product can be widely used as an adjunctive therapy for RA.

Disclosure

Author's Contribution

Conceptualization: Agnieszka Bajkacz and Izabela Polakowska; Methodology: Agnieszka Bajkacz, Izabela Polakowska, Agnieszka Roszyk, Kamil Orczyk, Alicja Dankowska, Małgorzata Zielonka, Zuzanna Głowacka, Jakub Białek, Patrycja Stygar and Agata Bęben; Formal analysis: Agnieszka Bajkacz, Izabela Polakowska, Alicja Dankowska and Agata Bęben; Resources: Agnieszka Bajkacz, Izabela Polakowska, Agnieszka Roszyk, Kamil Orczyk, Zuzanna Głowacka, Jakub Białek and Patrycja Stygar; Writing- original Draft Preparation, Agnieszka Bajkacz, Izabela Polakowska, Agnieszka Roszyk, Kamil Orczyk, Alicja Dankowska, Małgorzata Zielonka, Zuzanna Głowacka, Jakub Białek, Patrycja Stygar and Agata Bęben; Writing-Review and Editing, Agnieszka Bajkacz, Alicja Dankowska, Małgorzata Zielonka, Zuzanna Głowacka, Jakub Białek and Agata Bęben; Visualization: Agnieszka Bajkacz, Izabela Polakowska, Agnieszka Roszyk, Kamil Orczyk, Małgorzata Zielonka and Patrycja Stygar; Supervision: Agnieszka Bajkacz;

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