ABRAN ASSOCIAÇÃO BRASILEIRA DE NUTROLOGIA
International Journal of Nutrology
(Official Journal of the ABRAN - Brazilian Association of Nutrology)

REVIEW ARTICLE

DOI: 10.54448/ijn25S311

Nutrological management of meta-inflamation in patients with obesity: a systematic review

Carmen Melo do Vale[1*], Ana Angélica Nogueira Lima[1]

[1] Potiguar University - Medical School. Natal, Rio Grande do Norte, Brazil.

*Corresponding author: Dr. Carmen Melo do Vale.
Potiguar University- Medical School.
Natal, Rio Grande do Norte, Brazil.
E-mail: carmenmelodovale@gmail.com

DOI: https://doi.org/10.54448/ijn25S311
Received: 05-17-2025; Revised: 07-22-2025; Accepted: 07-29-2025; Published: 07-30-2025; IJN-id: e25S311

Editor: Dr. Luis Alberto Moreno Aznar, MD, Ph.D.

Abstract

Introduction: Obesity stands out as a multifactorial disease that can cause several public health problems. There are more than 2.2 billion overweight and obese people in the world, and Brazil is in fifth place in the world ranking. A healthy nutritional status promotes immune function and can prevent the onset of a serious inflammatory process and severe infections. **Objective:** It was to highlight the main clinical considerations of nutritional and dietary regulation in obese patients with marked inflammatory processes and meta-inflammation through a systematic review. **Methods:** The systematic review rules of the PRISMA Platform were followed. The research was carried out from May to June 2025 in Scopus, PubMed, Science Direct, Scielo, and Google Scholar databases. The quality of the studies was based on the GRADE instrument and the risk of bias was analyzed according to the Cochrane instrument. **Results and Conclusion:** A total of 127 articles were found. A total of 42 articles were fully evaluated and 29 were included and developed in the present systematic review study. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 32 studies at high risk of bias and 25 studies that did not meet the GRADE. Research has shown that unbalanced eating patterns, such as the Western diet, rich in simple sugars, refined carbohydrates, and saturated and trans fatty acids, lead to chronic inflammatory responses, increased adipose deposition, and future comorbidities associated with overweight and obesity. Calorie restriction decreased CRP in obese patients and diet administration over 12 weeks had a beneficial effect. Furthermore, obese patients with antioxidant supplementation had lower values of BMI, waist circumference, fasting blood glucose level, and assessment of the homeostasis model of insulin resistance when compared to the placebo group, as well as having lower levels of total cholesterol, triglycerides, LDL, malondialdehyde and tumor necrosis factor-alpha. n-3 PUFA supplementation can significantly reduce serum C-reactive protein (CRP), tumor necrosis factor α (TNFα), and interleukin 6 (IL-6) concentrations.

Keywords: Obesity. Inflammatory processes. Meta-inflammation. Diet therapy. Nutritional regulation.

Introduction

Obesity stands out as a multifactorial disease that can cause several public health problems **[1]**. Currently, more than 30% of the world's population is overweight or obese. By 2025, it is estimated that more than 60% of the world's population will be overweight or obese **[1]**. Currently, there are 2.0 billion people who are overweight or obese in the world, and Brazil ranks fifth in the world, with an estimated 18.0 million people **[3]**. In the United States, the prevalence of obesity is greater than 30.0% for both sexes, and obesity is the cause of death for 2.8 million people per year, affecting 26% of adults **[4]**. In Europe, it is estimated that 10 to 20% of men and 15 to 25% of women are obese **[5]**.

In this context, the cause of obesity is a complex

relationship between biological, psychosocial, and behavioral factors, including genetic makeup, socioeconomic status, and cultural influences **[6]**. Furthermore, obesity has been associated with microorganisms, epigenetics, increased maternal age, increased fertility, lack of sleep, endocrine disruptors, pharmaceutical iatrogenesis, and intrauterine and intergenerational effects **[6,7]**. Comorbid conditions and their treatments may also be a factor in the development of obesity **[8]**.

In this regard, it has been postulated that a healthy nutritional status promotes immune function and may prevent the onset of a severe inflammatory process and severe infections, especially during pandemics such as COVID-19 **[9-12]**. Thus, an optimal immune response depends on adequate diet and nutrition to keep infections under control. For example, sufficient protein intake is crucial for optimal antibody production. Furthermore, low levels of micronutrients, such as vitamin A or zinc, are associated with an increased risk of infection, as this deficiency promotes inflammatory processes and oxidative stress. Furthermore, dietary constituents with anti-inflammatory and antioxidant properties include vitamin C, vitamin E, carotenoids, and polyphenols **[13]**.

Thus, several of these dietary elements can interact with transcription factors such as NF-kB and Nrf-2. An important example is vitamin D, which protects tissue against viral cellular infection through angiotensin-converting enzyme 2 (ACE2). Dietary fiber and short-chain fatty acids have also shown anti-inflammatory effects **[13]**.

Therefore, this study highlighted the key clinical considerations of nutritional and dietary regulation in obese patients with marked inflammatory processes and meta-inflammation through a systematic review.

Methods

Study Design

The rules for a systematic review of the PRISMA Platform (Transparent Reporting of Systematic Reviews and Meta-Analysis - HTTP://www.prisma-statement.org/) were followed.

Data Sources and Search Strategy

The search strategies for this systematic review were based on the keywords (DeCS/MeSH Terms): "*Obesity. Inflammatory processes. Meta-inflammation. Dietary therapy. Nutritional regulation*" The search was conducted from May to June 2025 in the Scopus, PubMed, Science Direct, Scielo, and Google Scholar databases. Furthermore, a combination of keywords with the Boolean operators "OR," "AND," and "NOT" was used to target scientific articles of interest.

Study Quality and Risk of Bias

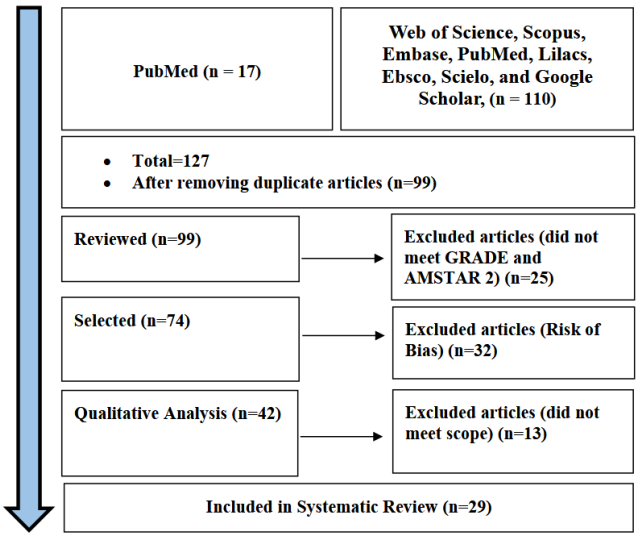
Study quality was based on the GRADE instrument. Methodological quality was assessed using the AMSTAR-2, and risk of bias was analyzed using the Cochrane tool.

Results and Discussion

Summary of Findings

A total of 127 articles were found. Initially, duplicate articles were excluded. After this process, the abstracts were evaluated and further exclusion was performed, removing articles that did not address the topic of this article, resulting in 74 articles. A total of 42 articles were evaluated in full, and 29 were included and developed in this systematic review study (Figure 1). Considering the Cochrane risk of bias tool, the overall assessment resulted in 32 studies with a high risk of bias and 25 studies that did not meet GRADE and AMSTAR-2 criteria.

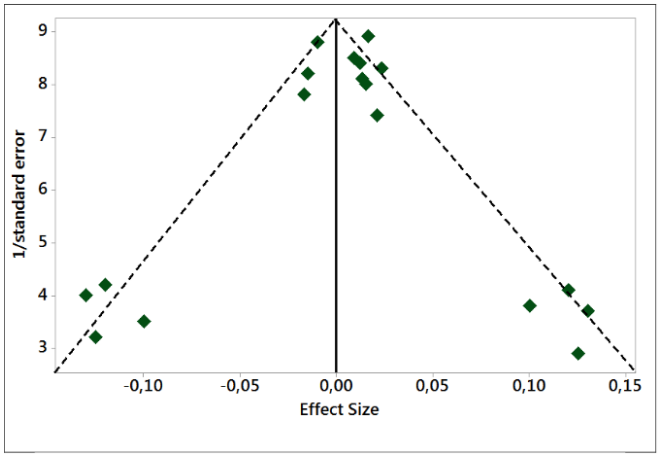
Figure 1. Flowchart of the article selection process.



Source: Own Authorship.

Figure 2 presents the results of the risk of bias of the studies using the Funnel Plot, showing the calculation of the Effect Size (Magnitude of the difference) using Cohen's d Test. The sample size was determined indirectly by the inverse of the standard error (1/Standard Error). This graph showed symmetrical behavior, suggesting no significant risk of bias, either among studies with small sample sizes (lower precision), which are shown at the bottom of the graph, or among studies with large sample sizes, which are shown at the top.

Figure 2. The symmetrical funnel plot suggests no risk of bias among the small sample size studies shown at the bottom of the graph. High-confidence and highly recommended studies are shown above the graph (Ntotal = 29 studies fully evaluated in the systematic review).



Source: Own Authorship.

Main Approaches – Meta-Inflammatory Processes in Obesity

In the setting of obesity, circulating levels of cytokines and acute-phase proteins associated with inflammation are elevated. Adipocytes secrete several cytokines and acute-phase proteins that increase the production and circulation of inflammatory factors. The inflammatory process may be due to insulin resistance and other obesity-associated disorders, such as hyperlipidemia and metabolic syndrome **[14,15]**.

There are three possibilities. The first reflects production and release from organs other than adipose tissue, primarily the liver and immune cells. The second explanation is that white adipose tissue secretes factors that stimulate the production of inflammatory markers by the liver and other organs. The third possibility is that adipocytes themselves are an immediate source of some, or many, of these inflammatory markers **[16-18]**.

In this context, it is worth highlighting that the adipokines associated with inflammatory processes are interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), leptin, and adiponectin **[17]**. Some studies have shown that low concentrations of the anti-inflammatory adipokine (adiponectin) are associated with the occurrence of various types of cancer, and high concentrations are associated with tumor growth inhibition. Others also include TNF-α, IL-6, IL-1, CC-chemokine ligand 2 (CCL2), visceral adipose-tissue-derived serine protease inhibitor (vaspin), and retinol-binding protein 4 (RBP4) **[17]**.

In this regard, adipokines have a significant impact on various bodily functions. Notable in this case are control of food intake and energy balance, the immune system, insulin sensitivity, angiogenesis, blood pressure, lipid metabolism, and body homeostasis, all of which are strongly correlated with cardiovascular disease **[15]**. Furthermore, high plasma adiponectin concentrations are associated with a reduced risk of myocardial infarction in men **[15]**.

In this context, obesity induces a change in the macrophage profile, with an increase in the M1 (pro-inflammatory) phenotype. This effect corresponds to an upregulation of inflammatory genes and a downregulation of anti-inflammatory genes **[19,20]**. Furthermore, the primary increase in the inflammatory response in obese patients functions as a predictor of the hyperinflammatory state observed in COVID-19. Therefore, this primary increase can be amplified by SARS-CoV-2 infection, increasing the production of cytokines such as TNF-α, IL-1, and IL-6 **[21]**.

As a corollary to this, meta-inflammation describes the combination of inflammation and metabolic changes that occur in the bodies of obese patients **[5]**. Several toxic mediators that contribute to the inflammatory state and tissue damage are present in obesity, such as free fatty acids (FFAs), toxic lipid derivatives such as diacylglycerol, toxic nitric oxide metabolites, and inflammatory mediators such as C-reactive protein, cytokines, chemokines, macrophages, and TNF-α. The imbalance in inflammatory mediators induced by excess nutrients underlies meta-inflammation in obesity. Obesity can cause multiple organ dysfunction, and meta-inflammation leads to myocardial dysfunction through direct damage to inflammatory mediators, as well as through dysfunction of other organs **[7]**.

In this scenario, obese patients stand out among the young population who develop severe COVID-19. The unfavorable outcome is possibly because these patients have a more inflamed and hyperreactive endothelium, which, when stimulated by SARS-CoV-2, exhibits an excessive response, leading to hyperinflammation with a cytokine storm. As a corollary to the exacerbated inflammatory process, the coagulation cascade is dysregulated, leading to hypercoagulability. Therefore, the endothelial dysfunction caused by SARS-CoV-2 explains why patients with blood vessel-related comorbidities such as cardiovascular disease, hypertension, diabetes, and obesity are more likely to develop severe COVID-19 symptoms, even death **[21]**.

This was also reported in a multicenter study involving 5,700 hospitalized patients in the New York metropolitan area. Obesity was described as the second most common comorbidity, present in approximately 40% of COVID-19 patients. During

hospitalization of the 2634 patients, 14.2% were treated in the ICU, and 12.2% received invasive mechanical ventilation. Mortality for those requiring mechanical ventilation was 88.1% **[22]**.

Other authors observed the relationship between obesity and the development of severe respiratory manifestations when analyzing 103 patients hospitalized with COVID-19. They reported that 47% of these patients were obese. In this study, patients with a BMI of 30 kg/m^2 or higher were among those most in need of ICU admission and mechanical ventilation **[23]**.

Nutrological Regulation of Meta-Inflammation

Nutritional status can have a significant impact on an individual's overall health, reducing comorbidities and susceptibility to infections such as COVID-19. However, according to the WHO, there is still no single food or natural remedy with proven scientific evidence to prevent COVID-19 infections **[24]**. Despite this, based on previous studies, nutritional status is known to play a significant role in patient outcomes **[25]**. Therefore, a diet characterized by anti-inflammatory properties is essential to benefit from or prevent COVID-19 **[26-29]**.

In this sense, evidence suggests that dietary patterns and individual nutrients can influence systemic markers of immune function. Therefore, maintaining nutritional status at this time is important, given that the fight against COVID-19 can last a long time. To maintain a healthy immune system, special attention should be paid to maintaining a healthy diet, lifestyle, and exercise regimen **[30]**.

Nutritional deficiencies in calcium, vitamin C, vitamin D, folate, and zinc exist among elderly populations **[31]**, making them immunocompromised **[32]**. Therefore, a healthy and balanced diet can provide the necessary macro and micronutrients, prebiotics, probiotics, and symbiotics in older adults, which can restore and maintain immune cell function **[33]**.

In this context, a review study analyzed the usefulness of early micronutrient intervention, focusing on zinc, selenium, and vitamin D, to alleviate the rise of COVID-19. The results revealed direct evidence of associations between zinc, selenium, and vitamin D and COVID-19. Adequate zinc, selenium, and vitamin D are essential for resistance to other viral infections, immune function, and reduced inflammation **[34]**.

In this scenario of nutritional triggers to promote immune-boosting responses, as well as improving the performance of mitosis, meiosis, and all cellular functions, all of this directly integrates with the body's energy balance and nutritional status. Metabolic byproducts and substrates that regulate epigenetic and signaling pathways are considered to have an instructive, rather than observer, role in regulating cell fate decisions. Metabolism encompasses the interactions between diet, the microbiome, and cellular enzymatic processes that generate the chemical pathways necessary to sustain life. Furthermore, endogenous metabolites and dietary nutrients can directly influence epigenetic enzymes. Epigenetic modifications to DNA and histone proteins alter cell fate by controlling chromatin accessibility and downstream gene expression patterns. Thus, most substrates and cofactors for chromatin-modifying enzymes are derived from metabolic pathways such as the tricarboxylic acid cycle, methionine cycle, folate cycle, glycolysis, β-oxidation, and the hexosamine pathway **[35]**.

In addition to the connection between metabolism and epigenetic pathways, nutrients can impact cellular state by modulating signaling pathway activity. A clear example is through the mechanistic target of rapamycin (mTOR) signaling pathway and, in particular, mTOR complex 1 (mTORC1), which regulates cell growth only when nutrients and growth factors are present. Depletion of specific nutrients, including arginine, leucine, and S-adenosyl methionine, prevents growth factor-induced mTORC1 activation by blocking Rag GTPase-mediated mTORC1 recruitment to the lysosome, where it can be activated by Rheb GTPase **[36]**.

Another way nutrients are sensed to impact cellular state is through AMP-activated protein kinase (AMPK), which, at low cellular ATP levels, phosphorylates substrates to restore the cell's energy balance and, in the process, regulates cell growth and autophagy. Furthermore, transcription factors can be directly regulated by metabolites. Tryptophan kynurenine is an endogenous agonist for the aryl hydrocarbon and alpha-ketoglutarate (α-K) receptor that binds to and activates IKKβ and initiates NF-κβ signaling **[35]**.

Thus, nutritional health directly affects the human gut microbiota, impacting metabolism and the immune system for tissue regeneration. Recent discoveries have focused on the role of the "nutritional microbiota" in mechanisms involved in tissue regeneration, particularly skin, liver, bone, and nervous system regeneration **[36]**.

In this regard, in the inflammatory phase, vitamin A increases cytokine release, bromelain and amino acids prevent prolonged inflammatory events, and vitamin C increases neutrophil migration and lymphocyte activation. In the proliferative phase, vitamin C is necessary for collagen synthesis, and glucosamine

increases hyaluronic acid production. Vitamin A promotes epithelial cell differentiation. Zinc is necessary for DNA and protein synthesis and cell division. In the remodeling phase, amino acids and proteins play a key role in wound scar stabilization **[37]**.

Age-related reduction in muscle repair efficiency contributes to the development of sarcopenia. Nutrients such as amino acids, polyunsaturated fatty acids, polyphenols, and vitamin D can improve skeletal muscle regeneration by targeting key functions of immune cells, muscle cells, or both **[38]**.

As supporting literature, a randomized, double-blind, placebo-controlled clinical trial evaluated the effects of propolis supplementation for 8 weeks (510 mg per day) on glucose homeostasis, lipid profile, liver function, anthropometric indices, and meta-inflammation in patients with non-alcoholic fatty liver disease (NAFLD). In this trial, 44 patients with NAFLD confirmed by ultrasound findings were randomly allocated to the "propolis" (n=23) or "placebo" (n = 21) group, along with a group on a calorie-restricted diet (-500 kcal d-1) for 8 weeks. Fasting serum levels of metabolic factors, liver enzymes, and inflammatory factors, as well as anthropometric indices, food intake, and appetite status, were assessed pre- and post-intervention. The liver fibrosis score, homeostasis model assessment of insulin resistance (HOMA-IR), and quantitative insulin sensitivity check index (QUICKI) were also calculated. Weight, body mass index (BMI), waist and hip circumferences, and waist-to-height ratio decreased significantly in both groups ($p<0.001$), while waist-to-hip ratio ($p=0.006$) and serum total cholesterol level ($p=0.038$) decreased only in the propolis arm. Fasting blood glucose ($p=0.037$), serum insulin level ($p=0.040$), HOMA-IR ($p=0.007$), sweet tooth ($p=0.005$), and NAFLD fibrosis score ($p=0.013$) decreased significantly in the propolis group compared to the placebo group, post-intervention, after adjusting for baseline values and potential confounders. However, QUICKI showed a significant increase ($p=0.015$) in the propolis arm compared to placebo at the study endpoint. Although there were significant reductions in serum levels of inflammatory factors, including tumor necrosis factor-α (TNF-α), toll-like receptor-4 (TLR-4), and monocyte chemoattractant protein-1 (MCP-1), as well as liver enzymes and fatty liver severity, the differences between groups were not statistically significant after adjusting for potential confounders **[39]**.

Chronic inflammation in obese patients can be controlled through a calorie-restricted diet, characterized by a reduction in C-reactive protein (CRP). Review studies based on randomized clinical trials have evaluated the role of this diet in CRP. The results showed that calorie restriction reduced CRP in obese patients, and that diet administration for more than 12 weeks had a beneficial effect **[40,41]**.

A meta-analysis study demonstrated heterogeneity in the beneficial effects of antioxidant supplementation in obese adults, exploring the differential effects of antioxidant supplementation on basic indicators of obesity, lipid metabolism, systemic antioxidant capacity, inflammatory biomarkers, and liver function. A total of 30 studies were included in this study, with a sample of 845 obese patients in the antioxidant supplementation group and 766 obese patients in the placebo control group. The meta-analysis showed that obese patients receiving antioxidant supplementation had lower BMI, waist circumference, fasting blood glucose levels, and homeostasis model assessment of insulin resistance when compared to the placebo group. Furthermore, obese patients receiving antioxidant supplementation had lower levels of total cholesterol, triglycerides, LDL, malondialdehyde, and tumor necrosis factor alpha when compared to the placebo group. Furthermore, obese patients receiving antioxidant supplementation had higher levels of HDL and superoxide dismutase when compared to the placebo group. Antioxidant supplementation did not affect other parameters analyzed, including waist-to-hip ratio, leptin, fat mass, interleukin-6, C-reactive protein, alanine transaminase, and aspartate transaminase in obese patients **[41]**.

Finally, a study with 32 meta-analyses demonstrated that n-3 PUFA supplementation significantly reduced serum C-reactive protein (CRP), tumor necrosis factor-α (TNFα), and interleukin-6 (IL-6) concentrations **[42]**.

Conclusion

It was concluded that unbalanced dietary patterns, such as the Western diet, rich in simple sugars, refined carbohydrates, saturated and trans fatty acids, lead to chronic inflammatory responses, increased adipose deposition, and future comorbidities associated with overweight and obesity. Calorie restriction decreased C-reactive protein in obese patients, and diet administration for more than 12 weeks had a beneficial effect. Furthermore, obese patients with antioxidant supplementation had lower BMI, waist circumference, fasting glucose levels, and homeostasis model assessment of insulin resistance when compared to the placebo group, as well as lower levels of total cholesterol, triglycerides, LDL, malondialdehyde, and tumor necrosis factor alpha. n-3 PUFA supplementation can significantly reduce serum C-reactive protein, tumor necrosis factor alpha (TNFα), and interleukin 6 (IL-6) concentrations.

CRediT

Author contributions: **Conceptualization-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Data curation-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Formal Analysis-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Investigation-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Methodology-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Project administration-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Supervision-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Writing - original draft-** Carmen Melo do Vale, Ana Angélica Nogueira Lima; **Writing-review & editing-** Carmen Melo do Vale, Ana Angélica Nogueira Lima.

Acknowledgment

Not applicable.

Ethical Approval

Not applicable.

Informed Consent

Not applicable.

Funding

Not applicable.

Data Sharing Statement

No additional data are available.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate[@].

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

References

1. Finkelstein EA, Khavjou OA, Thompson H, Trogdon JG, Pan L, Sherry B, et al. Obesity and severe obesity forecasts through 2030. Am J Prev Med 2012;42:563–570.
2. WHO–World Health Organization. Available at: https://www.sbcbm.org.br/endoscopia-e-obesidade/ Accessed on May 21, 2025. 3. Brazilian Institute of Geography and Statistics (IBGE). Accessed on May 22, 2025. Available at: http://www.ibge.gov.br.
3. Karamitri A, Jockers R. Melatonin in type 2 diabetes mellitus and obesity. Nat Rev Endocrinol. 2019 Feb;15(2):105-125. doi: 10.1038/s41574-018-0130-1.
4. Schetz M, De Jong A, Deane AM, et al. Obesity in the critically ill: a narrative review. Intensive Care Med. 2019;45(6):757-769. doi:10.1007/s00134-019-05594-1.
5. BRAZILIAN ASSOCIATION FOR THE STUDY OF OBESITY AND METABOLIC SYNDROME. Brazilian Obesity Guidelines 2016. 4th ed. São Paulo: ABESO, 2016. Available at: Accessed on June 25, 2025.
6. Apovian CM. Obesity: definition, comorbidities, causes, and burden. Am J Manag Care. 2016;22(7 Suppl):s176-s185.
7. Andolfi C, Fisichella PM. Epidemiology of Obesity and Associated Comorbidities. J Laparoendosc Adv Surg Tech A. 2018; 28(8):919-924. doi:10.1089/lap.2018.0380.
8. Wu D, Lewis ED, Pae M, Meydani SN. Nutritional modulation of immune function: Analysis of evidence, mechanisms, and clinical relevance. Front. Immunol. 2019;9:9. doi: 10.3389/fimmu.2018.03160.
9. Grant WB, Lahore H, McDonnell SL, Baggerly CA, French CB, Aliano JL, Bhattoa H.P. Evidence that vitamin d supplementation could reduce risk of influenza and COVID-19 infections and deaths. Nutrients. 2020;12:988. doi: 10.3390/nu12040988.
10. Childs CE, Calder PC, Miles EA. Diet and immune function. Nutrients. 2019;11:1933. doi: 10.3390/nu11081933.
11. Calder PC, Carr AC, Gombart AF, Eggersdorfer M. Optimal nutritional status for a well-functioning immune system is an important factor to protect against viral infections. Preprints. 2020;12:1181.
12. Iddir M, Brito A, Dingeo G, Fernandez Del Campo SS, Samouda H, La Frano MR, Bohn T. Strengthening the Immune System and Reducing Inflammation and Oxidative Stress through Diet and Nutrition: Considerations during the COVID-19 Crisis. Nutrients. 2020 May 27;12(6):1562. doi: 10.3390/nu12061562.

13. Tutuian R. Obesity and GERD: Pathophysiology and effect of bariatric surgery. Curr Gastroenterol Rep 2011;13:205-212.
14. Sood A, Shore SA. Adiponectin, leptin, and resistin in asthma: Basic mechanisms through population studies. J Allergy (Cairo) 2013;2013:785835.
15. Jensen P, Skov L. Psoriasis and Obesity. Dermatology. 2016;232(6):633-639. doi:10.1159/000455840.
16. Lauby-Secretan B, Dossus L, Marant-Micallef C, His M. Obésité et cancer [Obesity and Cancer]. Bull Cancer. 2019;106(7-8):635-646. doi:10.1016/j.bulcan.2019.04.008.
17. Landecho MF, Tuero C, Valentí V, Bilbao I, de la Higuera M, Frühbeck G. Relevance of Leptin and Other Adipokines in Obesity-Associated Cardiovascular Risk. Nutrients. 2019;11(11):2664. Published 2019 Nov 5. doi:10.3390/nu11112664.
18. Ghanim H, Aljada A, Hofmeyer D, Syed T, Mohanty P, Dandona P. Circulating Mononuclear Cells in the Obese Are in a Proinflammatory State. Circulation [Internet]. 21 set 2004 [capturado 8 jun 2020]; 110(12):1564-1571. DOI 10.1161/01.CIR.0000142055.53122.FA. Available on: https://www.ahajournals.org/doi/10.1161/01.cir.0000142055.53122.fa.
19. Varga Z, Flammer AJ, Steiger P, Haberecker M, Andermatt R, Zinkernagel AS, et al. Endothelial cell infection and endotheliitis in COVID-19. Lancet [Internet]. 02 maio 2020 [capturado 7 jun 2020]; 395(10234):1417-1418. DOI 10.1016/S0140-6736(20)30937-5. Available on: https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(20)30937-5/fulltext.
20. Kass DA, Duggal P, Cingolani O. Obesity could shift severe COVID-19 disease to younger ages. Lancet [Internet]. 30 abr 2020 [capturado 8 jun 2020]; 395(10236):1544- 1545. DOI 10.1016/S0140-6736(20)31024-2. Disponível em: https://www.thelancet.com/journals/lancet/article/ PIIS0140-6736(20)31024-2/fulltext
21. Becker R. COVID-19 update: Covid-19-associated coagulopathy. J Thromb Thrombolys [Internet]. 2020 [capturado 7 jun 2020]; 50:54-67. DOI 10.1007/s11239020-02134-3. Available on: https://link.springer.com/article/10.1007/s11239-02002134-3.
22. Kruglikov IL, Scherer PE. The Role of Adipocytes and Adipocyte-Like Cells in the Severity of COVID-19 Infections. Obesity (Silver Spring) [Preprint] [Internet]. 27 abr 2020 [capturado 8 jun 2020]. DOI 10.1002/ oby.22856. Available on: https://onlinelibrary.wiley.com/doi/full/10.1002/oby.22856.
23. World Health Organization Off-label Use of Medicines for COVID-19. [(accessed on 20 September 2021)]; Available online: https://www.who.int/newsroom/commentaries/detail/off-label-use-of-medicines-for-covid-19
24. Mechanick JI, Carbone S, Dickerson RN, Hernandez BJD, Hurt RT, Irving SY, Li DY, McCarthy MS, Mogensen KM, Gautier JBO, Patel JJ, Prewitt TE, Rosenthal M, Warren M, Winkler MF, McKeever L; ASPEN COVID-19 Task Force on Nutrition Research. Clinical Nutrition Research and the COVID-19 Pandemic: A Scoping Review of the ASPEN COVID-19 Task Force on Nutrition Research. JPEN J Parenter Enteral Nutr. 2021 Jan;45(1):13-31. doi: 10.1002/jpen.2036.
25. Georgousopoulou E.N., Kouli G.-M., Panagiotakos D.B., Kalogeropoulou A., Zana A., Chrysohoou C., Tsigos C., Tousoulis D., Stefanadis C., Pitsavos C. Anti-inflammatory diet and 10-year (2002–2012) cardiovascular disease incidence: The ATTICA study. Int. J. Cardiol. 2016;222:473–478. doi: 10.1016/j.ijcard.2016.08.007.
26. de Boer A., van de Worp WRPH, Hageman GJ, Bast A. The effect of dietary components on inflammatory lung diseases – a literature review. Int. J. Food Sci. Nutr. 2017;68:771–787. doi: 10.1080/09637486.2017.1288199.
27. Lago JHG, Toledo-Arruda AC, Mernak M, Barrosa KH, Martins MA, Tibério IFLC, Prado CM. Structure-activity association of flavonoids in lung diseases. Molecules. 2014;19:3570–3595. doi: 10.3390/molecules19033570.
28. Phillips CM, Chen LW, Heude B, Bernard JY, Harvey NC, Duijts L, Mensink-Bout SM, Polanska K, Mancano G, Suderman M, et al. Dietary inflammatory index and non-communicable disease risk: A narrative review. Nutrients. 2019;11:1873. doi: 10.3390/nu11081873.
29. Mattioli AV, Ballerini Puviani M. Lifestyle at time of COVID-19: How could quarantine affect cardiovascular risk. Am. J. Lifestyle Med. 2020;14:240–242. doi: 10.1177/1559827620918808.
30. Power SE, Jeffery IB, Ross RP, Stanton C, O'Toole PW, O'Connor EM, Fitzgerald GF. Food and nutrient intake of Irish community-dwelling elderly subjects: Who is at nutritional risk? J. Nutr. Health Aging. 2014;18:561–572. doi:

10.1007/s12603-014-0449-9.

31. Haase H, Rink L. The immune system and the impact of zinc during aging. Immun. Ageing. 2009;6:9. doi: 10.1186/1742-4933-6-9.

32. Gammoh NZ, Rink L. Zinc in infection and inflammation. Nutrients. 2017;9:624. doi: 10.3390/nu9060624.

33. Alexander J, Tinkov A, Strand TA, Alehagen U, Skalny A, Aaseth J. Early Nutritional Interventions with Zinc, Selenium and Vitamin D for Raising Anti-Viral Resistance Against Progressive COVID-19. Nutrients. 2020 Aug 7;12(8):2358. doi: 10.3390/nu12082358.

34. Shapira SN, Christofk HR. Metabolic Regulation of Tissue Stem Cells. Trends Cell Biol. 2020 Jul;30(7):566-576. doi: 10.1016/j.tcb.2020.04.004.

35. Shavandi A, Saeedi P, Gérard P, Jalalvandi E, Cannella D, Bekhit AE. The role of microbiota in tissue repair and regeneration. J Tissue Eng Regen Med. 2020 Mar;14(3):539-555. doi: 10.1002/term.3009.

36. Palmieri B, Vadalà M, Laurino C. Nutrition in wound healing: investigation of the molecular mechanisms, a narrative review. J Wound Care. 2019 Oct 2;28(10):683-693. doi: 10.12968/jowc.2019.28.10.683.

37. Domingues-Faria C, Vasson MP, Goncalves-Mendes N, Boirie Y, Walrand S. Skeletal muscle regeneration and impact of aging and nutrition. Ageing Res Rev. 2016 Mar;26:22-36. doi: 10.1016/j.arr.2015.12.004.

38. Nikbaf-Shandiz M, Tutunchi H, Khoshbaten M, Nazari Bonab H, Ebrahimi-Mameghani M. Propolis supplementation in obese patients with non-alcoholic fatty liver disease: effects on glucose homeostasis, lipid profile, liver function, anthropometric indices and meta-inflammation. Food Funct. 2022 Oct 20. doi: 10.1039/d2fo01280d.

39. Kemalasari I, Fitri NA, Sinto R, Tahapary DL, Harbuwono DS. Effect of calorie restriction diet on levels of C reactive protein (CRP) in obesity: A systematic review and meta-analysis of randomized controlled trials. Diabetes Metab Syndr. 2022 Mar;16(3):102388. doi: 10.1016/j.dsx.2022.102388.

40. Wang J, Liao B, Wang C, Zhong O, Lei X, Yang Y. Effects of Antioxidant Supplementation on Metabolic Disorders in Obese Patients from Randomized Clinical Controls: A Meta-Analysis and Systematic Review. Oxid Med Cell Longev. 2022 Sep 1;2022:7255413. doi: 10.1155/2022/7255413.

41. Merloto RF, Soares BR. Major microRNAs and regulation of gut microbiota in patients with obesity: a systematic review. MedNEXT J Med Health Sci [Internet]. 2024 Jan. 27 [cited 2025 Jul. 30];5(1). Available from: https://mednext.zotarellifilhoscientificworks.com/index.php/mednext/article/view/349

42. Kavyani Z, Musazadeh V, Fathi S, Hossein Faghfouri A, Dehghan P, Sarmadi B. Efficacy of the omega-3 fatty acids supplementation on inflammatory biomarkers: An umbrella meta-analysis. Int Immunopharmacol. 2022 Oct;111:109104. doi: 10.1016/j.intimp.2022.109104.

MetaScience Press
The Best Science
https://zotarellifilhoscientificworks.com/