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Short Communication

Involvement of Parasites in Host Cardiometabolic Disorders

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Abstract

Background: Parasitic diseases are considered a serious public health problem for humans due to their widespread distribution and high frequency, especially in low- to middle-income countries. However, they appear to have a benign effect on cardiometabolic diseases. Therefore, the objective of this article is to present relevant information in this regard.

Materials and Methods: A documentary review of existing information related to parasitic diseases and their role in metabolic disorders was conducted. Based on the analysis of the main aspects found, the key elements of this interaction are presented.

Results: For example, by reducing circulating pro-inflammatory cytokines and increasing anti-inflammatory cytokines, which affect the pathogenetic mechanism, nutrient absorption, and the intestinal ecosystem, parasites and their antigens may contribute to improvements in the clinical presentation of chronic metabolic diseases.

Conclusion: It is evident that there is a complex and dynamic interaction between parasitosis and cardiometabolic disorders that is beneficial to the host, a result of coevolution and clearly generalizable to the human host population.

Key Words: parasites; cardiometabolic disorders; diabetes mellitus; obesity; cytokines

Introduction

In rural and marginal urban areas of tropical and subtropical regions of low- and middle-income countries, parasitic infections represent a major public health problem (affecting approximately a quarter of the world's population) due to their endemicity in many of these countries, their widespread distribution, and their high morbidity and mortality rates. Parasitic diseases, specifically their pathogenesis, are determined by risk factors related to the environment and hygiene (access to drinking water and sanitation, humid environments, and barefoot walking habits), diet (contaminated food and untreated water), contact with animals and carriers, and individual factors (immunosuppression and malnutrition) [1-4].

Intestinal parasites have the highest prevalence, affecting 3.5 billion people worldwide, especially *Ascaris lumbricoides*, *Schistosoma haematobium*, *Trichuris trichiura*, *Ancylostoma duodenale*, *Hymenolepis nana*, *Cryptosporidium* spp., and *Entamoeba histolytica*, with 39 million disability-adjusted life years worldwide. In addition to the characteristic clinical presentations that define the disease, they are believed to participate in the genesis and/or progression of cardiometabolic disorders in the host, including obesity, metabolic syndrome, atherosclerosis, and type II diabetes mellitus (T2DM) (a chronic disorder with persistent hyperglycemia), which are associated with a lower quality of life and significant mortality [3, 5-8].

Since parasites can regulate the metabolism and immunity of the host in order to ensure its survival, whether or not it is a harmonious symbiotic relationship, it is necessary to show the possible association between metabolic disorders and parasitic infections, the main objective of this paper [3, 8, 9].

Discussion

There is a complex and dynamic relationship between parasitic infections and metabolic disorders, with mutual influence. For example, parasites and their antigens contribute to reducing blood glucose and improving insulin sensitivity by affecting metabolism, nutrient absorption, and the intestinal ecosystem as a pathogenetic mechanism, especially through a reduction in circulating pro-inflammatory cytokines and an increase in anti-inflammatory cytokines. On the other hand, a mechanism that allows for glycemic control is the depletion of the body's energy sources, which leads to weight loss, as well as the manipulation of the intestinal microbiome, both of which play a key role in blood glucose homeostasis [3, 10-13].

The regulatory role of parasites (e.g., eggs of the helminth *Schitosoma* spp.) in T2DM has been reported (with associated measures such as HbA1c, basal and postprandial glycemia and insulin, and pancreatic inflammation by histopathological analysis). However, age and other confounding factors should be considered in the interpretation of this and other findings from the metabolic perspective of the underlying mechanism of parasitic survival in patients with T2DM marked by humoral immunity disorders, innate immunity dysregulation, multiorgan impairment, and gut microbiota alterations (which can lead to mixed parasitic infections), as these patients may present generalized immune weakness and reduced T-cell responses [3, 14].

In this sense, the polarization of macrophages to M2 by helminths is involved in the alleviation of host metabolic diseases, with the aim of ensuring their survival. This strategy is effective against diseases related to metabolic disorders, since macrophage activation (M1) generates mild but persistent inflammation, which leads to obesity and related syndromes in the host. In this regard, studies with *Echinococcus granulosus* larvae in mice reveal increased lipolysis of adipose tissue in these animals, accompanied by increased arginine metabolism and activation of the PPAR-γ pathway, which plays a key role in M2 polarization. Furthermore, administration of antiparasitic treatment worsens glycemic control [15-21].

Obesity-induced cognitive decline can be limited by the effective inactivation of macrophages (M1) and their polarization to M2, as well as by the reduction of neuroinflammation induced by microglia and astrocytes in the brain by *E. granulosus* larvae (in the experimental mouse model). Furthermore, the immunoregulatory omega-1 glycoprotein from *S. mansoni* improves metabolic homeostasis in obese mice by inhibiting food intake. Succinate also regulates the immune response in macrophages by interacting with its receptor (G protein-coupled receptor 91 [GPR91] or succinate receptor 1 [SUCNR1]), which plays an important role in reducing overweight in the host [17, 19, 22-26].

Randomized trials report lower baseline levels of total and LDL cholesterol, and triglycerides, with increased HDL cholesterol (the risk of cardiovascular disease increases with HDL levels below 40 mg/dL) in subjects with infections with *S. mansoni*, *Strongyloides stercoralis*, and *Fasciola hepatica* (effects observed especially in parasites that affect the liver by altering intrahepatic fat processing). This is directly related to the intensity of the infection, but inversely related to the administration of anthelmintic treatment, since the average LDL cholesterol increases in those undergoing antiparasitic treatment [19, 27].

Regarding the measurement of parameters related to metabolic syndrome, studies in subjects infected with parasites reported lower body weight, blood glucose levels, insulin levels, and insulin resistance. The cardioprotection induced by parasitic infections appears to be greater in adult men than in the opposite sex, and the beneficial effects may derive from different mechanisms in young and older adults. However, the existing information in this regard is inconclusive, therefore, more rigorous studies are required to elucidate these arguments [19, 28].

Coronary artery disease and heart attack are decreased with parasitic infection, because findings show fewer atherosclerotic plaques in the aorta, carotid sinus, and other arteries, thereby improving blood flow to the heart. Likewise, parasitic infection decreases fat deposition in the kidneys and glomerular damage. However, an association between parasitic disease and blood pressure control is not possible [19, 26, 29].

Conclusion

Parasitic diseases affect a large portion of the world's population, with a sustained increase in morbidity and mortality, as well as serious complications, primarily in low- to middle-income countries. Human health, in relation to cardiometabolic disorders, may benefit from the coevolution between humans and parasites, as they have coexisted for millennia. Despite the variations between different studies in relation to sample size, population, area (urban, rural, endemic and non-endemic) and season of the study, the parasites detected, risk factors for metabolic diseases (alcoholism, smoking, among others), and the protocol for managing metabolic disorders, there is undoubtedly a beneficial effect of parasitic infections on these chronic diseases and their loss with antiparasitic treatment, through mechanisms such as the polarization of macrophages to the M2 type response, with an immediate reduction in the inflammatory response, which foresees a new perspective for the prevention of metabolic diseases that justify further research in this regard.

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