581

Institute of Medical Science
UNIVERSITY OF TORONTO

Public Health Ontario | Santé publique Ontario

Influence of Host on Nutriome on Immunological Control of Protozoal Infections

Emma Hagopian[1], Shveta Bhasker[1], Michael Klowak[2,3], Ruwandi Kariyawasam[2,3], David Harris[1], **Priyanka Challa[1]**, Celine Lecce[1], Rachel Lau[3], Andrea K. Boggild[1,2,3,4]

[1]Tropical Disease Unit, University Health Network, Toronto, ON, Canada, [2]Institute of Medical Sciences, University of Toronto, Toronto, Ontario, Canada, [3]Public Health Ontario Laboratories, Public Health Ontario, Toronto, ON, Canada, [4]Department of Medicine, University of Toronto, Toronto, Ontario, Canada

UHN Toronto General Hospital

UNIVERSITY OF TORONTO

Introduction

- Immunologic control of parasitic infections is a combination of humoral and cellular immunity
- Inadequate nutritional status impairs the functioning of the immune system potentially resulting in increased susceptibility to protozoal infections
- Laboratory, epidemiological, and other observational studies provide convincing evidence that micronutrient deficiencies contribute to the mortality and morbidity of infectious diseases[1,2]
- We aim to synthesize existing knowledge around the interrelationships between nutrients and immune function and demonstrate the ways in which nutrient deficiencies such as zinc, iron and vitamin A impact immune response and defence in patients with protozoal diseases such as Leishmaniasis and Chagas disease

Methods

- PubMed, Embase, Medline, Scopus, and LILACS were searched from database inception to March 13, 2019 using combinations of the search terms (Leish* OR Trypanasom* OR Protozoa*) AND (Vitamin A Deficiency OR Iron Deficiency OR Anemia OR Zinc Deficiency OR Nutrient Deficienc* OR Nutritional Deficienc* OR Nutritionally compromised OR Micronutrient* OR Malnutrition OR Nutrition OR Nutritional Status) AND (Immunology OR Immunity OR Immune System OR Immune Function OR Immune Impairment OR Immune Response OR Immune Status)

Future Directions

Screening → **PRISMA** → **Data Extraction**

- Titles, abstracts, and full-text articles will be systematically double screened by two reviewers with a tertiary arbitrator
- The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) will be implemented
- Data extraction will be performed by two reviewers and the quality of the articles will be critically evaluated using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach

Contact: Dr. Andrea K. Boggild; E-mail: andrea.boggild@utoronto.ca
@BoggildLab; Website: www.boggildlab.ca

Results

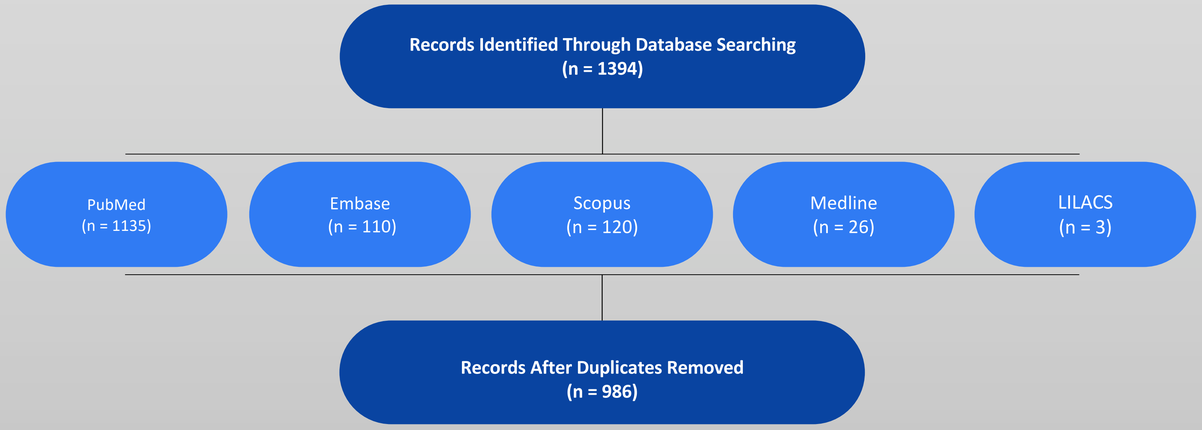

Table 1. Known immunological effects of micronutrients in cellular, murine, and in vivo models of leishmaniasis and Chagas disease.

Micronutrient	*Leishmania*	*Trypanosoma cruzi*
Zinc	- Oral treatment with zinc sulfate reduces lesion size and parasite load and enhances Th1 cytokine response in ulcers of *L. major* · Zinc-deficient diets lead to greater visceralization of *L. donovani*	· Mononuclear phagocytes have reduced capacity to associate with and kill *T. cruzi* in setting of zinc deficiency
Iron	· ron chelation delays development of cutaneous ulcers due to *L. major* · Paradoxically, systemic iron leads to reduced parasite load and footpad pathology ·Iron-deficient diets lead to greater visceralization of *L. donovani*	· Iron chelation decreases parasitemia and mortality in *T. cruzi infected* mice due to a trypanostatic effect
Vitamin A	· Levels of Vitamin A are lower in children with visceral leishmaniasis · Vitamin A supplementation promotes multiplication of *L. donovani* · Vitamin A deficiency leads to Th2 to Th1 switching, and greater production of Th1 cytokines	·Deficiency alters innate immunity by impairing normal regeneration of mucosal barriers damaged by *T. cruzi infection*, and by reducing the function of PMNs, macrophages, and NK cells
Vitamin C	·Prophylactic administration of Vitamin C reduces uptake of *L. donovani in* hamsters, thereby reducing parasite burden	·Vitamin C reduces parasitemia in *T. cruzi infected* mice compared to controls, but not compared to benznidazole · Combination vitamin C and benznidazole led to 100% survival and reduced acute-phase infection weight loss
Vitamin D	·Vitamin D binding protein is downregulated in visceral leishmaniasis	· Inflammatory reaction and cellular and tissue parasitism by *T. cruzi* reduced in animals given higher doses of Vitamin D
Vitamin B1	· Deficiency does not alter clinical course of visceral leishmaniasis in mice	· Deficiency leads to higher parasitemia and larger cardiac lesions in *T. cruzi* infected rats

References

[1]Taylor, C. E., & Higgs, E. S. (2000). Micronutrients and infectious diseases: Thoughts on integration of mechanistic approaches into micronutrient research. The Journal of Infectious Diseases, 182(Supplement_1), S1-S4.
[2]SanJoaquin, M. A., Molyneux, M. E. (2009). Malaria and vitamin A deficiency in African children: a vicious circle? Malaria Journal, 8(134).