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Levels of Macroelements and Microelements in Urine during Experimental Acanthamoebiasis

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Abstract

This study aims to investigate the influence of free-living amoebas on the excretion of both macroelements and microelements in urine. The research focuses on analyzing the concentrations of macroelements, including calcium (Ca), phosphorus (P), sodium (Na), potassium (K), and magnesium (Mg), as well as microelements like manganese (Mn), zinc (Zn), copper (Cu), iron (Fe), and chromium (Cr), during acanthamoebiasis, while taking into consideration the immunological status of the host. This groundbreaking study reveals, for the first time, notable changes in the urinary excretion of several elements in response to *Acanthamoeba* sp. infection in immunocompetent mice. Specifically, 16 days post-infection, there is an observed increase in the excretion of calcium, manganese, copper, iron, sodium, and chromium, alongside a decrease in potassium excretion. As the infection progresses to its later stage (24 days post-infection), there is a further reduction in urinary potassium excretion and lower levels of phosphorus in *Acanthamoeba* sp. infected immunocompetent hosts. In the context of acanthamoebiasis within immunosuppressed hosts, an initial increase in excretion of zinc, iron, and chromium is noted at the early phase of infection, accompanied by increased sodium excretion only at 16 days post-infection with *Acanthamoeba* sp. Furthermore, the immunosuppressive state of the host has an impact on the urinary concentrations of iron, chromium, zinc, copper, manganese, and calcium.

Keywords: Macroelements • Microelements • Urine

Introduction

Acanthamoeba sp., a pathogenic free-living amoeba, demonstrates a predilection for the central nervous system and is particularly prevalent in individuals undergoing intensive steroid therapy, organ transplants, HIV infection, and pediatric cases. Its spread encompasses multiple organs, with the kidneys becoming involved subsequent to initial skin, subcutaneous tissue, or lung colonization. Brain Acanthamoeba infection typically manifests with nonspecific symptoms like headache, nausea, dizziness, irritability, and mild fever. Differential diagnosis from bacterial leptomeningitis, tuberculous meningitis, or viral meningitis poses challenges [1]. Despite the significance of multi-organ invasion by Acanthamoeba, the underlying host-pathogen interactions remain enigmatic. Understanding these intricate mechanisms holds the key to mitigating invasion risks, given the rapid disease progression and high mortality rates.

The absence of a definitive therapy underscores the critical role of the immune system. Survival of the pathogen and host immune activation hinge on trace elements present, pivotal in coenzyme activation of immune cells, possibly influencing leukocyte production, maturation, and function. However, insights into macronutrient and micronutrient status during parasitic infections, despite kidneys being central detoxification organs and urine offering convenient analysis material, remain scant. Urinary magnesium (Mg) and zinc (Zn) excretion increases were observed in Trypanosoma brucei-infected rabbits. Schistosomiasis-afflicted children exhibited elevated iron (Fe) and copper (Cu) levels in urine, while acute Schistosoma haematobium infection lowered manganese (Mn) and cadmium (Cd) concentrations along with hematuria [2]. Immunocompetent mice displayed higher nephric selenium (Se) levels relative to control hosts.

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Description

Gastrointestinal nematode burden was linked to iron (Fe), molybdenum (Mo), copper (Cu), and zinc (Zn) losses due to excessive intake. Trace element metabolic imbalances can trigger metabolic disorders and pathophysiological cascades. Combined macronutrient and micronutrient deficiencies coexist with infections, potentiating detrimental clinical effects, especially in immunocompromised individuals. Zinc (Zn), iron (Fe), and selenium (Se) are recognized to modulate immunity, influence infection susceptibility, and counteract oxidative stress [3].

Fluctuations in urinary element concentrations may stem from inflammation, hormonal imbalances, absorption, excretion variations, and medication impacts, including immunosuppressants. Thus, these changes offer insights into overall health. Notably, there is a paucity of literature concerning macroelement and microelement concentrations in Acanthamoeba infection. The mechanisms driving element level shifts in the urine of infected hosts remain elusive. While Acanthamoebiasis primarily affects the immunocompromised, severe cases have been reported in immunocompetent individuals, necessitating a comprehensive understanding across immunological spectra. Thus, this study examines macroelements like calcium (Ca), phosphorus (P), sodium (Na), potassium (K), magnesium (Mg), and microelements including manganese (Mn), zinc (Zn), copper (Cu), iron (Fe), and chromium (Cr) in urine during Acanthamoeba infection, accounting for host immune status [4].

Inflammation-induced kidney alterations can manifest in blood indicators, including urea and creatinine. Our prior research on the same experimental model revealed kidney inflammation-related changes without significant serum creatinine and urea variations. Amoebas infiltrating the kidneys via the bloodstream could impair renal function and affect urinary element excretion.

While urinary trace element analysis has shown promise in indicating

bacterial and viral infections and disease severity, its relationship with parasitic infections like Acanthamoeba remains relatively unexplored. Unlike serum-focused research, urinary element analyses are limited. This study pioneers the revelation that free-living amoebas impact macroelement and microelement excretion in urine. Immunosuppression influenced element concentrations; methylprednisolone-administered immunosuppressed mice displayed higher Mn, Fe, and Cr levels during early Acanthamoeba infection, contrasting immunocompetent counterparts. However, at 16 days post-infection, immunosuppressed mice exhibited reduced Ca, Mn, Zn, Cu, and Fe excretion. Corticosteroid-associated potassium loss was also noted at 24 days post-infection [5].

Increased urinary trace element excretion during disease could disturb mineral balance, heightening vulnerability. Supplementation interventions, shown effective in managing inflammatory processes, could potentially be adapted for Acanthamoeba infection. Certain elements like chromium, iron, selenium, and zinc impact HIV progression and treatment. Monitoring trace element excretion during Acanthamoeba infection might prevent deficiencies, impacting treatment efficacy, necessitating further research [6].

Conclusion

The findings of this study underscore the influence of free-living amoebas on the excretion of both macro and microelements in urine, potentially opening avenues for deeper exploration of element metabolism. Notably, this investigation marks the first instance where the 16th day of *Acanthamoeba* sp. infection emerges as pivotal for the urinary excretion of calcium (Ca), manganese (Mn), copper (Cu), iron (Fe), sodium (Na), chromium (Cr), and potassium (K) in immunocompetent mice. Within immunosuppressed hosts grappling with acanthamoebiasis, initial elevation in zinc (Zn), iron (Fe), and chromium (Cr) excretion is followed by a subsequent surge in sodium (Na) excretion on the 16th day post *Acanthamoeba* sp. infection. Moreover, the immune-compromised state of the host significantly impacts the urinary concentrations of iron (Fe), chromium (Cr), zinc (Zn), copper (Cu), manganese (Mn), and calcium (Ca). The identification of shifts in urinary element levels in *Acanthamoeba* sp. infected hosts holds promise for expanding our comprehension of the pathophysiology underlying disseminated acanthamoebiasis.

Acknowledgment

None.

Conflict of Interest

None.

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