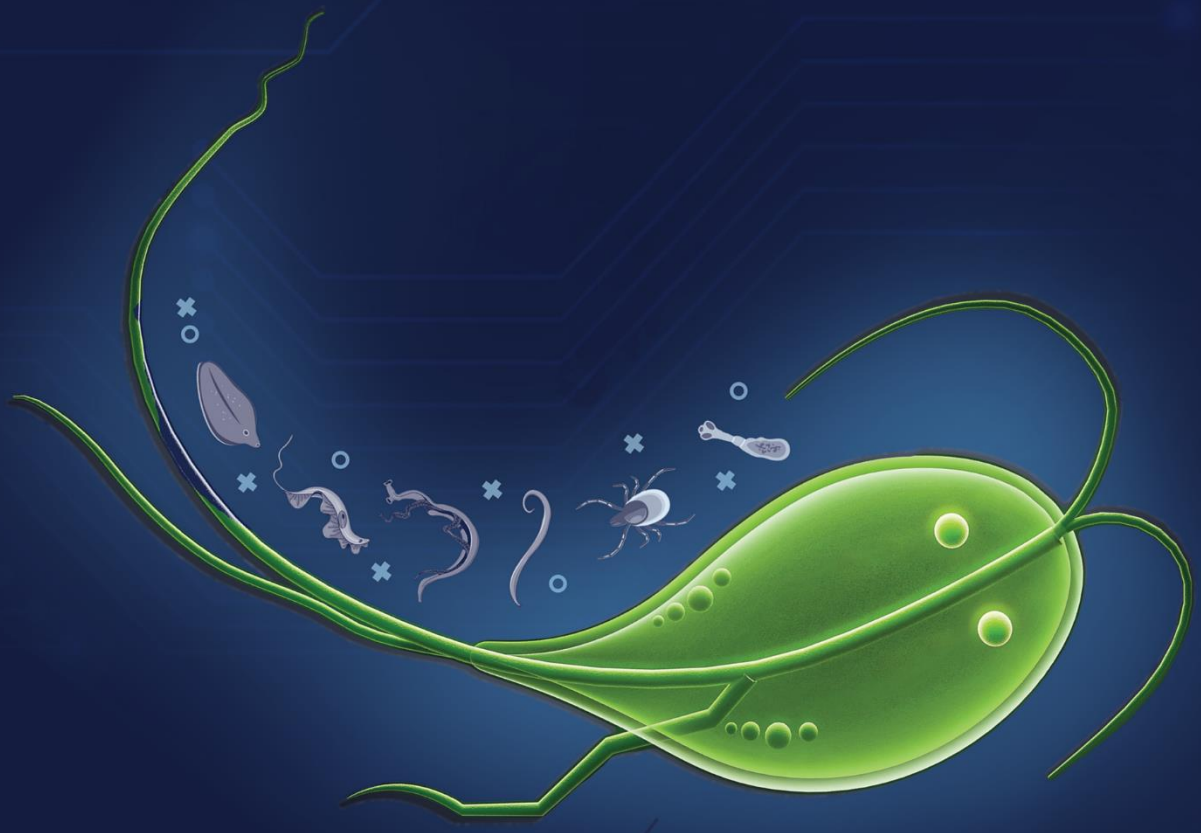
3rd National Congress Of Animal parasitic Diseases and Zoonoses

PROCEEDING

17-18 september 2025, Mashhad, iran

Ferdowsi University of Mashhad



سومین کنگره ملی بیماری‌های انگلی دام و بیماری‌های انگلی مشترک انسان و حیوان

3rd

دانشگاه فردوسی مشهد

دانشکده دامپزشکی

شهریور ماه ۱۴۰۴

National Congress of Animal Parasitic Diseases and Zoonoses

Ferdowsi University of Mashhad - September 2025

سومین کنگره ملی
بیماری‌های انگلی دام و انگل‌های مشترک انسان و حیوان

The 3rd National Congress Of Animal Parasitic Diseases and Zoonoses

دانشگاه فردوسی مشهد، ۲۶ و ۲۷ شهریور ۱۴۰۴

مدیریت نوین بیماری‌های انگلی، تأمین سلامت یکپارچه و امنیت غذایی

دارای امتیاز باز آموزی

ثبت نام از اول اردیبهشت ۱۴۰۴

ISC ۰۴۲۵۰-۵۸۱۷۳

محورهای کنگره

بیماری‌های انگلی مشترک انسان و حیوان

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هوش مصنوعی و سایر فناوری‌های نوین در تشخیص و درمان بیماری‌های انگلی

آدرس دبیرخانه: ایران، مشهد، میدان آزادی، دانشگاه فردوسی مشهد، دانشکده دامپزشکی.

تلفن دبیرخانه: ۰۵۱-۳۸۸۰۳۷۵۰ ۰۹۳۳۶۵۷۸۰۶۳ فکس: ۰۵۱-۳۸۸۰۷۰۷۶

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Physiological Modeling of the Autonomic Nervous System Response to Parasitic Infections in Animals

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Article code: 1295

Abstract

Background: Parasitic infections in animals are not only associated with immune and physiological consequences, but also induce significant changes in autonomic neural responses. The neurophysiological axis of these responses, which is mainly mediated by the sympathetic and parasympathetic nervous systems, plays an important role in maintaining homeostasis under chronic stress conditions caused by infection. In this article, the physiological modeling of these responses was investigated with emphasis on factors such as heart rate changes , pupil dilation, feeding behaviors, and restlessness or immobility in animals infected with parasites.

Methods: In this study, a number of articles related to the relationship between parasitic infections in animals and disruption of the autonomic nervous system were reviewed, and interesting findings were found regarding this effect.

Results: Since the aim of this text was to analyze the neural mechanisms involved in regulating autonomic responses to the presence of parasites and to evaluate the role of neurotransmitters and neural pathways in facilitating or inhibiting these responses, a review of studies has shown that parasites such as Trypanosoma cruzi, Toxoplasma gondii, and Plasmodium species can cause changes such as tachycardia, anorexia, and disruption of physiological rhythms by activating the HPA axis and stimulating certain areas in the brainstem and cortical and subcortical areas of the brain.

Conclusion: Finally, these autonomous responses, although initiated with the aim of maintaining host survival, may in some circumstances act in the parasite's favor, including by suppressing immunity or altering host behavior to enhance transmission. A detailed understanding of these pathways could open new horizons in the treatment of parasitic diseases and clinical management of parasitic diseases.

keywords: Autonomic nervous response, Parasitic infections, Physiological modeling, Anorexia, Feeding behaviors.

Introduction:

The physiological responses of animals to pathogens have always been of interest to neuroscientists and physiologist. But what has been less systematically studied are the responses of the autonomic nervous system (ANS). Can these responses be predicted? Is there a consistent pattern across species? Then, most importantly, are these responses simply involuntary reactions or do they play an active role in the survival of the host and even the parasite?

As the master regulator of the body's involuntary activities, the autonomic nervous system is highly sensitive to internal threats such as actions. When pathogens enter that body, activities such as increased heart rate, changes in pupil diameter, and readjustment of blood flow and heart rate can all be triggered. These responses, which appear to be responses to a problem or pain, are actually part of the body's defense mechanism. But the point is that in some parasitic infections, the host's defensive reactions to the presence of the parasitic infection can even cause damage to the host's body (1).

From this perspective, there is a need for detailed physiological modeling, as only detailed understanding of the function of these pathways can lead us to a better understanding of disease processes, prognosis, and even the design of targeted drugs. The existence of a suitable model of the process of organ activity in a healthy animal and the course of change until disease can provide the best platform for the researcher to design animal-based experimental research.

Materials and Methods:

In this study, some articles and research related to the relationship between parasitic infections in animals and disorders occurring in the autonomic nervous system and the development of neurological diseases were reviewed, and the findings on the relationship were summarized, indicating the mutual influence of these two factors.

Results and Discussion:

Cardiovascular, gastrointestinal, and pupillary responses to parasitic infections

One of the most prominent indicators of ANS (autonomic nervous system) response is the change in heart rate. In many animals, including poultry, parasitic infections such as Eimeria can cause tachycardia (increased HR). This reaction occurs due to the secretion of some cytokines such as TNF-α and IL-1β from immune cells, which are then transmitted to sympathetic pathways via visceral afferent neurons and brain centers associated with the ANS (example: nucleus solitarius) (2). But this is not always the case; in some chronic infections,

such as Toxoplasma gondii, a decrease in heart rate (bradycardia) has been seen as a long-term, adaptive response (3).

Also, significant changes are observed at the digestive level. Parasitic infections are often associated with disturbances in intestinal motility and digestive secretions. Decreased peristalsis, gastric distension, and nausea (in mammals) can be due to the dominance of parasympathetic pathways or even their inhibition by inflammatory mediators (4).

The interesting thing is that in many cases, despite the brain receiving these environmental messages on a large scale, it only brings a portion of them to the level of perception and awareness in the somatosensory cortex. In other words, the body knows, but the mind does not. On the other hand, pupil diameter, a well-known indicator of sympathetic activity, can dilate in response to infection. In rodents infected with Trypanosoma cruzi, pupil dilation has been reported even at rest, indicating a sustained stimulation of the sympathetic system.

The question here is whether this change is simply a consequence of an inflammatory process or part of the host's neural programming for survival? This is a question that cannot yet be answered definitively. As we have seen, the early ANS responses to infection are not only rapid and autonomous, but in some cases mandatory. Although their function is often interpreted to maintain homeostasis, parasites may have created mechanisms to exploit these responses over time for survival (1).

Interaction of neurons, immune mediators, and the role of parasites in altering ANS settings

The autonomic nervous system's response to parasitic infections is not limited to direct reactions, but rather involves a two-way interaction between neurons and immune cells. For example, macrophages infected with Leishmania major can release molecules such as prostaglandin E2, which, through specific receptors, stimulate certain visceral afferent neurons. This stimulation then triggers sympathetic nervous system responses that potentially reduce blood flow to the affected area, creating hypoxic conditions for the parasite, thus affecting parasite survival (5,6).

In this regard, the role of the vagus nerve (tenth cranial nerve) is also very crucial. The vagus nerve is involved in the transmission of peripheral signals to the central nuclei of the brain , including the noradrenergic areas of the forebrain cortex (7). In particular, in poultry infected with Ascaridia galli, evidence has been reported of altered vagal excitation patterns and reduced parasympathetic tone, which could play a role in the weakening of digestive activity such as intestinal motility and longer persistence of the parasite in the digestive tract.

What's even more interesting is that some parasites can actively manipulate and recruit neural pathways! In Toxoplasma gondii infection, the parasite can lodge in the host's neurons, increase transmitters such as dopamine, and in this way, direct autonomic responses in favor of its own

survival. In these cases, the ANS response can no longer be considered solely the result of the presence of the parasite, but rather it should be considered part of the parasite's plan and strategy to control the host (3,8).

General pattern of responses, modeling, and future applications

Physiological modeling of these responses requires integrating different data from different organs such as the heart, gut, brain, eye, and even skin. Systems such as mathematical models of neural-immune networks or agent-based multi-agent models can help simulate these complex interactions. For example, using the previously mentioned experimental data on heart rate, body temperature, and pupil diameter, a model can be created for early detection of a specific type of parasitic infection.

On the other hand, in behavioral studies of infected animals, whether mammals, rodents, or birds, changes in feeding, aggression, or social withdrawal (all of which are regulated by the ANS) can be modeled (9). The use of these models will play an important role not only in understanding the pathophysiology of infection, but also in designing non-invasive diagnostic tests. This is the horizon that animal neurophysiology studies, especially in the field of veterinary medicine, must strive to achieve and ultimately exploit (10).

Finally, the fact that some responses are symmetrically eliminated or spontaneously silenced reminds us that what is seen in the host body is only part of the host's neural response. Many internal processes, such as the rearrangement of neuronal pathways, the alteration of receptor gene expression in ganglia, and the inhibition of regulatory feedback, occur behind the scenes and remain hidden from clinical view, making diagnosis difficult.

Therefore , physiological modeling of the ANS response to parasites is not only a scientific challenge, but also a necessity to advance our understanding of the biology of parasitic diseases as well as improve management strategies in modern veterinary medicine.

Acknowledgment:

Author would like to thank the Ferdowsi University of Mashhad for their support**.**

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