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REVIEW ARTICLE

Trichinella Spiralis and Trichinellosis: Morphological, Biological and Immunological Perspectives

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ABSTRACT

Background: *Trichinella spiralis (T. spiralis)* is an important zoonotic parasitic worm that is distributed all over the world. Unlike other parasites, it develops in a single host throughout the whole of its life cycle. Humans develop the infection through ingestion of raw or insufficiently cooked meat of pigs or other animal hosts containing *Trichinella spiralis* encysted larva. *Trichinella spiralis* is unique among helminthes by its intriguing abilities not only live and thrive inside the skeletal muscle tissues but have also evolved complex mechanisms for remodeling that niche. *T. spiralis* triggers a complicated and multidimensional immunological response that includes both innate and adaptive immune systems. During the intestinal phase of infection, the Th1 and Th2 immune responses are both involved in the immunological response to trichinosis. Th1 responses are initially produced, followed by a dominating Th2 response, which is characterized by the production of high amounts of cytokines IL-4, IL-5, IL-9, IL-10, and IL-13.

Conclusions: Apart from its significant role in causing sickness and mortality, trichinosis is also thought to be a source of income loss for farmers and meat processors. Consequently, public health officials around the world are concerned about trichinosis. It is necessary to raise awareness of the intricate biology and morphology of trichinosis to create successful prevention and control programs. Hence, in this article, we aspire to provide detailed insights into the morphological, biological and immunological aspects of *Trichinella spiralis* infection.

Keywords: Trichinella spiralis; Biology; Morphology; Immune response; Trichinosis

INTRODUCTION

The words "trichinellosis," "trichinosis," and "trichiniasis" all describe infections caused by parasitic nematodes of the genus *Trichinella* in both their larval and adult stages. Apart from Antarctica, these parasites are common in wildlife on every continent. The important features of this infection are that it is zoonotic and that the infectious larvae are transmitted through meat. Humans and variety of other animals acquire the infection by consuming undercooked contaminated meat [1].

Trichinella spiralis, T. nativa and its related genotype Trichinella T6, T. britovi and its related genotype Trichinella T8, T. pseudospiralis, T. murrelli and its related genotype Trichinella T9, T. nelsoni, T. papuae, and T. zimbabwensis are the eight species and three genotypes currently recognized in the genus Trichinella. All species can grow in mammals; however, some reptile species also have T. papuae and T. zimbabwensis, and T. pseudospiralis can grow in birds. There are no physical variations between species and genotypes, and biochemical or molecular investigations are the most reliable methods of distinguishing them [2].

New eating habits based on raw or undercooked pork products may be brought about by human migration, and in endemic nations, these behaviors have caused outbreaks of trichinosis in unaware immigrant communities. Numerous reports of tourists who had Trichinella infections while traveling or hunting in endemic

areas and then experienced clinical symptoms upon returning home have been made because of the growing number of foreign visitors. Since the infections typically manifested as isolated cases, diagnosis was difficult in most cases [3].

MORPHOLOGICAL ASPECTS

1. Adult stages (Figure 1) [4]**:**

Despite being colorless, male and female adult worms can be distinguished by the following physical traits [5]:

Male worms:

T. spiralis male worms have total width from 25µm to 33µm and total length from 0.62 mm to 1.58 mm. Despite being smooth, the cuticle displays pseudo segmentation, and it is periodically interrupted by dorsal and ventral pairs of hypodermal gland cells. Genital terminal consists of a pair of flattened copulatory appendages and accessory papillae. The capillary esophagus, midgut with brush border, hindgut, and mouth cavity make up the alimentary tract. The worm's anterior region contains the stichosome. There is only one testis in the reproductive system.

Female worms:

T. spiralis female worms have total width from 29µm to 38µm and total length from 1.26 mm to 3.35 mm. They have no copulatory appendages, yet the cuticle resembles that of males. The vulva is present in the posterior end of the stichosome.

Muscle larvae (Figure 2) [6]**:** Also known as the first-stage (L1) larvae. They are coiled in a lemon-shaped capsule when they encyst inside the muscle tissue of the host. The size of the cyst in the human host is approximately 400 by 260 µm. The coiled larvae are 800–1,000 µm long inside the cyst. At this stage, the larvae are completely contagious [7].

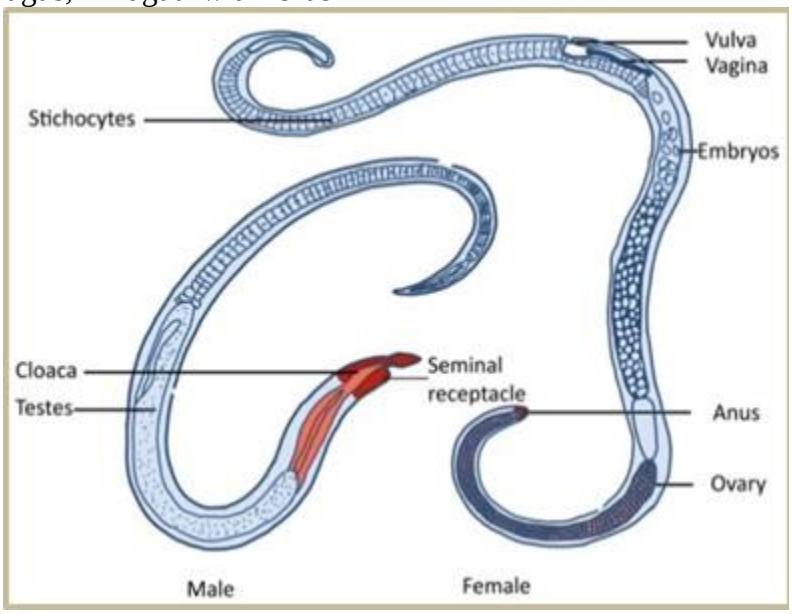

Figure (1): A schematic demonstration of *T. spiralis* adult stages [4].

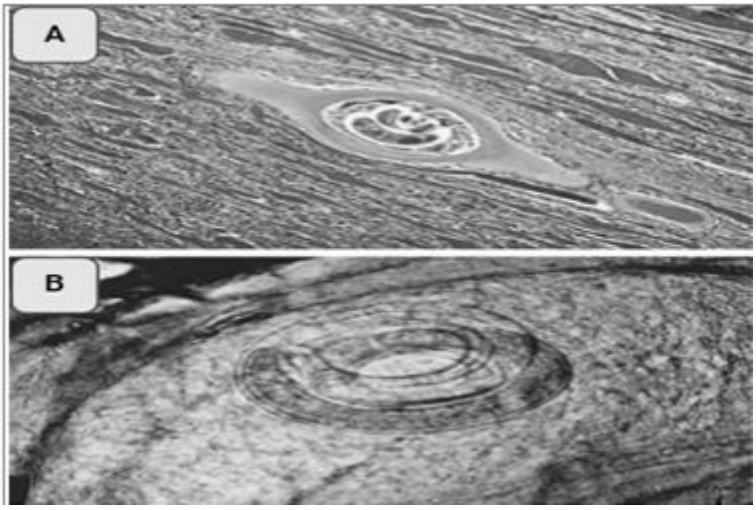

Figure (2): (A): Encysted larvae of *Trichinella spiralis* found in muscle tissue surrounded by fibrous tissue replaced adjacent muscle and infiltrated by chronic inflammatory cells. **(B):** *T. spiralis coiled larva in wet mount* muscle preparation [6].

BIOLOGICAL ASPECTS

The free-living stage:

An important adaptation of the parasite, which facilitates its transmission, is a physiological mechanism utilized by muscle larvae to promote its survival in decaying tissues. The greater the persistence of larval viability, the higher the probability of being ingested by a scavenging host. Despite the larva-induced angiogenesis that develops around the nurse cells after larval penetration of the muscle cell, larval metabolism is basically anaerobic [8], which favors its survival in decaying tissues, probably longer for the encapsulated than for the non-encapsulated species.

The environment also affects the persistence of larvae; low temperatures and high humidity prolong the encapsulated larvae's existence even after the muscle tissue has completely liquefied.

This condition has been proposed as the environment of the 'free-living' stage, resembling the egg stage of most of other nematode species [3].

Resistance to freezing:

The anaerobic metabolism prefers survival in putrefying flesh, along with the ability of larvae of some species to survive freezing, are two separate mechanisms that strongly increase the survival of the parasite in nature. At lower temperatures, the survival time is quickly reduced. It is also important to indicate that the survival of muscle larvae to freeze occurs mainly when these larvae parasitize striated muscles of carnivores (bears, wolves, and foxes), whereas the survival time to freeze is strongly reduced to a few days or weeks when muscle larvae of the same strain parasitize other mammalian hosts such as pigs or rodents [9].

How does *Trichinella spiralis* make itself at home?

Trichinella inhabits the host cell without causing death, in contrast to most intracellular parasites. It is therefore regarded as one of the most effective parasitic symbionts. A unique host-parasite structure called a "nurse cell" (NC) is formed when the striated muscle cells are infected with the newly born larvae (NBL), causing developmental changes. Nurse cells are composed of cellular components and a collagenous wall that help shield the parasite from the host immune response, increasing its long-term viability [10].

The nurse cell–parasite complex develops in a matter of 15 to 20 days after the larva invades that striated muscular cell type. The development of NC is complex and is thought to be chiefly attributed to the secreted

tyvelosylated proteins of the larvae. From the seventh day following infection, these proteins are consistently found in the larvae's intracellular niche. They might be in charge of rerouting host genomic expression, which would result in the production of nurse cells [11].

IMMUNOLOGICAL ASPECTS

Protective immune mechanisms against helminths:

Helminthic infections and their associated immunological reactions of the host are the outcome of a long-term dynamic interaction between the parasite and its host. Parasites benefit from tricking the host into producing an inadequate immune response, as well as finding a good location for maturation and proliferation while avoiding killing or hurting the host. The host must create an adequate immunological response to eliminate the parasite and reduce its detrimental impacts, while maintaining its capacity to effectively react to other diseases [12].

The protective immune response against many helminth parasites is known as the type 2 response, or Th2 response. A variety of cytokines, such as interleukin 4, IL-13, IL-21, and IL-25, which are expressed during the Th2-type response, result in the suppression of Th1-type and Th17-type reactions and the inflammation they generate [13].

The frontline innate cells, including granulocytes, macrophages, and epithelial cells, detect the entry of an infection and stimulate T-cell differentiation by presenting antigens or cytokines. After that, antigen-specific T lymphocytes begin to release a variety of cytokines that encourage innate cells to eradicate parasites [14].

A strong Th2 response triggered by helminths promotes collagen deposition, mucus secretion, and wound healing processes, all of which are essential for helminths expulsion. Helminthes can frequently remain in the host for a long period of time even after triggering the defensive Th2 response, leading to chronic infection [15].

Role of the innate effector cells during helminth infections:

The Th2-type immune response's initiation and effector phases depend on innate immune cells. Once activated, innate-cell populations in turn support and encourage the formation of the Th2 effector cells. CD4+ Th2 effector cells primarily use cytokine synthesis to enhance and direct the innate effector cell response. This leads to a comprehensive effector response made up of linked cells that target and coordinate effector actions against invasive helminths [16].

Macrophages:

Macrophages are considered the first of the innate immune system's phagocytic cells and are also characterized with various roles crucial for defense mechanisms, tissue development and homeostasis. They are produced from hematopoietic progenitors and begin to colonize peripheral tissues in the early stages of fetal life, a process that continues until adulthood [17].

Role of macrophages in nitric oxide (NO) production:

M1 macrophages, or classically activated macrophages, use mechanisms unrelated to complement or antibodies to engulf and eliminate various pathogens. They can eradicate infectious parasites based on the inducible nitric oxide synthase activity and the cytokine activation of interferon-gamma (IFN-γ) and tumor necrosis factor-alpha (TNF-a) [18]. So, this pathway is associated with high (NO) output**.**

M2 macrophages, or alternative activation of macrophages, are a common immunological response, especially when helminthic infections are present. The cytokines IL-4 and IL-13, which are secreted by Th2 cells and innate cells such as mast cells, basophils, and eosinophils, cause alternative activation of macrophages. Until recently, macrophages were believed to be quiescent during (Th2)-immune responses, having a secondary role in eosinophils and mast cells. Yet, different studies have demonstrated a distinct alternative macrophage activation and

rapid recruitment to sites of infection during (Th2)-type responses to helminths [19].

Nitric oxide (NO):

Until recently, the highly reactive gas nitric oxide (NO) was thought to belong to a family of possible carcinogens. It is one of the main cytotoxic mediators of immunological effector cells [20]. The role of (NO) in parasitic diseases is still debatable.

Synthesis and Physiological functions of NO:

Nitric oxide is generated from the oxidative deamination of L-arginine to produce L-citrulline. The conversion of arginine to citrulline and (NO) is catalyzed by nitric oxide synthase (NOS) enzyme, of which three isoforms have been identified. The three isoforms are endothelial (eNOS), neuronal (nNOS), and inducible isoform (iNOS) [21,22]. NOS isoforms vary in their pathophysiological roles, anatomical distribution, and genetic origin. [23]. Numerous physiological functions, such as vasodilatation and nerve transmission, involve the constitutive form of NO [24]. The long-term regulation of synaptic transmission is mediated by NOS in the central nervous system. Also, it has a role in central blood pressure regulation [25]. Endothelial (NOS) regulates several cardiovascular processes as a homeostatic regulator. One powerful inhibitor of platelet aggregation and vascular wall adhesion and controls the expression of genes involved in atherogenesis is endothelial (NOS)-derived NO [26]. In contrast to the constitutive (NOS) isoforms, iNOS is not present in resting cells and it is quickly expressed in reaction to stressors such as infections and inflammation [27].

Role of (NO) in parasitic infections:

The role of NO in the defense against helminths was sustained by various studies, such as the utilization of NO donors in different experiments and the evaluation of their action on the stages of the biological cycle of the helminths. This approach has been used to investigate how this molecule affects *Trichinella spiralis*, *Brugia malayi*, and *Echinococcus granulosus* [28].

Role of (NO) during Trichinella spiralis infection:

Numerous investigations were carried out to investigate the potential effects of (NO) during infection with *T. spiralis*. According to Lawrence and colleagues [29], the production of NO by iNOS was not crucial for the elimination of *T. spiralis* from the infected mice; however, it played a notable role in the enteropathy linked to the infection. It is currently unclear how nitric oxide (NO) affects the immune system's reaction to an infection with *Trichinella spiralis (T. spiralis*). NO has a minor protective effect against adult worms during the intestinal stage of the infection, but when the condition worsens, it has been shown to cause intestinal pathology [30].

***Trichinella spiralis* biology of infection and polarization of the immune response:**

T. spiralis is a parasite that creates a long-lasting infection within the host's skeletal muscles. Based on the lifespan of the host, the parasite can potentially survive until the host's death (in rodents) and can remain up to years following infection in human and higher species. Unlike several other intracellular parasites, *T. spiralis* lives inside the host's muscle cells without killing them [31].

Acute inflammatory responses are mediated by the parasite's surface and excretory-secretory (ES) products. The human immune system is profoundly impacted by ES products. After completion of nurse cell formation, ES products remain acting as antigens and immuno modulators. ES products have been characterized as serpins, glycans, mucins, lectins or cytokine homologs that could influence antigen processing, presentation and subsequent T-cell polarization [32].

Intestinal immune response*:*

Before the Th2-mediated reaction removes all adult forms from the intestines, *T. spiralis* rapidly grows and multiplies. Consequently, the immune response during the intestinal phase presents a mix of Th1 and Th2, initially showing a predominance of the Th1 response, and the subsequent domination of Th2 characterized by elevated levels of cytokines

IL-4, IL-5, IL-9, IL-10, as well as immunoglobulin E [33].

The interaction of cytokines with different intestinal mucosal immune cells, such as mast cells, eosinophils, goblet cells, and dendritic cells (DCs), drives the initial immune response in the intestine. DCs are essential in focusing the immune response on Th2 and regulatory types [34].

Immune response at the muscular level*:*

T. spiralis muscle larvae (ML) can survive several years in the host, even though the immune system of the host is activated to hasten the removal of the worm from the intestine. One possible reason for this is that the formation of nurse cells during the muscular stage shields the parasite from antibody attacks that occur after the fourth week of infection. Another possibility is that the parasite can alter the host's immune system to increase its chances of surviving by utilizing secretory products and surface antigens [35].

The muscular phase is additionally defined by the presence of regulatory T cells (Treg cells). Persistent exposure to the excretory-secretory (ES) product from *T. spiralis* larvae (ES L1) that enters the bloodstream during the muscular stage of the infection may stimulate regulatory network components to maintain homeostasis [31].

Evasion mechanisms elicited by Trichinella spiralis:

Several ways to evade the host's immunological response have been documented:

***Antigen-dependent mechanisms*:**

Stage-specificity of the antigens is demonstrated by observation that early antibodies are specific for adult worms but do not recognize newborn larva (NBL) antigens [36]. Although encysted in the muscle fibers, muscle larvae (L1) interplay with the host, releasing antigens and continuously stimulating the host immune response. When encapsulated organisms cause infections, they are separated, protecting them from antibodies and effector cells such as eosinophils and macrophages [37].

Mechanisms affecting the host immune response [35]**:** Which include the following strategies:

Induction of immune suppression:

Products obtained from *T. spiralis* contain the component that can inhibit the response to thymus dependent parasite antigens, but not to thymus-independent parasite antigens. This immunomodulation acts during the primary and secondary responses to *Trichinella* infection [38].

Polyclonal lymphocyte activation:

The activation of polyclonal lymphocytes in both humans and infected experimental animals elevates IgG and IgM levels [39]. However, elevated total IgE levels are characteristic of *T. spiralis* infection that may be seen as an immune response tactic [40].

Induction of eosinophilia in blood and tissue:

An elevated eosinophil number in the blood and tissues is a characteristic feature of helminth infections, including those developed by *Trichinella* species. Eosinophil function can cause defending the host or aiding the parasite is a topic of significant debate [41].

Down-regulation of (NOSII) expression:

The influence of *Trichinella* infection on the production of (NOS) II has garnered significant attention. The local inflammation in the Jejunum caused *by T. spiralis* leads to a systemic reduction in NOS II gene transcription, as well as reduced enzyme activity and protein expression. Furthermore, even when endotoxins activate this enzyme, the infection-induced suppression also reduces its expression, and this impact is exclusive to this specific NOS isoform [42].

CONCLUSIONS

Trichinella spiralis infection imposes worldwide public health significance. They constitute medical and veterinary concerns. Therefore, it is imperative that modern diagnostic equipment, clinical management practices, and strict meat hygiene regulations be implemented. A proper understanding of the parasite's biological and immunological features would facilitate effective control designs and would potentially aid the

development of accurate diagnostic technologies.

Conflict of Interest: None

Financial Disclosures: None

REFERENCES

1. Pozio E, Zarlenga DS. Recent advances on the taxonomy, systematics and epidemiology of *Trichinella*. Int Parasitol. 2005; *35*(11-12): 1191-204.

2.Andrews JR, Bandi C, Pozio E, Gomez Morales MA, Ainsworth R, Abernethy D. Identification of *Trichinella pseudospiralis* from a human case using random amplified polymorphic DNA. Am J Med. 1995; *53*(2): 185- 8.

3. Pozio E, Murrell KD. Systematics and epidemiology of *Trichinella*. Adv parasitol. 2006; *63*: 367-439.

4. Chaudhury A. Trichinellosis. In Textbook of Parasitic Zoonoses. Singapore: Springer Nature Singapore, 2022; 415- 26.

5. Bughdadi FA. Ultrastractural studies on the parasitic worm *Trichinella Spiralis*. JTUMED. 2010; *3*(1): 33-8.

6. Garcia, Lynne Shore MS, MT, CLS, F (AAM). Diagnostic Medical Parasitology, 5th ed. 2007; 283-318.

7. Murrell KD. Trichinosis. In G. T. Strickland (ed.), Hunter's Tropical Medicine and Emerging Infectious Diseases, 8th ed The W. B. Saunders Co., Philadelphia, Pa. 2000; 780-7.

8. Despommier DD. *Trichinella spiralis*: the worm that would be virus. Parasitol. Today 1990; *6*(6): 193-6.

9. Pozio E. New patterns of *Trichinella* infection. Vet. Parasitol. 2001; *98*(1-3): 133-48.

10.Despommier DD. *Trichinella spiralis* and the concept of niche. J. Parasitol. 1993; 79(4):472-82.

11. Despommier DD. How does *Trichinella spiralis* make itself at home? Parasitol. Today. 1998; *14*(8): 318.

12. Anthony RM, Rutitzky LI, Urban JFJ, Stadecker MJ, Gause WC. Protective immune mechanisms in helminth infection. *Nat. Rev. Immunol*. 2007; (12): 975–87.

13. MacDonald AS, Arauj MI, Pearce EJ. Immunology of Parasitic Helminthes Infections. *Infect. Immun.* 2000; (70): 427–33

14. Finkelman FD, Shea-Donahue T, Morris SC. Interleukin-4–and interleukin-13–mediated host protection against intestinal nematode parasites. *Immunol. Rev*. 2004; (201): 139–55.

15. Allen JE, Maizels RM. Diversity and dialogue in immunity to helminthes. *Nat. Rev. Immunol.* 2011; 11: 375–88.

16. Díaz A, Allen JE. Mapping immune response profiles: the emerging scenario from helminthes immunology. *Eur. J. Immunol.*2007; (12): 3319–26.

17. Fearon DT, Locksley RM. The instructive role of innate immunity in the acquired immune response. *Science.* 1996; (272): 50-3.

18. Mantovani A, Sozzani S, Locati M, Allavena P, Sica A. Macrophage polarization: tumor-associated macrophages as a paradigm for polarized M2 mononuclear phagocytes. *Trends Immunol.* 2002; (11): 549-55.

19. Omar M, Abdelal HO. Nitric oxide in parasitic infections: a friend or foe*? J. Parasit. Dis*. 2022; 46(4): 1147- 63.

20. Culotta E, Koshland DE. NO news is good news. *Science*. 1992; 258: 1862–65.

21. Bredt DS, Snyder SH. Isolation of nitric oxide synthetase, a calmodulin-requiring enzyme. *Proc. Natl. Acad. Sci. USA.* 1990; 87: 682–5.

22. Aquilano K, Barbato DL, Rosa CM. The multifaceted role of nitric oxide synthases in mitochondrial biogenesis and cell differentiation. *Commun. Integr. Biol*.2015; 8: 1–3.

23. Nahrevanian H. Involvement of nitric oxide and its up/down stream molecules in the immunity against parasitic infections. *Braz. J. Infect. Dis*.2009; (6): 440-8.

24. Ascenzi P, Bocedi A, Gradoni L. The anti-parasitic effects of nitric oxide. *IUBMB Life* .2003; 55: 573-8.

25. Fo¨rstermann U, Closs EI, Pollock JS, Nakane M, Schwarz P, Gath I, et al. Nitric oxide synthase isozymes: Characterization, purification, molecular cloning, and functions. *Hypertension* .1994; 23: 1121–31.

26. Rudic RD, Shesely EG, Maeda N, Smithies O, Segal SS, Sessa WC. Direct evidence for the importance of endothelium-derived nitric oxide in vascular remodeling. *J. Clin. Invest* .1998; 101: 731–6.

27. Maloney J, Keselman A, Li E, Singer SM. Macrophages expressing arginase 1 and nitric oxide synthase 2 accumulate in the small intestine during *Giardia lamblia* infection. *Microbes Infec*. 2015; t 6: 462-9.

28. Muro A, Pérez-Arellano JL. Nitric oxide and respiratory helminthic diseases. *J. Biomed. Biotechnol*. 2010; 1-8.

29. Lawrence CE, Paterson JC, Wei XQ, Liew FY, Garside P, Kennedy MW. Nitric oxide mediates intestinal pathology but not immune expulsion during *Trichinella spiralis* infection in mice. *J. Immunol* 2000; 164: 4229-34.

30. Muñoz-Carrillo JL, Muñoz-Escobedo JJ, Maldonado-Tapia CH, Chávez-Ruvalcaba F, Moreno-García MA. Resiniferatoxin lowers TNF-α, NO and PGE2 in the intestinal phase and the parasite burden in the muscular phase of *Trichinella spiralis* infection. *Parasite Immunol*. 2017; 39: 1.

31. Ilic N, Gruden-Movsesijan A, Sofronic-Milosavljevic L. *Trichinella spiralis:* shaping the immune response. *Immunol Res*. 2012; 52(1-2): 111-9.

32. Nagano I, Wu Y, Takahashi Y. Functional genes and proteins *of Trichinella* spp. *Parasitol.Res* 2009; 104:197–207.

33. Ishikawa N, Goyal PK, Mahida YR, Li FP, Wakelin D. Early cytokine responses during intestinal parasitic infections. *Immunology*.1998; 93: 257–63.

34.Vallance BA, Blennerhassett PA, Deng Y, Matthaei KI, Young IG, Collins SM. IL-5 contributes to worm expulsion and muscle hypercontractility in a primary *T.*

spiralis infection. *Am. J. Physiol*. 1999; 277(2 Pt1), G400–G8.
35. Bruschi F, Chiumiento L. Immunomodulation in trichinellosis: does *Trichinell*a really escape the host immune system? *Endocr. Metabol. Immune Dis.Drug Target.* 2012; 12: 4–15.
36. Jungery M, Clark NWT, Parkhouse RME. A major change in surface antigens during the maturation of newborn larvae of *Trichinella spiralis*. *Mol. Biochem. Parasitol.* 1983; 7: 101–9.
37. Stewart GL. Myopathogenesis and myoredifferentiation in trichinosis. *Bas. Appl. Myol*.1995; 5: 213–22.
38. Leiro J, Santamarina MT, Sernández L, Sanmartín ML, Ubeira FM. Immunomodulation by *Trichinella spiralis*: primary versus secondary response to phosphorylcholine-containing antigens. *Med.Microbiol. Immunol.* 1988; 177: 161–7
39. Bruschi F. The immune response to the parasitic nematode *Trichinella* and the ways to escape it. From experimental studies to implications for human infection. *Curr. Drug Targets Immun. Endocr. Metabol. Disord* .2002; 2: 269–80.
40. Watanabe N, Bruschi F, Korenaga M. IgE: a question of protective immunity in *Trichinella spiralis* infection. *Trends Parasitol*. 2005; 21(4): 175–8.
41. Bruschi F, Korenaga M, Watanabe N. Eosinophils and *Trichinella* infection: toxic for the parasite and the host? *Trends Parasitol*. 2008; 24: 462–7.
42.Bian K, Harari Y, Zhong M, Lai M, Castro G, Weisbrodt N. Down-regulation of inducible nitric-oxide synthase (NOS-2) during parasite- induced gut inflammation: a path to identify a selective NOS-2 inhibitor. *Mol. Pharmacol*. 2001; 59: 939–47.

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