Journal of the Egyptian Society of Parasitology, Vol.52, No.3, December 2022
J. Egypt. Soc. Parasitol. (JESP), 52(3), 2022: 431 – 442
(Online: 2090-2549)

TRICHINOSIS (TRICHINELLOSIS) IN MAN AND IN DOMESTIC AND WILD ANIMALS WITH REFERENCE TO EGYPT: AN OVERVIEW

By
TOSSON A. MORSY[1], TAREK ABDEL KADER SALLAM[2] and SARYA MOHAMED HAWAM[3]
Department of Parasitology, Faculty of Medicine, Ain Shams University, Cairo 11566[1], and Military Medical Academy, Cairo, 11291[2], Egypt (*Correspondence: tossonmorsy@med.asu.edu.eg, or morsyegypt2014@gmail.com, Orcid.org/0000-0003-2799 2049, **trksaam35@gmail.com, ***saryahawam@yahoo.co.uk)

Abstract

Trichinella spiralis is the smallest viviparous nematode parasite, occurring in rodents, pigs, bears, hyenas and humans, causing trichinosis. It is sometimes referred to as the pork worm due to typical encountered in undercooked pork products. It should not be confused with the distantly related pork tapeworm. Trichinosis (trichinellosis) causes headaches, fevers, myalgias, chills, cough, swelling of the face and eyes, aching joints and muscle pains, itchy skin, diarrhea, or constipation may follow the first symptoms. But, early clinical diagnosis is so difficult without specific trichinellosis signs or symptoms. Man becomes infected when eat raw or undercooked meats, particularly bear, pork, wild feline (such as a cougar), fox, dog, cat, wolf, horse, seal, or walrus may carry the parasite. Host animals' ingested even high numbers of *Trichinella* larvae from infectious meat don't develop clinical symptoms such as those occurred in human patients. Person-to-person spread does not occur. Mild to moderate infections most symptoms fatigue, weakness, muscle pain, and diarrhea may last for months subside within a few months. In heavy infection (myocarditis & encephalitis) patients may experience difficulty coordinating movements, with heart and breathing problems can be fatal. Mebendazole® and/or Albendazole® killed adults, and thus preventing more release of infected larvae and stopping infection within the patient.

Key words: Trichinosis, Man, Animals, Pathogenicity, Treatment, Prevention, Overview.

Introduction

Trichinellosis is caused by the zoonotic nematode parasite *Trichinella.* Although trichinellosis was reported worldwide, with prevalence of human infection is highest in China, Thailand, Mexico, Argentina, Bolivia, the former Soviet Union, and other parts of Central Europe (Jongwutiwes *et al*, 1998). Human trichinosis was reported in 55 (27.8%) countries worldwide, with infection in domestic animals, mainly in pigs, in 43 countries, and the wildlife in 66 countries (Pozio, 2007). Devleesschauwer *et al.* (2015) in Belgium reviewed data between 1986 and 2009, reported an estimated annual global incidence rate of 469.2 to 985.3 cases per billion persons, and annual global mortality rate was 0.300-0.828 per billion persons.

Zarlenga *et al.* (2020) in USA reported that globally *Trichinella* distribution was the geographical mirrors in domestic animals (pig, horse, dog) and wild ones (boars, bear, badger, cougar, jackal, walrus, lizard & turtle), the dietary habits (eating raw meat), and the countries social and economic development. Rostami *et al.* (2017) added that trichinosis outbreaks were reported in South East Asia countries (Cambodia, Thailand, and Vietnam) and South America (Argentina). Pozio (2014) reported low human trichinellosis prevalence in African Countries due to the dietary and religious habits. He added that despite the Islamic Religious Laws, infection was documented in the Muslim Countries such as Turkey. Akkoc *et al.* (2009) in Izmir reported a trichinellosis outbreak between January and March 2004 caused by consumption of raw meat balls made of beef deceptively mixed with *T. britovi* infected pork. The beef illegally mixed with pork of unknown origin, by a wholesale butcher who had sold this product to restaurants and street vendors at a lower price than

the prevailing market price of beef, was the cause of this large-scale outbreak in a country with a predominantly Muslim population.

Review and General Discussion

Conversely to the older conventional scientific recognition of *Trichinella spiralis* was the only member of the genus *Trichinella* (Campbell, 1983). There are eight species and 12 genotypes of *Trichinella*, and these are divided into those that encapsulate in host muscle tissue of mammals only, or those don't encapsulate and infect mammals, birds (one species), or reptiles. Seven species of *Trichinella* are recognized to infect humans, including *T. spiralis*, which is found worldwide in a great variety of carnivorous and omnivorous animals (Murrell and Pozio, 2000). The commonest *Trichinella* species which can cause human disease, *Trichinella spiralis*, although other *Trichinella* species implicated in human disease are: *T. nativa*, *T. nelson*, *T. britovi*, *T. pseudospiralis*, *T. murelli*, & *T. papuae* (Ortega-Pierres *et al,* 2000). There separate geographic regions: *T. nativa* is found in arctic regions and infects bears and foxes, *T. nelsoni* is present in Africa, south of Sahara, common in felid predators and scavenger animals, like hyenas and bush pigs, *T. britovi* is found in temperate areas of Europe and western Asia in carnivores but not domestic swine, *T. pseudospiralis* is found in mammals and birds worldwide (Ranque *et al,* 2000), *T. murrelli* is common in wild mammals in the States (Pozio and La Rosa, 2000). Jonwutiwes *et al.* (1998) in Thailand reported an outbreak by *T pseudospiralis* with patients' clinical course was unusually prolonged. *T. papuae* was reported in Papua New Guinea in domestic and feral pigs and in turtles and lizards from Thailand (Khumjui *et al,* 2008) and Taiwan (Lo *et al,* 2009). Human trichinosis was not reported with other species. *T. zimbabwensis* was common in the Tanzanian crocodiles (Pozio, 2003). Pozio *et al.* (2009) in Italy reported that *Trichinella* is a genus which members are easily propagated in the laboratories, have been used as models to investigate host-parasite relationships and parasitism among organisms, and represent a poorly investigated link between the Phylum Nematoda, and other Metazoans. The import ance of *T. spiralis* in better understanding the tree of life was so recognized that in 2004, its genome was carefully selected as one of only the nine key non-mammalian organisms to be sequenced to completion. Since it was discovered in 1835, this genus has expanded from mono-specific to eight species including four other genotypes of undetermined taxonomic rank. Information on species and genotype distribution and host range can be downloaded from the International Trichinosis.

Life cycle of all *Trichinella* species divides into two stages: 1- Domestic cycle and 2- Sylvatic cycle. The domestic one affects domestic animals, particularly swine, rodents, and horses, but the sylvatic one affects wildlife as bear, wild boar, and moose. It comprises a broad range of host species (mammals, birds, and reptiles), but only humans become clinically affected. Human infections are acquired by eating inadequately cooked meat contained *Trichinella* cysts. The major trichinosis cause is *T. spiralis* acquired by consumption of inadequately cooked pork from domestic pigs (Ancelle *et al,* 1993). For *T. spiralis* and the other *Trichinella* species, wild carnivorous animals harbor and perpetuate infections (sylvatic cycle). Thus, walrus, bear, cougar, and wild boar can also be sources of trichinellosis. Cattle are herbivores; they are not naturally infected, but meat beef can become contaminated, incidentally or deliberately, with larvae from pork during processing. Horsemeat has been associated with trichinellosis outbreaks (CDC, 2004). Since human infection is strongly associated with the consumption of raw or undercooked meat, cultural factors and food preferences play an important role in the disease epidemiology (Moorhead *et al,* 1999).

Naturally, trichinosis infection was reported from more than 100 species of mammals, 7 avian species, and 3 reptile species (Dick and

Pozio, 2001). In the United States, a total of 66 trichinellosis cases were reported between 2002 & 2007. Clusters of cases occurred when groups consumed meat from a common infected animal, but by improvements in swine production, infection declined steadily in the States. The undercooked wild game, predominantly bear meat re-emerged as a predominant source of trichinosis (Roy *et al,* 2003). Larvae in meat were rendered noninfectious by heating up to 77ºC, freezing at -15ºC for three weeks, as indoors freezer would also generally kill larvae, but arctic species were more resistant to freezing, and remained viable (Kociecka, 2000).

Clinical manifestations: The severity of infection generally correlates with the numbers of ingested larvae. Mild infections, with less than 10 larvae/g of muscle, can be subclinical. The clinical disease severity of *T. spiralis* infection (and other species) is strongly dependent on and directly correlated with the number of infective larvae ingested by the person or patient (Murrell and Bruschi, 1994). So, infection may result in a large spectrum of clinical forms range from asymptomatic to fatality.

With clinical infections, incubation period is generally 7 to 30 days, but the period varied with the invasive dose, how meat was prepared (raw, partially cooked), the host's immune status, and the species of *Trichinella* involved (Gould, 1970). Shorter incubation periods are generally associated with a more severe disease course. Muscles are mainly affected during the parenteral phase, including the myocardium. The central nervous system, lungs, kidney, and skin may be affected. Trichinellotic syndrome is characterized by facial oedema, muscle pain and swelling, weakness, and frequently fever; anorexia, headache, conjunctivitis, and urticaria less frequently (Pawlowski**,** 1983). Fever, usually remittent, generally begins at two weeks, and peaks after four weeks, with values up to 41°C in severe cases. But, despite fever, patients may appear in good condition. Ocular signs (oedema of eyelids, chemosis, conjunctivitis, conjuctival hemorrhages, disturbed vision, and ocular pain) at this time help in diagnosis. Periorbital oedema is peculiar to trichinellosis, ranged from 17% to 100% of patients in over 2000 trichinellosis cases reviewed. Oedema is probably resulted in an allergic response (Tassi *et al*, 1991).

With heavier infections, two stages may be recognized (MacLean *et al,* 1992): 1- Intestinal stage occurs between the second and seventh days after ingestion, when encysted larvae are liberated from the meat by gastric juices. They mature into adults that burrow into the intestinal mucosa. Fertilized females release new larvae at about one week after ingestion and continue for up to five weeks, depending upon the infection severity. This stage may be asymptomatic or may be accompanied by intestinal symptoms, including abdominal aches, nausea, vomiting, and prolonged diarrhea lasting for weeks was attributed to repeated reinfections in an infected and sensitized patient (Akar *et al,* 2007). 2- Muscle stage develops after the first week and represents the period when adult-derived larvae in intestines enter the bloodstream and disseminate hematogenously, larvae then enter skeletal muscle. For all species except *T. pseudospiralis*, each larva encysts within a host-derived cell, termed a nurse cell; *T. pseudospiralis* larvae remain in muscle without forming cysts. Encysted and free *Trichinella* larvae are viable for years.

During muscle phase of infection, the cardinal clinical findings of trichinellosis develop. These include subungual splinter hemorrhages, conjunctivas and retinal hemorrhages, periorbital edema and chemosis, disturbed vision, and ocular pain. Larvae enter skeletal muscles, pain, tenderness, swelling, and weakness occurs. Pain can be so extreme as to limit all movement, including the breathing or moving tongue with high fever lasting for weeks often occurred. The least manifestations were macular or urticarial rashes, headache, cough, dyspnea, and dysphagia.

Lazarević *et al.* (1999) in Turkey found that

in a *Trichinella* outbreak involved 98 patients, the most frequent musculoskeletal symptoms were muscle pain (87.8%), joint pain (84.7%), muscle weakness (76.5%), and joint movement' restriction (64.3%). Calves, upper arm, neck and shoulder girdle, and forearms were the most affected muscle groups. Muscle pain was found more frequently in the upper than in the lower extremities and during activity, but joint pain happened more frequently at the rest. But, without evidence of arthritis and objective muscle weakness was noted on physical examination in any patient.

Laboratory findings: During early intestinal stage there was no specific laboratory abnormalities. However, leukocytosis and eosinophilia appear during the second week of the muscle stage. Nonspecific findings were elevated serum muscle enzymes (creatine kinase & lactate dehydrogenase) and hypergammaglobulinemia (Ibrahim *et al,* 1981). Muscle enzymes serum levels measurement was a critical part in the evaluation of patients presented with weakness or myalgias, and important in monitoring the muscular disorders course and therapy responses. The CK, LDH, ALT, AST, and aldolase were serum enzymes measured in clinical practice (Bohlmeyer *et al,* 1994).

Zhang *et al.* (2012) in China reported that muscle enzymes sometimes can provide clues for many similar neuromuscular disorders. They added that muscle enzyme profiles in large cohort patients with neuromuscular diseases have some implications to make decision when diagnosis was hard. Yang *et al.* (2022) in China reported that immuno-mediated necrotising myopathy (IMNM) is a subset of idiopathic inflammatory myopathies (IIM) characterized by significantly elevated creating kinase level, muscle weakness and prdominant muscle fiber necrosis in muscle biopsy. They concluded that it was simple to distinguish IMNM from other IIM subtypes clinically and myositis specific antibodies profiles but, distinguishing IMNM from disorders clinically similar to non-IIM needs combined clinical, serological and histopathological features.

Eosinophilia is a hallmark of clinical trichinellosis present at some time in every case. Its proportion rose to a maximum of 20 to 90% in third or fourth week, without relationship between disease clinical course and eosinophil level increase (Gottstein *et al,* 2009). Eosinophilia may disappear in some heavily infected patients and considered to be a poor prognostic sign. Eosinopenia in this setting is probably due to superimposed bacterial infections and inflammation. Although larvae encyst only in skeletal muscle, there may be pulmonary, cardiac, or central nervous system involvement in risky infections, yet fatal in acute trichinosis usually was myocarditis, pneumonia or encephalitis (Compton *et al,* 1993).

Cardiac disease: Although cardiac involvement is not common, cardiac complications are the most frequent cause of death in severe infection (Fourestie *et al,* 1993). Larvae don't encyst in cardiac muscle, but elicit an eosinophil-enriched inflammatory response and myocarditis (Puljiz *et al,* 2005). Among 154 hospitalized trichinellosis patients 56% showed ECG abnormalities, mainly nonspecific ST-T wave changes without a poor prognosis (Mawhorter and Kazura, 1993), but life-threatening arrhythmias occurred and caused death in many trichinellosis myocarditis patients.

Neurology: Neurologic manifestations may include signs of meningitis or encephalitis developed in 10- 24% of severe trichinosis patients (Gelal *et al,* 2005). The neurologic disease may develop early or late, and can be diffuse or focal in nature. Headache is common and often exacerbated by movement. Pathologic findings can include edema, hemorrhage, emboli, infarctions, and perivascular infiltrates in fatal cases. CT & MRI may show multifocal small lesions located in cerebral cortex and white matter (Januszkiewicz, 1967). Genrally, neurotrichinosis was manifested with clinical symptoms, and signs of meningitis, encephalitis, polyradiculoneuritis, poliomyelitis, myastenia gravis, paresis and paralysis, with

the connective tissue clinical picture of systemic disease involved the nervous system and, extremely rare, as a sinus thrombosis (Nikolić *et al,* 1998). The most frequent CNS clinical features consisted of some non-specific meningo-encephalitic findings, such as headaches, confusion, spatial and temporal disorientation, and meningeal signs, including neck stiffness and Kerning signs. Psychiatric and behavioral disturbances were not very common and cognitive impairments were mainly in recent memory deficits (Batzlaff *et al,* 2014). Also, patients may show focal brain damage, indicated mainly by motor deficits, most frequently hemiparesis with relatively rare cerebellar and cranial nerve involvement (Rosca *et al,* 2021).

Pulmonary disease: Serious pulmonary involvement in trichinosis is infrequent recorded in only 6.5% of 856 hospitalized patients with acute disease (Robin *et al,* 1960). Pulmonary involvement may result from direct larva invades pulmonary tissues, myositis causing respiratory muscles, or secondary pyogenic pneumonia. Respiratory symptoms can lead to congestive heart failure due to myocarditis.

Direct pulmonary involvement: Early stage of muscle invasion, when intravascular larvae are passing through lungs, a dry, nonproductive cough a common symptom. Chest x-ray at this time may reveal patchy basilar infiltrates, small micro-nodular lesions, or pleural effusions. Radiographic findings resolve spontaneously over 1 to 2 weeks. Bronchitis was common between the third and fifth weeks of infection. Bronchitis was observed in 40% of patients in one epidemic, with many eosinophils' mucoid sputum (Guerra *et al,* 2016).

Respiratory myositis: Myositis develops in response to encysted larvae. Diaphragm usually has a relatively high encysted larvae density compared to other skeletal muscles (Cho *et al,* 2012). Diaphragmatic involvement can cause lower thoracic or epigastric pain and produce sufficient weakness of the diaphragm to compromise respiratory function. Painful intercostal myositis may further impair respiratory function. Symptoms are often most prominent in second & third weeks of severe infection. Symptomatic involvement of upper airway muscular may occur. Patients may present with hoarseness or dysphagia due to involvement of the laryngeal muscles or muscles of deglutition, respectively (Pozio, 2001).

Secondary pneumonia: Superimposed bacterial pneumonia due to prolonged bed rest and impaired pulmonary toilet may develop in hospitalized patients with trichinellosis in the later stages of infection (Eimori *et al,* 2016). Yan *et al.* (2019) reported that with people aging in China, total number of elderly people over 60 years exceeded 200million, and disabled elderly ones exceeds 33 million.

Course: With many *Trichinella* infections, progressive muscle encystment was associated with resolution of clinical manifestations even though they remained viable for several years before calcification and death (Capo and Despommier, 1996). An exception to this general rule was *T. pseudospiralis*, which larvae don't encyst. Human *T. pseudospiralis* infection showed manifestations such as fatigue, post-exercise weakness, and myositis for months up to years (Andrews *et al,* 1994).

Diagnosis: Trichinellosis should be considered in patients with periorbital edema, myositis, and eosinophilia. However, about half of died patients didn't show an increase in eosinophils count, and a lack of eosinophilia indicated a poor outcome in patients with a severe symptomatology (Neghina *et al,* 2011). Infection must be suspected in patients with symptoms and a history of ingesting either inadequately cooked meat, mainly pork, or meat ingested by other symptomatic individuals (Murrell and Bruschi, 1994).

Serologic tests: Many different serologic assays are available, including ELISA, indirect immunofluorescence, and latex agglutination, which are reliable, especially with ELISA the most sensitive one. Results can be confirmed with a Western blot. Antibody levels don't indicate early diagnosis since they are not dete-

cted except after three or more weeks of infection (Moskwa *et al,* 2009). Antibody tests may remain positive for years after cessation of clinical symptoms, and don't correlate with the clinical course severity. Other helminthes and autoimmune diseases can cause false positive data. Circulating antigens tests were developed, but with relatively poor sensitivity only 47% in trichinosis patients (Ivanoska *et al,* 1989). Animals can be tested by anti-*Trichinella* antibodies in serum or in meat juice either upon antemortem or upon postmortem examination, but serology was suitable for epidemiological surveillance of domestic and wildlife (Gamble *et al,* 2004). A multiplex PCR for *Trichinella* unequivocal differentiated species and genotypes, but not yet commercially available (Zarlenga *et al,* 2001).

Muscle biopsy: Definitive diagnosis by detecting larvae in biopsied muscle only for patients with doubted diagnosis, such as the yield were highest in symptomatic muscles near a tendinous insertion. Gullotta and Fröscher (1983) reported that in chronic trichinosis biopsy samples, morphological and enzyme-histochemical changes typical of a progressive neurogenic muscular atrophy were present and encapsulated but, still living, enzyme-positive parasites and signs of focal myositis were detected. Also, in routine histopathologic, muscle must be examined after enzymatic digestion to free larvae, but can be examined undigested in an unfixed muscle preparation compressed between two microscopic slides (Bruschi and Murrell, 2002). But, morphology didn't differentiate species, only with molecular genetic assays (Rombout *et al,* 2001).

Differential diagnosis: 1- Gastroenteritis, viral or bacterial, 2- Polymyositis and dermatomyositis (autoimmune), 3- Periorbital cellulitis, & 4- Eosinophilia-myalgia syndrome. But, eosinophilia can be present in other helminthes like fascioliasis, schistosomiasis, toxocariasis, cysticercosis, visceral larva migrans, and sarcocystosis (Abdel Fadil *et al,* 2018).

Treatment: Clinical course of most trichinellosis is uncomplicated and self-limited. Consequently, specific therapy is often not needed.

Symptomatic treatment with analgesia and antipyretics are appropriate for mild infections. For infections associated with inflammation in heart, CNS or other organs, principal therapy given is for anti-inflammatory corticosteroids (Ericson-Neilsen *et al,* 2014). Prednisone is given at a dose of 50 to 60mg/day for 10 to 15 days. But, contraindications included hypersensitivity to any component of the formulation, concurrent administration of live or when using immunosuppressive dosages, systemic fungal infection, osteoporosis, uncontrolled hyperglycemia, diabetes mellitus, glaucoma, joint infection, uncontrolled hypertension, herpes simplex keratitis, and varicella infection as well as peptic ulcer, congestive heart failure, and uncontrolled viral or bacterial infections (Liu *et al,* 2013).

Antiparasitic Mebendazole® (200 to400mg 3 times a day orally for 3 days, and then 400 to 500mg three times a day orally for 10 days or Albendazole® (400mg twice a day orally for 8 to 14 days) are appropriate in setting of symptomatic infection, mainly in complicated infections of CNS, myocardium, or respiratory muscles (CDC, 2020). Drugs were useful to treat larvae actively invading gastrointestinal tract, but their benefit in setting of bloodborne or muscle-encysted larvae was uncertain (Watt *et al,* 2000). Mebendazole treated patients with less myositis and muscle pain than those received placebo, but with viable encysted muscle larvae (Pozio *et al,* 2001).

Children and pregnant women: Larvae transplacental transmission didn't lead to symptomatic congenital infection and none reported serious intranatal or postnatal infections (Nuñez *et al,* 2002). Both mebendazole and albendazole was not generally advised during pregnancy (class C) or in children aged < 2 years (Dupouy-Camet *et al,* 2002). For both of them with intestinal and muscle stages, anthelminthic treatment usually not indicated. But, albendazole or mebendazole was used in pre-

gnant ones with severe infection without any adverse fetal effect (Gyorkos *et al,* 2006). In pregnancy, with severe trichinosis, corticosteroid must be given (Nuñez *et al,* 2008).

Pyrantel® (combantrin) is given in a single dose of 10 to 20mg/kg of body weight, repeated for 2 to 3 days. For pregnant women and children, it acted only against worms in gut, but without effect on newborn and muscle larvae (Dupouy-Camet and Murrell, 2007).

Prevention & Control: When humans fail to implement a proper management of domestic animals and wildlife, *T. spiralis*, *T. britovi* & *T. pseudospiralis* infections were transmitted from the sylvatic environment into the domestic one, sometimes via synanthropic (intermediary between domestic and sylvatic) animals (Pozio and Zarlenga, 2005). Once suspected human case, data on raw or undercooked meat or meat products consumption, including place and purchase time (or receipt) and consumption the health authorizes must be notified.

The best way to prevent trichinellosis is to cook meat to safe temperatures external icon. A food thermometer must measure the internal cooked meat temperature. Don't taste pigmeat until completely cooked. Also: 1- Wash hands with warm water and soap after handling raw meat, 2- Curing (salting), drying, smoking, or microwaving meat alone don't consistently kill infective worms; homemade jerky and sausage were the cause of many trichinellosis cases reported, 3- Freeze pork less than 6 inches thick for 20 days at 5°F (-15°C) to kill any worms, 4- Freezing wild game meats, unlike freezing pork products, may not effectively kill all worms because some worm species that infect wild game animals are freeze-resistant, 5- Clean meat grinders thoroughly after each use. 5- Don't feed pigs on garbage and preventing illegal slaughter outside the slaughterhouses Also, to prevent *Trichinella* infection in animal populations don't allow pigs or wild animals to eat uncooked meat, scraps, or carcasses of any animals, including rats that may be *Trichinella* infected (CDC, 2019).

Selected publications in Arab Countries: Therizol *et al.* (1975) in France reported that five acute trichinosis cases were imported from Egypt (four of them were anatomically and serologically proved) observed at the Ambroise-Paré Hospital. Among these patients, only one showed atypical clinical picture. Another one suffered from myocarditis, which made prognostic more severe. In 3/5 patients, immunological tests and blood eosinophilia were right, but one case remained doubtful or negative. Thiabenzadole associated with corticotherapy were effective. Also, Blancou (2001) in France stated that "the origin of trichinellosis, which existed in ancient times as testified by the discovery of parasite larvae on an Egyptian mummy, unfolded in several stages: discovery of encapsulated larvae (in 1820s), identification and scientific description of these larvae (Paget and Owen, 1835), followed by experimental infections of animals (dogs, pigs, rabbits, mice) or of humans as from 1850. The main occurrences of trichinellosis were followed with particular attention in Europe (Germany, Denmark, France, etc.) and in the United States of America at the end of the 19th Century.

Siam *et al.* (1979) reported that the sylvatic trichinosis was in the Mediterranean and African Regions with only domestic *T. spiralis* in Egypt. Morsy *et al.* (1981) Cairo reported *T. spiralis* in two street cats. Also, Morsy *et al.* (1980; 1989); Michael and Morsy (1980), Barakat *et al.* (1982); Azab *et al.* (1988), Abdel Aal *et al.* (1988); Makarem *et al.* (1989) and Loutfy *et al.* (1999) evaluated different available serologic tests and detected natural anti-*Trichinella* antibodies in pigs and/or rodents or carnivores in all the Nile Delta Governorates. Michael and Morsy (1984) recommended Oxfendazole® to treat intestinal larval phase. El Shazly *et al.* (2002) established mice immunization by a crude *T. spiralis* antigen. Hassanain *et al.* (2004) in Giza didn't find significant difference between 16 hospitalized symptomatic patients and 12 asymptomattic ones at sw-

ine slaughterhouse. They concluded that the 45.00, 75.355, &25.389 and 57.989 KDa poly peptides of adult antigen and 26.00, 24.00, & 46.994 KDa proteins of adult E/S products were diagnostic for human, swine and rat trichinellosis, respectively. Abdel-Hafeez *et al.* (2015) in Minia City reported among slaughtered 100 cattle, 100 goats, & 100 pigs, parasites were *Sarcocystis* spp, *Toxoplasma gondii, Taenia saginata, T. solium* and *T. spiralis*. Othman *et al.* (2016) in Tanta studied impact of host's biochemical environment upon trichinosis infection course by atorvastatin® and metformin®. They found that the oxidative stress and expression of vascular endothelial growth factor in muscles were significantly reduced with both drugs, but total larval counts in muscles were only significantly reduced in atorvastatin-receiving mice. Also, marked reduction in inflammatory cellular infiltration, cyclooxygenase-2 expression, and oxidative stress occurred in small intestines of treated mice compared to positive control, and concluded that anti-inflammatory and anti-angio-genic effects must be in mind when treating patients with atorvastatin or metformin. Saad *et al.* (2016) studied Acetazolamide® efficacy against different *T. spiralis* in mice; reported that it reduced adult and muscle larval counts, when given early (62.7%) against adults, efficacy for muscle larvae increased when given late (63%). They concluded that acetazolamide; carbonic anhydrase inhibitor proved effective against adults and larvae. Abou Rayia *et al.* (2017) evaluated the in vitro & in vivo Artemisinin® effect on *T. spiralis* versus mebendazole; found that adult teguments significantly degenerated and destructed with both drugs and significant reduction of both adult & larval counts. They reported that artemisinin can be an alternative drug for human cases.

Salama *et al.* (2021) assessed the antiparasitic and anti-inflammatory effects of *Citrus limon* and *Capsicum frutescens* on Swiss albino trichinosis compared to albendazole and prednisolone, found the efficacy of *C. frutescens* and *C. limon* extracts gave a marked decrease in adult and larval counts and decreased tumour necrosis factor-α levels in intestinal and muscular phases. They reported that both extracts proved promising for treatment of experi mental trichinosis, particularly *C. frutescens*. Fahmy and Diab (2021) in Giza reported that combined albendazole-mefloquine gave an almost complete recovery of albino mice, and its low dose regimen highly reduced parasite burden to normal histological architecture.

Mohammed *et al.* (2022) reported that there was a scarcity of Egyptian data on trichinosis in pigs and humans. Trichinoscopic diagnosed 33812 pigs slaughtered during a year at Al-Basateen Governmental Abattoir; showed a total prevalence of 1.06%, with maximum infection in autumn (1.18%). They added that in Qena and Sohag Governorates human trichinosis ELISA antibodies was 10%, with a significant age association. Sarhan *et al.* (2022) in Sharkia studied the efficacy of selenium (Se), Se nanoparticles (SeNPs) and Egyptian propolis compared to albendazole for treating murine trichinosis. They found that the combination of SeNPs and propolis had anti-inflammatory and anti-angiogenic effects on trichinosis. They added that combined therapies can be used as a natural alternative therapy to albendazole to treat zoonotic trichinosis.

In Lebanon, Haim *et al.* (1997) in a South Village reported trichinosis outbreak in January 1995 with a population of 800-1000 persons. Previous outbreaks occurred under very similar circumstances, which indicated a need to control and prevent pork meat trading was not under veterinary control, and to raise people awareness. Khalil *et al.* (2022) reported that trichinosis in the Lebanon outbreaks date back to late 19th Century. The first published outbreaks were attributed to the consumption of wild boar meat, while those that followed incriminated pork. They added that hunting wild boar was currently re-emerging in Lebanon given the recent economic crisis that was

limited the purchase of livestock meat.

In Palestine, Abuseir (2021) found that many parasitic infections mainly transmitted by pork, although widespread worldwide, were rare in the Arab Countries, primarily due to religious proscriptions.

Conclusion

Zoonotic trichinosis infection is not commonly encountered in Egypt. Infection is characterized by generalized fever, abdominal pain, diarrhea, nausea, vomiting, or myalgias, or severe like myocarditis and encephalitis. But, it must be differentiated from gastroenteritis and some helminthiasis. Only domestic *T. spiralis* infection was reported in pigs, cats, and wild carnivores.

Mebendazole and Albendazole proved to be effective, but not advised during pregnancy or little infants.

Authors' declaration: Authors declared thatthey neither have conflict of interest nor received fund.

References

Abdel Aal, TM, Azab, ME, Morsy, TA, Makarem, SS, El Hady, HM, *et al,* 1988: Humoral and cellular responses in experimental trichinosis. J. Egypt. Soc. Parasitol. 18, 2:519-28

Abdel Fadil, EE, Ismail, MAM, Morsy, TA, 2018: Eosinophilia with special reference to helminthiasis. J. Egypt. Soc. Parasitol. 48, 2:465-74

Abdel-Hafeez, EH, Kamal, AM, Abdelgelil, N H, Abdel-Fatah, M, 2015: Parasites transmitted to human by ingestion of different types of meat, El-Minia City, El-Minia Governorate, Egypt. J. Egypt. Soc. Parasitol. 45, 3:671-80.

Abou Rayia, DM, Saad, AE, Ashour, DS, Oreiby, RM, 2017: Implication of artemisinin nematocidal activity on experimental trichinellosis: In vitro & in vivo studies. Parasitol Int. 66, 2:56-63.

Abuseir, S, 2021: Meat-borne parasites in the Arab world: A review in a one health perspective. Parasitol. Res. 120, 12:4153-66.

Akar, S, Gurler, O, Pozio, E*, et al,* 2007: Frequency and severity of musculoskeletal symptoms in humans during an outbreak of trichinellosis caused by *Trichinella britovi*. J. Parasitol. 93:341-8.

Akkoc, N, Kuruuzum, Z, Akar, S, Yuce, A, Onen, F, *et al,* 2009: A large-scale outbreak of trichinellosis caused by *Trichinella britovi* in Turkey; Izmir Trichinellosis Outbreak Study Group. Zoonoses Publ. Hlth. 56, 2:65-70

Ancelle, T, Dupouy-Camet, J, Desenclos, JC, *et al,* 1993: A multifocal outbreak of trichinellosis linked to horse meat imported from North America to France in 1993. Am. J. Trop. Med. Hyg. 59: 615-8.

Andrews, J, Ainsworth, R, Abernethy, D, 1994: *Trichinella pseudospiralis* in humans: Description of a case and its treatment. Trans. R. Soc. Trop. Med. Hyg. 88:200-6.

Azab, ME, Morsy, TA, Abdel-Aal, TM, Safar, EH, Makaram, SS, *et al,* 1988: Current prevalence of trichinosis in pigs in Egypt. J. Egypt. Soc. Parasitol. 18, 2:383-9.

Barakat, RM, El-Sawy, MF, Selim, MK, Raswan, A, 1982: Trichinosis in some carnivores and rodents from Alexandria, Egypt. J. Egypt. Soc. Parasitol. 12, 2:445-51.

Batzlaff, CM, Pupaibool, J, Sohail, MR, 2014: Acute renal failure associated with albendazole therapy in a patient with trichinosis. BMJ Case Rep. 2014 doi: 10.1136/bcr-2013-200668

Blancou, J, 2001: History of trichinellosis surveillance. Parasite 8, 2:S16-9.

Bohlmeyer, TJ, Wu, AH, Perryman, MB, 1994: Evaluation of laboratory tests as a guide to diagnosis and therapy of myositis. Rheum. Dis. Clin. North Am. 20:845-50.

Bruschi, F, Murrell, KD, 2002: New aspects of human trichinellosis: The impact of new *Trichinella* species. Postgrad. Med. J. 78:15-20.

Campbell, WC, 1983: Modes of transmission. In: *Trichinella* and Trichinosis. Plenum Press, London, UK.

Capo, V, **Despommier, DD, 1996:** Clinical aspects of infection with *Trichinella* spp. Clin. Microbiol. Rev. 9:47-54.

CDC, 2004: Trichinellosis associated with bear meat--New York and Tennessee, 2003. MMWR. Morb. Mortal. Wkly. Rep. 53:606-10.

CDC, 2019: Parasites- Trichinellosis (also known as Trichinosis) Prevention & Control. https://www. cdc.gov› parasites›.

CDC, 2020: Parasites- Trichinosis. https://www. cdc.gov› parasites›.

Cho, MK, Park, MK, Kang, SA, Choi, SH, Ahn, SC, *et al,* 2012: *Trichinella spiralis* infection suppressed gut inflammation with CD4 (+), CD25 (+), Foxp3 (+) T cell recruitment. Kor. J. Parasitol. 50, 4:385-90.

Compton, SJ, Celum, CL, Lee, CL, *et al,* 1993: Trichinosis with ventilator failure and persistent myocarditis. Clin. Infect. Dis. 16:500-8.
Devleesschauwer, B, Praet, N, Speybroeck, N, Torgerson, PR, Haagsma, JA, *et al*, 2015: The low global burden of trichinellosis: Evidence and implications. Int. J. Parasitol. 45:95-9.
Dick, TA, Pozio, E, 2001: *Trichinella* spp. and trichinellosis. In: Parasitic Diseases of Wild Mammals. Samuel, WM, Pybus, MJ, Kocan, AA (ed.) 2nd ed. Iowa State University Press, Ames, IA
Dupouy-Camet, J, Kociecka, W, Bruschi, F, *et al,* 2002: Opinion on the diagnosis and treatment of human trichinellosis. Expert. Opin. Pharmacother. 3:1117-22.
Dupouy-Camet, J, Murrell, KD, 2007: Management and diagnosis of human trichinellosis. FAO/ WHO/OIE guidelines for the surveillance, management, prevention and control of trichinellosis: World Organization for Animal Health Press, Paris, France. Geneva, Switzerland.
Eimori, K, Endo, N, Uchiyama, S, Takahashi, Y, Kawashima, H, *et al,* 2016: Disrupted bone metabolism in long-term bedridden patients. PLoS One 11:e0156991.
El Shazly, AM, El Shewey, Kh, El Hamshary, E, Habib, FSM, El Garhy, MF, *et al,* 2002: Mice immunization using crude *Trichinella spiralis* antigen. J. Egypt. Soc. Parasitol. 32, 2:391-403.
Ericson-Neilsen W, Kaye AD, 2014: Steroids: Pharmacology, complications, and practice delivery issues. Ochsner. J. 14, 2:203-7**.**
Fahmy, AM, Diab, TM, 2021: Therapeutic efficacy of albendazole and mefloquine alone or in combination against early and late stages of *Trichinella spiralis* infection in mice. Helminthologia 58, 2:179-87.
Fourestie, V, Douceron, H, Brugieres, P, *et al,* 1993: Neurotrichinosis: A cerebrovascular disease associated with myocardial injury and hypereosinophilia. Brain 116, Pt 3:603-9.
Gamble, HR, Pozio, E, Bruschi, K, Nöckler, C M, Kapel, O, *et al,* 2004: International Commission on Trichinellosis: Recommendations on use of serological tests for the detection of *Trichinella* infection in animals and man. Parasite 11:3-13.
Gelal, F, Kumral, E, Vidinli, BD, *et al,* 2005: Diffusion-weighted and conventional MR imaging in neurotrichinosis. Acta Radiol. 46:196-200.
Gottstein, B, Pozio, E, Nöckler, K, 2009: Epidemiology, diagnosis, treatment, and control of trichinellosis. Clin. Microbiol. Rev. 22:127-32.
Gould, SE, 1970: Clinical manifestations. In: Trichinosis in Man and Animals, Gould, SE (Ed.), Charles C Thomas, Springfield, IL.
Guerra, M, Crichiutti, G, Pecile, P, Romanello, C, Busolini, E, *et al,* 2016: Ultrasound detection of pneumonia in febrile children with respiratory distress: A prospective study. Eur. J. Pediatr. 175, 2:163-7.
Gyorkos, TW, Larocque, R, Casapia, M, Gotuzzo, E, 2006: Lack of risk of adverse birth outcomes after deworming in pregnant women. Pediatr. Infect. Dis. J. 25:791-9.
Gullotta, F, Fröscher, W, 1983: Chronic trichinosis, and neuromuscular diseases: Morphologic and pathogenic aspects. Arch. Psychiat. Nervenkr. 232, 6:479-87.
Haim, M, Efrat, M, Wilson, M, Schantz, PM, Cohen, D, *et al,* 1997: An outbreak of *Trichinella spiralis* infection in southern Lebanon. Epidemiol. Infect. 119, 3:357-62.
Hassanain, MA, Hassanain, NA, El-Mogazy, FM, 2004: Identification of immunoreactive proteins of Trichinella spiralis adult and adult excretory/secretory (E/S) antigens in sera of human and animals. J. Egypt. Soc. Parasitol. 34, 1:281-95.
Ibrahim, GA, Zweber, BA, Awad, EA, 1981: Muscle and serum enzymes and isoenzymes in muscular dystrophies. Arch. Phys. Med. Rehabil. 62, 6:265-9.
Ivanoska, D, Cuperlović, K, Gamble, HR, Murrell, KD, 1989: Comparative efficacy of antigen and antibody detection tests for human trichinellosis. J. Parasitol. 75:38-43.
Januszkiewicz, J, 1967: Participation of the respiratory system in trichinosis. Epidemiol. Rev. 21: 169-72.
Jongwutiwes, S, Chantachum, N, Kraivichian, P, *et al,* 1998: First outbreak of human trichinellosis caused by *Trichinella pseudospiralis*. Clin. Infect. Dis, 26:111-8.
Jonwutiwes, S, **Chantachum, N, Kraivichian, P, *et al*, 1998: First** outbreak of human trichinellosis caused by *Trichinella pseudospiralis*. Clin. Infect. Dis. 26:111-5
Khalil, G, Marty, P, Hage, K, Sfeir, S, El Hage, J, *et al,* 2022: Could the re-emerging practice of wild boar hunting linked to the recent economic crisis lead to new outbreaks of trichinellosis in Lebanon? Parasite 29:11. doi: 10.1051/parasite/2022 2011.

Khumjui, C, Choomkasien, P, Dekumyoy, P, *et al,* 2008: Outbreak of trichinosis caused by *Trichinella papuae*, Thailand, 2006. Emerg. Infect. Dis. 14:1913-8.

Kociecka, W, 2000: Trichinosis: Human disease, diagnosis and treatment. Vet. Parasitol. 93:365-8.

Lazarević, AM, Nesković, AN, Goronja, M, *et al,* 1999: Low incidence of cardiac abnormalities in treated trichinosis: a prospective study of 62 patients from a single-source outbreak. Am. J. Med. 107:18-22.

Liu, D, Ahmet, A, Ward, L, Krishnamoorthy, P, Mandelcorn, ED, *et al,* 2013: A practical guide to the monitoring and management of the comp lications of systemic corticosteroid therapy. Allergy Asthma Clin. Immunol. 9, 1:30-8.

Lo, YC, Hung, CC, Lai, CS, *et al,* 2009: Human trichinosis after consumption of soft-shelled turtles, Taiwan. Emerg. Infect. Dis. 15:2056-60.

Loutfy, NF, Awad, OM, El-Masry, AG, Kandil, GM, 1999: Study on rodents' infestation in Alexandria and prevalence of *Trichinella spiralis* infection among them. J. Egypt. Soc. Parasitol. 29: 897-909.

MacLean, JD, Poirier, L, Gyorkos, TW, *et al,* 1992: Epidemiologic and serologic definition of primary and secondary trichinosis in the Arctic. J. Infect. Dis. 165:908-12.

Makarem, SS, Morsy, TA, Abou El Seoud, SF, 1989: The use of three immunologic tests in detection of antibodies against *Trichinella spiralis.* J. Egypt. Soc. Parasitol. 19, 1:331-6

Mawhorter, SD, Kazura, JW, 1993: Trichinosis of the central nervous system. Semin. Neurol. 13: 148-51.

Michael, SA, Morsy, TA, 1980: Studies on trichinosis indirect haemagglutination tests in experimentally infected albino rats. J. Egypt. Soc. Parasitol. 10, 1:247-54.

Michael, SA, Morsy, TA, 1984: Therapeutic effect of Oxfendazole on the intestinal phase of *Trichinella spiralis* larvae. J. Egypt. Soc. Parasitol. 14, 1:151-4.

Mohammed, ES, Youseef, AG, Mubarak, AG, Mawas, AS, Khalifa, FA, *et al,* 2022: Epidemiological perspective associated with principal risk factors of *Trichinella spiralis* infection in pigs and humans in Egypt. Vet World. 15, 6:1430-7.

Moorhead, A, Grunenwald, PE, Dietz, VJ, Schantz, PM, 1999: Trichinellosis in the United States, 1991-1996: Declining but not gone. Am. J. Trop. Med. Hyg. 60:66-9.

Morsy, TA, Michael, SA, Aboul Seoud, SF, 1980: Trichinosis antibodies in rodents collected at Suez Canal Zone, A.R.E. J. Egypt. Soc. Parasitol. 10, 2:259-63.

Morsy, TA, Sadik, MS, Abdel-Hamid, NY, 1981: Internal parasites of stray cats in Cairo, Egypt. J. Egypt. Soc. Parasitol. 11: 331-346.

Morsy, TA, Makarem, SS, Aboul Seoud, SF, Ambroise-Thomas, P, 1989: Comparison of three immuno-diagnostic tests in detection of trichinosis in naturally infected rats. J. Egypt. Soc. Parasitol. 19, 2:465-70.

Moskwa, B, Bien, J, Cabaj, W, *et al,* 2009: The comparison of different ELISA procedures in detecting anti-*Trichinella* IgG in human infections. Vet. Parasitol. 159:312-6.

Murrell, KD, Bruschi, F, 1994: Clinical trichinellosis. In: Progress in Clinical Parasitology, Sun, T (Ed.), CRC Press, Boca Raton.

Murrell, KD, Pozio, E, 2000: Trichinellosis: The zoonosis that won't go quietly. Int. J. Parasitol. 30: 1339-42.

Neghina, R, Neghina, AM, Marincu, I, Iacobiciu, I, 2011: Reviews on trichinellosis (II): Neurological involvement. Foodborne Pathog. Dis. 8: 579-85.

Nuñez, GG, Costantino, SN, Gentile, T, Venturiello, SM, 2008: Immunoparasitological evaluation of *Trichinella spiralis* infection during human pregnancy: A small case series. Trans. R. Soc. Trop. Med. Hyg. 102:662-8.

Nikolić, S, Vujosević, M, Sasić, M, Poluga, J, Misić, S, *et al,* 1998: Neurologic manifestations in trichinosis. Srp. Arh. Celok. Lek. 126, 5/6:209-13.

Nuñez, GG, Gentile, T, Calcagno, ML, Venturiello, SM, 2002: Increased parasiticide activity against *Trichinella spiralis* newborn larvae during pregnancy. Parasitol. Res. 88:661-8.

Ortega-Pierres, MG, Arriaga, C, Yépez-Mulia, L, 2000: Epidemiology of trichinellosis in Mexico, Central and South America. Vet. Parasitol. 93, 3/4:201-25.

Othman, AA, Abou Rayia, DM, Ashour, DS, Saied, EM, Zineldeen, DH, *et al,* 2016: Atorvastatin and metformin administration modulates experimental *Trichinella spiralis* infection. Parasitol Int. 65, 2:105-12.

Pawlowski ZS, **1983:** Clinical aspects in man. In: *Trichinella* and Trichinosis. Campbell, WC, Ed. New York: Plenum Press.

Pozio, E, 2001: New patterns of *Trichinella* infection. Vet. Parasitol. 98:133-8.
Pozio, E, 2003: Foodborne and waterborne parasites. Acta Microbiol. Pol. 52, l:S83-6.
Pozio, E, 2007: World distribution of *Trichinella* spp. infections in animals and humans. Vet. Parasitol. 149:3-21.
Pozio, E, 2014: Searching for *Trichinella*: Not all pigs are created equal. Trends Parasitol. 30:4-11.
Pozio, E, La Rosa, G, 2000: *Trichinella murrelli* new species: Etiological agent of sylvatic trichinellosis in temperate areas of North America. J. Parasitol. 86:86:134-8.
Pozio, E, Zarlenga, DS, 2005: Recent advances on the taxonomy, systematics and epidemiology of *Trichinella*. Int. J. Parasitol. 35:1191-204.
Pozio, E, Sacchini, D, Sacchi, L, *et al*, 2001: Failure of mebendazole in the treatment of humans with *Trichinella spiralis* infection at the stage of encapsulating larvae. Clin. Infect. Dis. 32:638-42.
Pozio, E, Hoberg, E, La Rosa, G, Zarlenga, DS, 2009: Molecular taxonomy, phylogeny and biogeography of nematodes belonging to the *Trichinella* genus. Infect. Genet. Evol. 9, 4:606-16.
Puljiz, I, Beus, A, Kuzman, I, Seiwerth, S, 2005: Electrocardiographic changes and myocarditis in tric- hinellosis: A retrospective study of 154 patients. Ann. Trop. Med. Parasitol. 99:403-8.
Ranque, S, Faugère, B, Pozio, E, *et al,* 2000: *Trichinella pseudospiralis* outbreak in France. Emerg. Infect. Dis. 6:54350.
Robin, ED, Crump, CH, Wagman, RJ, 1960: Low sedimentation rate, hypofibrinogenemia and restrictive pseudo-obstructive pulmonary disease associated with trichinosis. N. Engl. J. Med. 262: 758-62.
Rombout, YB, Bosch, S, Van Der Giessen, JW, 2001: Detection and identification of eight *Trichinella* genotypes by reverse line blot hybridization. J. Clin. Microbiol. 39:642-6.
Rosca, EC, Tudor, R, Cornea, A, Simu, M, 2021: Central nervous system involvement in trichinellosis: A systematic review. Diagnostics (Basel); Jun, 11(6): 945. doi: 10.3390/diagnostics.
Rostami, A, Gamble, HR, Dupouy-Camet, J, Khazan, H, Bruschi, F, 2017: Meat sources of infection for outbreaks of human trichinellosis. Food Microbiol. 64:65-71.
Roy, SL, Lopez, AS, Schantz, PM, 2003: Trichinosis Surveillance: United States, 1997-2001. M-MWR. Surveill. Summ. 52:1-8.
Saad, AE, Ashour, DS, Abou Rayia, DM, Bedeer, AE, Saad AE, 2016: Carbonic anhydrase enzyme as a potential therapeutic target for experimental trichinellosis. Parasitol. Res. 115, 6:2331-9.
Salama, MAM, Mostafa, NE, Abd El-Aal, NF, Mostafa, EM, Hammad, SK, *et al,* 2021: *Capsicum frutescens* and *Citrus limon*: A new take on therapy against experimental trichinellosis. J. Helminthol. May 25; 95:e26. doi: 10.1017/S0022149.
Sarhan, MH, Farghaly, A, Abd El-Aal, NF, Farag, SM, Ali, AA, *et al,* 2022: Egyptian propolis and selenium nanoparticles against murine trichinosis: A novel therapeutic insight. J. Helminthol. Jul 20; 96:e50 doi: 10.1017/S0022149X22000359.
Siam, MA, Michael, SA, Ghoneim, NH, 1979: Studies on the isolation of the infective stages of *Trichinella spiralis* and *Toxoplasma gondii* from fresh and processed pork in Egypt. J. Egypt. Publ. Hlth. Assoc. 54:163-70
Therizol, M, Levy, R, Coulbois, J, Brochard, A, Bétourné, A, 1975: Acute trichinosis: Several recent cases imported from Egypt. Bull. Soc. Pathol. Exot. Filiales 68, 4:407-15.
Watt, G, Saisorn, S, Jongsakul, K, *et al,* 2000: Blinded, placebo-controlled trial of antiparasitic drugs for trichinosis myositis. J. Infect. Dis. 182: 37-4.
Yan, HM, Zhang, M, Zhang, X, Xia, YT, Shen, T, *et al,* 2019: Study of epidemiology characteristics of metabolic syndrome and influencing factors in elderly people in China. Zhonghua Liu Xing Bing Xue Za Zhi. 40:284-9
Yang, H, Tian, X, Zhang, L, Li, W, Liu, Q, *et al,* 2022: Clinical and pathological features of immune-mediated necrotizing myopathies in a single centre muscle biopsy cohort. BMC Musculoskelet Disord. May 6;23(1):425. doi: 10.1186/s12891-022-05372-z
Zhang Y, Huang, JJ, Wang, ZQ, Wang, N, Wu, ZY, 2012: Value of muscle enzyme measurement in evaluating different neuromuscular diseases. Clin. Chim. Acta 413, 3/4:520-4.
Zarlenga, DS, Chute, MB, Martin, A, Kapel, C M, 2001: A single, multiplex PCR for differentiating all species of *Trichinella.* Parasite 8:S24-8.
Zarlenga, D, Thompson, P, Pozio, E, 2020: *Trichinella* species & genotypes. Res. Vet. Sci. 133: 289-96.