Invited Review

History of the discovery of the life cycle of Toxoplasma gondii

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ABSTRACT

It has been 100 years since the discovery of Toxoplasma gondii in 1908. Its full life cycle was not discovered until 1970 when it was found that it is a coccidian parasite of cats with all non-feline warm blooded animals (including humans) as intermediate hosts. The discovery of the environmentally resistant stage of the parasite, the oocyst, made it possible to explain its worldwide prevalence. In the present paper, events associated with the discovery of its life cycle are recalled.

1. Introduction

Until 1970 only asexual stages (tachyzoites or trophozoites, bradyzoites or cysts) of Toxoplasma gondii were known. Its sexual cycle and the environmentally resistant stage, the oocyst, were discovered in 1970. During the past 40 years, I had the fortune of being associated with many aspects of the discovery of the life cycle of this parasite. I will attempt to review these events of the finding of the sexual phase of the parasite and its impact on disease and control.

Toxoplasma gondii was found by scientists working with Leishmania or viruses (Nicolle and Manceaux, 1908, 1909; Splendore, 1908). Transmission of the parasite was a mystery ever since its discovery in the rodent Ctenodactylus gundi. Chatton and Blanc (1917) found that gundis were not infected naturally, but acquired infection in captivity. Gundis live in the foothills and mountains of southern Tunisia. They were used in research on leishmaniasis at the Pasteur Institute in Tunis. Chatton and Blanc (1917) suspected that T. gondii was transmitted by arthropods because it was found in the blood of the host. Chatton and Blanc (1917) in Tunis and Woke et al. (1953) and others in the USA investigated possible transmission by several species of arthropods with essentially unsuccessful results (Frenkel, 1970, 1973).

2. Transmission

2.1. Congenital

Three pathologists, Wolf, Cowen and Paige from New York, USA, first conclusively identified T. gondii in an infant girl who was delivered full-term by Caesarean section on May 23, 1938 at Babies Hospital, New York (Wolf et al., 1939a,b). The girl developed convulsive seizures at 3 days of age and lesions were noted in the maculae of both eyes through an ophthalmoscope. She died when 1 month old and an autopsy was performed. At post mortem, brain, spinal cord and the right eye were removed for examination. Free and intracellular T. gondii were found in lesions of encephalomyelitis and retinitis of the girl. Portions of cerebral cortex and spinal cord were homogenized in saline and inoculated intracerebrally into rabbits and mice. These animals developed encephalitis. Toxoplasma gondii was demonstrated in their neural lesions and T. gondii from these animals was successfully passaged into other mice.

Congenital transmission was later found to occur in many species of animals, particularly sheep (Hartley and Marshall, 1957) and rodents. Congenital infections can be repeated in some strains of mice (Beverley, 1959), with infected mice producing congenitally infected offspring for at least 10 generations. Beverley discontinued his experiments because of high mortality in some lines of congenitally infected mice and because the progeny from the last generation of infected mice were seronegative and presumed not to be infected with T. gondii. Jacobs (1964) repeated these experiments and found that congenitally infected mice may be infected but might not develop antibodies due to immune tolerance.
et al. (1995) isolated viable T. gondii from seronegative naturally-infected mice. These findings are of epidemiological significance because serological surveys in mice underestimate the prevalence of T. gondii in mice.

2.2. Carnivorous

Weinman and Chandler (1954) suggested that transmission might occur through the ingestion of undercooked meat. Jacobs et al. (1960) provided evidence to support this idea by demonstrating the resistance to proteolytic enzymes of T. gondii derived from tissue cysts. They found that the cyst wall was immediately dissolved by such enzymes but the released bradyzoites survived long enough to infect the host. This hypothesis of transmission through the ingestion of infected meat was experimentally tested by Desmonts et al. (1965) in an experiment with children in a Paris sanatorium. They compared the acquisition rates of T. gondii infection in children before and after admission to the sanatorium. The 10% yearly acquisition rate of T. gondii antibody rose to 50% after adding two portions of barely cooked beef or horse meat to the daily diet and to a 100% yearly rate after the addition of barely cooked lamb chops. Since the prevalence of T. gondii is much higher in sheep than in horses or cattle this illustrated the importance of carnivores in transmission of T. gondii. Epidemiological evidence indicates it is common in humans in some localities where raw meat is routinely eaten. In a survey in Paris, Desmonts et al. (1965) found over 80% of the adult population sampled had antibodies to T. gondii. Kean et al. (1965) described toxoplasmosis in a group of medical students who had eaten under-cooked hamburgers.

2.3. Faecal-oral, sexual cycle, oocyst

While congenital transmission and carnivorous can explain some of the transmission of T. gondii it does not explain the widespread infection in vegetarians and herbivores. A study in Bombay, India found the prevalence of T. gondii in strict vegetarians to be similar to that in non-vegetarians (Rawal, 1959). Hutchison (1965) first discovered T. gondii infectivity associated with cat faeces. Bill Hutchison was a parasitologist in the Department of Biology, Strathclyde University, Glasgow, Scotland. He had never researched protozoa before. In a preliminary experiment, Hutchison (1965) fed T. gondii tissue cysts to a cat infected with the nematode Toxocara cati and collected faeces containing nematode ova. Faeces floated in 33% zinc sulphate solution and stored in tap water in an open beaker for 12 months to embryonate T. cati eggs induced toxoplasmosis in mice. This discovery was a breakthrough because, until then, both known forms of T. gondii (i.e. tachyzoites and bradyzoites) were killed by water. Microscopic examination of faeces revealed only T. cati eggs and Isospora oocysts. In Hutchison’s report, T. gondii infectivity was not attributed to oocysts or T. cati eggs. He repeated the experiment with two T. cati-infected and two T. cati-free cats. Toxoplasma gondii was transmitted only in association with T. cati infection. On this basis, Hutchison (1967) hypothesized that T. gondii was transmitted through nematode ova. He suspected transmission of T. gondii through the eggs of the nematode Toxocara similar to the transmission of the fragile flagellate Histomonas through Heterakis eggs. Hutchison initially wanted to test the nematode theory using Toxocara canis and T. gondii transmission in the dog but decided on the cat and T. cati model because there was no place to house dogs (see Dubey, 2008). Transmission of T. gondii by T. cati eggs made more sense because of the known zoonotic potential of T. canis; T. cati was not, at that time, known to infect humans but T. canis was. Discovery of the life cycle of T. gondii would have been delayed if Hutchinson had worked with dogs instead of cats. I had known of Hutchison’s discovery in September, 1964, a year before its publication and it profoundly affected my research (Dubey, 1996).

Hutchison’s (1965) report stimulated other investigators to examine faecal transmission of T. gondii through T. cati eggs (Dubey, J.P., 1966. Toxoplasmosis and its transmission in cats with special reference to associated Toxocara cati infestations. Ph.D. Thesis. University of Sheffield, England; Dubey, 1968; Hutchison et al., 1968; Frenkel et al., 1969; Sheffield and Melton, 1969). The nematode egg theory of transmission was discarded after Toxoplasma infectivity was dissociated from T. cati eggs (Frenkel et al., 1969) and Toxoplasma infectivity was found in faeces of worm-free cats fed T. gondii (Frenkel et al., 1969; Sheffield and Melton, 1969). Finally, in 1970, knowledge of the T. gondii life cycle was completed by discovery of the sexual phase of the parasite in the small intestine of the cat (Frenkel et al., 1970). Toxoplasma gondii oocysts, the product of schizogony and gametogony, were found in cat faeces and characterized morphologically and biologically (Dubey et al., 1970a,b).

2.4. Discovery of oocyst


Although discovery of the oocyst stage in the life cycle of a coccidian parasite would be expected, proving T. gondii to be a coccidian parasite was a big challenge at that time for the following reasons. Firstly, faecal infectivity (oocyst) was linked with Toxocara infectivity (Hutchison, 1965, 1967). Second, the oocyst had been called a new cyst (Work and Hutchison, 1969). Toxoplasma gondii oocysts were morphologically identical to oocysts of the previously described coccidian parasite (Isospora bigemina) of cats and dogs (Dubey et al., 1970a). In light of these facts, the evidence provided by us to link T. gondii oocysts to faecal infectivity is noteworthy (Frenkel et al., 1970; Dubey et al., 1970a). We subjected the newly discovered faecal stage (oocyst) to the following mutually independent tests to accumulate critical evidence for or against its identity to T. gondii (Dubey et al., 1970a): (i) the use of newborn kittens and littermate controls were used to avoid as far as possible pre-existing coccidial infections; (ii) comparison of the development of oocysts and of infectivity in relation to heat, cold, oxygenation and chemicals; (iii) comparison by filtration of the size of the infection’s entity with oocysts size; (iv) comparison of the density characteristics of oocysts and of infectivity; (v) comparison of the eukaryotic characteristics of oocysts and infectivity; (vi) antigenic comparison of oocysts with the standard RH strain of Toxoplasma by means of the fluorescent antibody test; (vii) identification of the endogenous cycle preceding the development of oocysts and linking it antigenically to Toxoplasma infectivity of oocysts before and after excystation; (viii) comparison of the appearance of oocysts and Toxoplasma infectivity in faeces of cats after feeding of cysts, trophozoites and oocysts (Dubey et al., 1970a).

In retrospect the discovery and characterization of the T. gondii oocyst in cat faeces was also delayed because until 1970 coccidian oocysts were sporulated in 2.5% potassium dichromate. Chromation of the oocyst’s wall interfered with excystation of the sporozoites when oocysts were fed to mice and thus the mouse infectivity titer of the oocysts was lower than expected from the number of oocysts administered (Dubey et al., 1970a). These findings led to the use of 2% sulphuric acid as the best medium for sporulation
and storage of *T. gondii* oocysts. Unlike dichromate, which was difficult to wash off the oocysts, sulphuric acid could be easily neutralized and the oocysts could be injected into mice without washing (Dubey et al., 1972). Unlike other coccidians, *T. gondii* oocysts were found to excyst efficiently when inoculated parenterally into mice (Table 1). This observation alleviated the need for oral inoculation for bioassay of oocysts (Dubey and Frenkel, 1973).

Ben Rachid (1970) fed *T. gondii* oocysts to gundis which died 6–7 days later from toxoplasmosis. This knowledge about the life cycle of *T. gondii* probably explains how gundis became infected in the laboratory of Nicolle. At least one cat was present in the Pasteur laboratory in Tunis (Dubey, 1977).

### 2.5. Discovery of life cycle

Although *T. gondii* has a wide host range it has retained the definitive-host specificity restricted to felids. Dr. J.K. Frenkel deserves the credit for initiating testing of many species of animals, including wild felids, for oocyst shedding under difficult housing conditions (Miller et al., 1972). It was an experience of a life-time for me, handling bobcats and ocelots in cages. I was a post doctoral fellow in Dr Frenkel’s laboratory and still the memory of one incident is vivid in my mind. My courageous neighbour who had a pet ocelot, loaned me the animal for testing for oocyst shedding. Three of us, Jack Frenkel, Nancy Miller (she was a biological technician) and myself were sweating trying to obtain a blood sample (without giving anaesthesia) from this 15-kg ocelot before feeding it tissue cysts. The ocelot shed oocysts and we returned the animal to the owner. Of the many species of animals experimentally infected with *T. gondii*, only felids shed *T. gondii* oocysts (Table 2).

Seroepidemiological studies on isolated islands in the Pacific (Wallace, 1969), Australia (Munday, 1972) and the USA (Dubey et al., 1997) have shown an absence of *T. gondii* on islands without cats, confirming the important role of the cat in the natural transmission of *T. gondii*. Vaccination of cats with a live mutant strain of *T. gondii* on eight pig farms in the USA reduced the transmission of *T. gondii* infection in mice and pigs (Mateus-Pinilla et al., 1999), thus supporting the role of the cat in natural transmission of *T. gondii*. Although *T. gondii* can be transmitted in several ways, it has adapted to be transmitted most efficiently by carnivorism in the cat and by the faecal-oral (oocysts) route in other hosts (Table 2). Pigs and mice (and presumably humans) can be infected by ingesting even one oocyst (Dubey et al., 1996), whereas a dose of 100 oocysts might not infect cats (Table 1). Additionally, the ingestion of only a few oocysts by certain hosts (e.g. mice, New World monkeys) can be lethal whereas oocysts are not pathogenic orally to cats, irrespective of the dose and the strain of *T. gondii* (Table 1).

<table>
<thead>
<tr>
<th>Definitive host</th>
<th>Oocyst shedding</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>African wild cat (<em>Felis lybica</em>)</td>
<td>Yes*</td>
<td>No</td>
</tr>
<tr>
<td>Amur leopard cat (<em>Felis bengalensis</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Asian leopard (<em>Felis bengalensis</em>)</td>
<td>Yes*</td>
<td>No</td>
</tr>
<tr>
<td>Bobcat (<em>Lynx rufus</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Cheetah (<em>Acinonyx jubatus</em>)</td>
<td>Yes*</td>
<td>No</td>
</tr>
<tr>
<td>Cougar (<em>Felis concolor</em>)</td>
<td>Yes*</td>
<td>No</td>
</tr>
<tr>
<td>Cougar (<em>Felis concolor vaconvenerensis</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Geoffroy's cat (<em>Oncifelis Geoffroyi</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Iriomote cat (<em>Felis iriomotensis</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Jaguarundi (<em>Felis yagouaroundi</em>)</td>
<td>Yes*</td>
<td>No</td>
</tr>
<tr>
<td>Lion (<em>Panthera leo</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Mountain lion (<em>Felis concolor</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Ocelot (<em>Felis pardalis</em>)</td>
<td>Yes*</td>
<td>No</td>
</tr>
<tr>
<td>Pallas cat (<em>Felis manul</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Pampas cat (<em>Oncifelis colocolo</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Siberian tiger (<em>Panthera tigris altaica</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
<tr>
<td>Wild cat (<em>Felis silvestris</em>)</td>
<td>No</td>
<td>Yes*</td>
</tr>
</tbody>
</table>

*Confirmed by bioassay in mice.  
*b Confirmed by bioassay in pigs.  
*c Immunohistochemical post-mortem examination.

Cats can shed millions of oocysts after ingesting only one bradyzoite, while ingestion of 100 bradyzoites may not infect mice orally (Table 2). This information has proved very useful in conducting epidemiological studies and for the detection by feeding to cats of low numbers of *T. gondii* in large samples of meat (Dubey et al., 2005). Thus, it has been possible to detect a few bradyzoites in a 500-kg piece of meat by feeding it to a cat and then examining cat faeces for oocyst shedding (Dubey et al., 1995) or to detect a few oocysts in contaminated animal feed or large quantities of water by bioassay in pigs (de Moura et al., 2006).

Cats shed oocysts after ingesting any of the three infectious stages of *T. gondii*, i.e. tachyzoites, bradyzoites and sporozoites (Frenkel et al., 1970). Pre-patent periods (time to the shedding of oocysts after initial infection) and frequency of oocyst shedding vary according to the stage of *T. gondii* ingested. Pre-patent periods are 3–10 days after ingesting tissue cysts, 18 days or longer after ingesting oocysts, irrespective of the dose (Dubey and Frenkel, 1972, 1976; Dubey, 1996, 2001). The pre-patent period after ingesting tachyzoites may vary (Dubey, 2005). Fewer than 50% of cats shed oocysts after ingesting tachyzoites or oocysts, whereas nearly all cats shed oocysts after ingesting tissue cysts (Dubey and Frenkel, 1972, 1976; Dubey, 1996, 2001, 2002, 2005, 2006).

**Table 1**

| Infectivity of *Toxoplasma gondii* oocysts to cats and mice.*
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of oocysts</td>
<td>Cats</td>
<td>Mice</td>
</tr>
<tr>
<td>-----------------</td>
<td>-------</td>
<td>-------</td>
</tr>
<tr>
<td>1,000,000</td>
<td>12/12b</td>
<td>5/5</td>
</tr>
<tr>
<td>100,000</td>
<td>1/1</td>
<td>5/5</td>
</tr>
<tr>
<td>10,000</td>
<td>3/4</td>
<td>5/5</td>
</tr>
<tr>
<td>1,000</td>
<td>3/4</td>
<td>5/5</td>
</tr>
<tr>
<td>100</td>
<td>1/4</td>
<td>5/5</td>
</tr>
<tr>
<td>10</td>
<td>0/4</td>
<td>4/5</td>
</tr>
<tr>
<td>1</td>
<td>Not done</td>
<td>2/5</td>
</tr>
<tr>
<td>&lt;1</td>
<td>Not done</td>
<td>1/5</td>
</tr>
</tbody>
</table>

*b No. of cats seroconverted to *T. gondii*/no. of cats fed oocysts. All cats remained asymptomatic.  
*c No. of mice infected of five mice inoculated. All infected mice died of toxoplasmosis.  

**Table 2**

Wild felids as definitive host for *Toxoplasma gondii*.
The discovery of the life cycle of *Toxoplasma gondii* in the cat proved very useful in understanding the basic biology of the parasite. For example, differences in the pre-patent periods of oocyst shedding after the ingestion of different stages provided conclusive evidence that bradyzoites can develop very early in infection (as early as 3 days p.i.), in the absence of any immune factors (in cell cultures without antibodies) and on occasion can transform directly into bradyzoites without conversion to tachyzoites (Dubey et al., 1998; Dubey, 2005). The transition of bradyzoite to tachyzoite and tachyzoite to bradyzoite is biologically important because clinical toxoplasmosis in AIDS patients occurs as a result of tissue cyst rupture and conversion of bradyzoites to tachyzoites. The transition of bradyzoite to tachyzoite and tachyzoite to bradyzoite is not an all-or-none phenomenon and the transitional stage has not been morphologically and biologically characterized. For example, tachyzoites lack periodic acid Schiff (PAS)-positive granules that are numerous in bradyzoites and their synthesis and accumulation is gradual (Dubey and Frenkel, 1976). The loss or synthesis of stage-specific molecule surface antigen 1 (SAG1) for tachyzoites or bradyzoite antigen 1 (BAG1) for bradyzoites is also gradual and both can be found simultaneously in the same infected host cell. The intermediate pre-patent periods (11–17 days) found in cats fed acutely infected mice suggested a transitional stage between tachyzoites and bradyzoites. Scientists working with *T. gondii* have traditionally obtained tachyzoites from ascites in mice inoculated i.p. with *T. gondii* stages, however pre-patent period data in cats suggests that all three stages (tachyzoites, bradyzoites and transitional stages) may be present simultaneously in the peritoneal exudate (Dubey, 2005).

From a parasitological viewpoint point only the bradyzoite-induced endogenous cycle is known in cats (Dubey and Frenkel, 1972). After the ingestion of tissue cysts by cats, the tissue cyst wall is dissolved by proteolytic enzymes in the stomach and small intestine. The released bradyzoites penetrate the epithelial cells of the small intestine and initiate the development of numerous generations of *T. gondii*. Five morphologically distinct types of *T. gondii* develop in intestinal epithelial cells before gametogony begins. These stages are designated types A to E instead of generations because there are several generations within each *T. gondii* type. These asexual stages in the feline intestine are structurally distinct from tachyzoites that also develop in the lamina propria. After asexual development (types A to E), the sexual cycle starts 2 days after tissue cysts are ingested by the cat. Gamonts are found throughout the small intestine but most commonly in the ileum, 3–15 days after inoculation. After fertilization, an oocyst wall is formed around the parasite. Infected epithelial cells rupture and discharge oocysts into the intestinal lumen (Table 3).

### 2.6. Oocyst-associated outbreaks

Oocysts shed into the environment have caused several outbreaks of disease in humans (Teutsch et al., 1979; Benenson et al., 1982; Bowie et al., 1997; de Moura et al., 2006). *Toxoplasma gondii* oocysts found in the faeces of naturally-infected cougars (Arambini et al., 1998) were epidemiologically linked to a Canadian waterborne outbreak of toxoplasmosis in humans (Bowie et al., 1997), but oocysts were not isolated from the water reservoir (Isaac-Renton et al., 1998). *Toxoplasma gondii* was isolated for the first time from drinking water stored in small water tanks on school roofs; these tanks were served by the reservoir epidemiologically linked to the Brazilian outbreak (de Moura et al., 2006).

The intensity of symptoms and generalized disease in these oocyst-associated outbreaks in humans (Table 4) suggests that oocysts are more pathogenic than bradyzoites or tachyzoites, irrespective of the dose. After being fed oocysts some hosts, particularly mice, can die of severe enteritis before distant organs are involved whereas many tissue cysts are needed to produce the same effect (Dubey and Frenkel, 1973; Dubey et al., 1997; Dubey, 1997).

After the discovery of the life cycle of *T. gondii* in the cat it became clear why Australasian marsupials and New World monkeys are highly susceptible to clinical toxoplasmosis. The former evolved apparently in the absence of the cat (there were few or no cats in Australia and New Zealand before settlement by Europeans) and the latter live in tree tops, not exposed to cat faeces. In contrast, marsupials in America and Old World monkeys are resistant to clinical toxoplasmosis (Dubey and Beattie, 1988).

One of the most intriguing recent findings related to oocyst biology is widespread *T. gondii* infection in marine mammals. Who would have thought that non-captive marine mammals would be dying of toxoplasmosis acquired from freshwater run-off from land contaminated with oocysts (Miller et al., 2002; Dubey et al., 2003; Conrad et al., 2005; Dubey and Jones, 2008)? Sea otters eat approximately 25% of their body weight in invertebrate prey each day (Conrad et al., 2005). Cold blooded animals, including fish, are not a host for *T. gondii* and there was no evidence that it multiplied in cold blooded animals (Dubey and Beattie, 1988). With respect to oocysts, it is not known if the sporozoite excysts after ingestion by cold blooded animals. Molluscs, however, can act as transport hosts for *T. gondii* oocysts (Arkush et al., 2003; Lindsay et al., 2004; Miller et al., 2008). In addition, sea otters might ingest oocysts directly from marine water but how much marine water is cycled through the gut of sea otters in a day is unknown, however it is likely to be large quantities.

### Table 3

<table>
<thead>
<tr>
<th>No of free bradyzoites</th>
<th>Cats</th>
<th>Mice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1000</td>
<td>8/13</td>
<td>61.5</td>
</tr>
<tr>
<td>100</td>
<td>11/16</td>
<td>68.7</td>
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<tr>
<td>10</td>
<td>5/18</td>
<td>27.7</td>
</tr>
<tr>
<td>1</td>
<td>3/18</td>
<td>16.6</td>
</tr>
<tr>
<td>S.C. %</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data pooled from two studies (Dubey, 2001 VEG strain and Dubey, 2006 TgC-kAr23 strain).

*No. of cats shed oocyst/no. of cats fed bradyzoites.
No. of mice infected with *T. gondii*/no. of mice inoculated bradyzoites.*

### Table 4

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Patients with symptoms (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Atlanta, USA (35 patients)</td>
</tr>
<tr>
<td>Fever</td>
<td>94</td>
</tr>
<tr>
<td>Lymphadenopathy</td>
<td>88</td>
</tr>
<tr>
<td>Headache</td>
<td>88</td>
</tr>
<tr>
<td>Myalgia</td>
<td>63</td>
</tr>
<tr>
<td>Stiff neck</td>
<td>57</td>
</tr>
<tr>
<td>Anorexia</td>
<td>57</td>
</tr>
<tr>
<td>Sore throat</td>
<td>46</td>
</tr>
<tr>
<td>Arthralgia</td>
<td>26</td>
</tr>
<tr>
<td>Rash</td>
<td>23</td>
</tr>
<tr>
<td>Confusion</td>
<td>20</td>
</tr>
<tr>
<td>Earache</td>
<td>17</td>
</tr>
<tr>
<td>Nausea</td>
<td>17</td>
</tr>
<tr>
<td>Eye pain</td>
<td>14</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>11</td>
</tr>
</tbody>
</table>

*From Teutsch et al. (1979).
From Benenson et al. (1982).
Not reported.
De Moura et al. (2006).*

Symptoms Patients with symptoms (%)
---|--|--|--
Fever | 94 | 90 | 82
Lymphadenopathy | 88 | 77 | 75
Headache | 88 | 77 | 87
Myalgia | 63 | 68 | 80
Stiff neck | 57 | 55 | NR
Anorexia | 57 | NR | 69
Sore throat | 46 | NR | NR
Arthralgia | 26 | 29 | 61
Rash | 23 | 0 | 7
Confusion | 20 | NR | NR
Earache | 17 | NR | NR
Nausea | 17 | 36 | 38
Eye pain | 14 | 26 | NR
Abdominal pain | 11 | 55 | NR

Frequency of symptoms in people with post-natally acquired toxoplasmosis.
3. Conclusion

One of the most obvious impacts of the discovery of the life cycle of *Toxoplasma gondii* is on animal management. It is now possible to raise pigs and cats *Toxoplasma gondii*-free by keeping the cat out of the pig houses. The prevalence of *T. gondii* in market age pigs has been reduced drastically during the last decade and in the USA this low seroprevalence in pigs has been reflected in a similar decline in humans. Although some of the mystery about the transmission of *T. gondii* has been solved, we must not forget that it is a very 'clever' parasite and prevention of oocyst shedding by the “alley cat” is not going to be easy. Also, reverting back to organic farming will be associated with costs to human health with respect to toxoplasmosis.

References


