REVIEW ARTICLE
A review of toxoplasmosis in humans and animals in Ethiopia

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SUMMARY
Toxoplasmosis caused by the protozoan parasite, Toxoplasma gondii, is a worldwide zoonosis. In this paper published information on toxoplasmosis in humans and other animals in Ethiopia is reviewed. Limited data indicate that the prevalence of T. gondii in humans in Ethiopia is very high, up to 41% of children aged 1–5 years were reported to be seropositive. There is little information on seroprevalence data in pregnant women and no data on congenital toxoplasmosis in children. About 1 million adults in Ethiopia are considered to be infected with HIV with less than one-third likely receive highly active antiviral therapy. Based on a conservative T. gondii seroprevalence of 50%, thousands might die of concurrent opportunistic infections, including toxoplasmosis. However, exact figures are not available, and most serological surveys are not current. Serological surveys indicate up to 79% of goats and sheep have T. gondii antibodies. However, there is no information on losses due to toxoplasmosis in livestock or the presence of viable T. gondii in any host in Ethiopia.

Key words: Epidemiology, Ethiopia, humans, Toxoplasma gondii.

INTRODUCTION
Toxoplasmosis, caused by the protozoan Toxoplasma gondii, is a worldwide zoonosis [1]. In general its seroprevalence is very high in South America and low in Asia. Fragmentary reports indicate a high prevalence of T. gondii infections in Africa [1]. Toxoplasmosis is usually asymptomatic in immunocompetent adults, but can cause mortality in the very young and the immunocompromised. Many patients infected with human immunodeficiency virus (HIV) infection die of toxoplasmosis. This is of particular concern in many African countries because of the high prevalence of HIV and lack of resources to manage it. We summarize the current status of T. gondii infection in humans and other animals in Ethiopia.

Humans and other animals become infected with T. gondii mostly by ingesting undercooked meat of infected animals or by ingesting food or water contaminated with oocysts [1]. Cats are essential in the life-cycle of T. gondii because they are the only hosts that can excrete the environmentally resistant oocysts in nature. The prevalence of T. gondii antibodies varies with age, lifestyle of the cat (stray vs. pet), the serological test utilized, the screening dilution, and

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other undefined factors. In general, infection in cats increases with age and the prevalence is higher in stray cats. Infected cats can shed millions of oocysts in a matter of a few days, and after sporulation oocysts can survive in the environment for months or even years depending on the moisture and ambient temperature. Cats are thought to become infected by ingesting infected prey soon after they begin to hunt. Cats usually shed oocysts only for a short time and once in their life. However, poor nutrition, concurrent infections, and immunosuppression may affect the immune status of the cat and lead to increased oocyst shedding.

TOXOPLASMOSIS IN HUMANS IN ETHIOPIA

Limited data indicate a high seroprevalence of T. gondii antibodies in humans in Ethiopia (Table 1). Among these reports, the study by Guebre-Xabier et al. [7] is of note. They tested 1016 sera from different age groups from six geographical locations in Ethiopia. Seroprevalence varied from 47–96% with high rates in 97 children (aged 14–18 years) from leprosy families (85-5%) and from 427 blood donors (50–92%). This high prevalence in blood donors is important because toxoplasmosis can be transmitted by blood transfusion, especially in immunosuppressed persons or during acute infection. In an earlier report, unfortunately only published as an abstract, T. gondii antibodies were found in 42% of 614 persons sampled in 1:50 serum dilution tested with an in-house ELISA [4]. These authors reported that the prevalence was 41% in children aged 1–5 years but the number of children tested is not given [4].

Little is known of clinical toxoplasmosis in people or animals in Ethiopia. Tsega & Belehu [3] found T. gondii antibodies in five (82%) of 61 patients with lymphadenopathy but not in 39 control patients; however, these data are insufficient to imply that T. gondii was a cause of lymphadenopathy in the five patients. Shibre et al. [17] found no relationship between T. gondii infection and effectiveness of schizophrenia therapy in Addis Ababa based on treatment with the anti-T. gondii drug trimethoprim.

Much medical attention is being focused on the acquired immune deficiency syndrome (AIDS) epidemic in Africa. Ethiopia is the second-most populous nation in the horn of Africa, with over...
82 million inhabitants, and a high rate of AIDS. The finding of 93.3% seroprevalence of *T. gondii* antibodies in HIV patients by Shimelis et al. [12] is notable. Although clinical toxoplasmosis has been suspected in many HIV-infected patients treated with highly active antiviral therapy (HAART), and immune reconstitution [18–20], there are no histologically verified cases of toxoplasmosis in HIV-infected or immunocompetent persons in Ethiopia because histological diagnosis has not been pursued. Of 566 HIV-related deaths in a teaching hospital in Addis Ababa, a central mass lesion (with suspicion of toxoplasmosis) was diagnosed in brains of 74 (13.1%) by Bane et al. [21], but the diagnosis was not pursued further. The strongest positive evidence of clinical toxoplasmosis in AIDS is that reported by Amogne et al. [22]. They diagnosed neurological toxoplasmosis in 323 AIDS patients solely on the basis of clinical signs and favourable response to treatment with the anti-*T. gondii* medicine sulfadoxine-pyrimethamine (Fansidar; Hoffmann-La Roche, Switzerland). Serology for HIV was positive in all patients, and *T. gondii* IgG antibodies were found in 19 (83%) of 23 cases; no *T. gondii* serological data were given for the remaining 300 patients. The symptoms reported in these 323 patients were headache (91%), fever (83%), altered sensorium (62%), and seizures (48%). Radiographically, 79% of patients had enhancing lesions. In total, 248 (77%) patients responded to treatment with clinical improvement, 64 (20%) died in hospital, and 11 (3%) did not show clinical improvement. There is no mention of post-mortem examination. In summary, all the evidence presented is presumptive, and there are no definitive data with respect to clinical toxoplasmosis in humans in Ethiopia.

### TOXOPLASMOSIS IN OTHER ANIMALS IN ETHIOPIA

There are no reports of clinical toxoplasmosis in other animals. Serological surveys indicate a high prevalence of *T. gondii* antibodies in sheep and goats, although these surveys are more than a decade old (Table 2). Recently, Teshale et al. [16] reported 74.9% seroprevalence in 641 goats from central and southern regions of Ethiopia. Seroprevalence in cattle was low [13]. To our knowledge, there is no report of isolation of viable *T. gondii* from animals (or humans) in Ethiopia.

### PROSPECTIVE

Limited data in Table 1 indicate that the prevalence of *T. gondii* in humans in Ethiopia is very high. As noted above, a study conducted in 1981–1982, mentioned a seroprevalence of 41% in children aged 1–5 years, which is quite high for this age group, but the number of children tested out of a total of 614 people tested was not given [4]. About 1 million adults in Ethiopia are considered to be infected with HIV, with less than one-third of them likely receive HAART [23]. Based on a conservative *T. gondii* seroprevalence of 50%, thousands might die of concurrent opportunistic infections, including toxoplasmosis. However, exact figures are not available, and most serological surveys are not current. To start, a planned survey is needed for *T. gondii* prevalence in different age groups, especially pregnant women. Attempts should be made to isolate viable *T. gondii* from food animals, cats, and humans because nothing is known of the genetic diversity of *T. gondii* strains prevalent in humans and other animals in Ethiopia.

### Table 2. Summary of Toxoplasma gondii prevalence in animals in Ethiopia

<table>
<thead>
<tr>
<th>Subject</th>
<th>Year sampled</th>
<th>Population surveyed</th>
<th>No.</th>
<th>Serological test, cut-off titre*</th>
<th>% prevalence</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Not given</td>
<td>Not given</td>
<td>133</td>
<td>IHAT-2</td>
<td>19-5</td>
<td>Deconinck et al. [14]</td>
</tr>
<tr>
<td></td>
<td>Not given</td>
<td>Nazareth</td>
<td>58</td>
<td>MAT, 32</td>
<td>24-1</td>
<td>Nagash et al. [15]</td>
</tr>
<tr>
<td></td>
<td>Not given</td>
<td>Not given</td>
<td>94</td>
<td>IHAT-2</td>
<td>25-6</td>
<td>Deconinck et al. [14]</td>
</tr>
</tbody>
</table>

* IHAT-1, Indirect hemagglutination test (Wellcome Diagnostics, UK); IHAT-2, indirect haemagglutination test (Toxoplasmose Fumouze, France); ELISA, enzyme-linked immunosorbent assay (Enzygnost, bioMérieux, France); MAT, modified agglutination test.
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DECLARATION OF INTEREST
None.

REFERENCES