In an attempt to isolate and characterize Toxoplasma gondii from the State of Minas Gerais, Brazil, musculature samples from 72 pigs, 25 dogs, 28 free-range chickens and 50 chickens produced in industrialized farms were collected. Antibodies to T. gondii have not been detected in pigs, but were found in nine (40.9 %) of 22 dogs, and in 15 (53.6 %) of 28 free range chickens. T. gondii was not isolated from pigs and industrialized chickens, but from eight dogs and 11 free range chickens. In order to determine T. gondii virulence, female BALB/c mice were inoculated with $10^3$, $10^4$, $10^5$ and $10^6$ tachyzoites of the 19 isolates. The strains RH (virulent) and ME49 (non-virulent) were used as references. Isolates were divided into three groups according to the virulence phenotype: five isolates were classified into virulent in mice, one into non-virulent and 13 into intermediate virulent. Nested-PCR of T. gondii SAG2 locus amplified DNA from 21 out of 22 DNA samples directly extracted from heart of free range chickens. These samples were genotyped through a PCR-RFLP assay. Seventeen (80.9 %) were classified into type I; one (4.8 %) into type III and three (14.3 %) into type I or II.

**KEY WORDS:** Toxoplasma gondii, strains, domestic animals, virulence, PCR-RFLP, Brazil.

**INTRODUCTION**

Toxoplasmosis is a worldwide infection distributed among homoeothermic animals with a high prevalence in humans (15-85 % seropositivity) in different populations. Humans acquire toxoplasmosis through ingestion of tissue cysts in raw or undercooked meat or even through water and raw vegetables contaminated with oocysts from cat feces (Dubey & Beattie, 1988). Although the infection is usually benign, HIV immunodeficient patients and fetuses may develop severe neurological symptoms (Gellin & Soave, 1992). Serological studies have evidenced a high infection rate in animals for human consumption, mainly pork, lamb and goat (Tenter et al., 2000). According to these authors, pork is the main source of human T. gondii infection in the US. Literák & Hejilček (1993) remarked that free range chickens may represent a risky source of contamination when manipulation of raw meat is performed with no hygiene measures. Moreover, such chickens keep direct ground contact, becoming a major instrument for determining the level of environmental contamination by T. gondii oocysts (Devada et al., 1998). Dogs play a secondary role in T. gondii transmission in Brazil, once they are not a food source for man. On the other hand, Lindsay et al. (1997) have reported high risk rates of human contamination through direct contact with dogs. Molecular analysis of T. gondii strains worldwide has shown that most strains fit into one of the three genotypes, designated as type I, II and III (Howe & Sibley, 1995). Howe et al. (1997) developed a typing system...
for *T. gondii* strains based on analysis of restriction fragments of the gene SAG2, amplified by nested-PCR, which allows a differentiation among strain genotypes of the three clonal lineages, previously described by Howe & Sibley (1995). In Brazil, this method was used for analyzing *T. gondii* isolates from São Paulo (Dubey et al., 2002), Rio de Janeiro (Dubey et al., 2003a) and Paraná (Dubey et al., 2003b), which showed to be predominantly type I. Ferreira et al. (2006), using a multiplex PCR-RFLP, demonstrated that Brazilian strains present recombinant genotypes with typical alleles of the strains types I, II, and III in the majority of the loci. The RAPD-PCR and SSR-PCR analysis of these same strains placed them into two different clusters correlated with virulence for BALB/c mice (Ferreira et al., 2004). The aim of the current study was to obtain *T. gondii* isolates directly from tissues of pigs, dogs and chickens in the State of Minas Gerais, Brazil, and to determine their virulence in BALB/c mice. We have also attempted to determine *T. gondii* genotype of the SAG2 locus in chicken through PCR-RFLP, using readily obtained DNA from heart samples.

**MATERIALS AND METHODS**

**PIGS**

Muscle tissue from 72 pigs was bioassayed in outbred Swiss Webster mice. Out of these, a number of 37 heart samples were collected between September and October 1999 in a slaughterhouse in Santa Luzia, State of Minas Gerais (MG). The 35 other samples comprised 21 whole tongues and 14 whole hearts, obtained from slaughterhouse of Ribeirão das Neves, MG, in 2000. It was not possible to assure whether the pieces, heart and tongue came from the same animal. Swine sera could not be collected either.

**DOGS**

Diaphragms from 25 dogs with leishmaniasis were bioassayed. The samples were obtained from February to June 2000 and the animals were collected by the Center of Zoonosis Control in the city of Belo Horizonte, MG, and killed at the Universidade Federal de Minas Gerais (UFMG).

**CHICKENS**

Heart samples from 28 free range chickens in residences from the Minas Gerais State were collected in 2000 (six samples) and 2003 (22 samples) and bioassayed; 50 other heart samples from industrialized chicken farms acquired in a slaughterhouse in Belo Horizonte, MG, in 2001, were also evaluated. Muscle and serum from each animal were transported to the Department of Parasitology, UFMG.

**BIOASSAY IN MICE**

Muscle samples from pigs, dogs and chickens were digested in accordance with Dubey et al. (1995), with no previous knowledge on the animal serological status. Samples of approximately 10 g (from pigs and dogs) and half of each chicken heart were ground and submitted to digestion with pepsin at 37°C for 90 min. To each sample, phosphate buffer saline (PBS) pH 7.2 with penicillin (1,000 units) and streptomycin (100 µg/mL) were added. A volume of 0.5 ml from each sample was inoculated into non-infected Swiss mice, from the Animal House of UFMG, via intraperitoneal (i.p.). Dead mice or those with infection characteristics were assessed for tachyzoite or tissue cyst search. Surviving mice were killed at day 30 post-inoculation (p.i.), and a serum dilution of 1:16 to 1:64 from each mouse was tested for *T. gondii* antibodies by IFAT (Camargo, 1964). Brains were also analyzed in order to verify the presence of tissue cysts (Dubey & Beattie, 1988).

**SEROLOGY**

Sera from dogs and chickens were diluted fourfold from an initial dilution of 1:16 and assayed for *T. gondii* antibodies by IFAT. Tissue liquid, obtained according to Wingstrand et al., (1997), from pig muscle samples at 1:40 dilution was assayed for *T. gondii* antibodies search through ELISA.

**DETERMINATION OF *T. gondii* ISOLATES VIRULENCE**

*T. gondii* virulence in mice was determined in accordance with Ferreira et al. (2001). Successive dilutions from 10^3 to 10^9 tachyzoites were prepared in sterile PBS pH 7.2. 20 female BALB/c mice were inoculated with each *T. gondii* isolate. Mice were obtained from the Animal House of UFMG, aged between four and six weeks, and divided into four groups with five animals each. Each group was inoculated with different concentrations of tachyzoites: 10^3, 10^4, 10^5 and 10^6 tachyzoites/animal i.p., in a volume of 0.2 ml. Five control animals were inoculated with 0.2 ml of sterile PBS pH 7.2. The virulent strain RH and non-virulent ME49 were used as reference strains (Ferreira et al., 2001). Mice mortality was daily observed throughout 30 days, when the survivors were tested for *T. gondii* antibodies (IFAT) and brain cysts.

**GENETIC ANALYSIS OF CHICKEN ISOLATES**

DNA extraction was performed according to Aspinall et al. (2002) in each heart sample of the 22 chickens collected in 2005. DNA extraction was not possible to be performed in heart samples obtained in 2000. The
presence of *T. gondii* was identified by means of the 
SAG2 locus analysis through nested-PCR, as described
by Howe et al. (1997). Products of SAG2 amplification
by nested-PCR were digested by the restriction enzymes
Sau3AI and *H*baI (Promega). Digested products were
visualized in 5 % silver stained polyacrylamide gel
(Santos et al., 1993). As a reaction control, the strains
RH (type I), ME49 (type II) and VEG (type III) (Fux et
al., 2003) were used.

**RESULTS**

*T. gondii* was not isolated from pigs. Antibodies
against *T. gondii* were not found in their tissue
liquids. Out of the 25 muscle samples from dogs,
eight (32 %) isolates were classified into D1 to D8
(Table I). *T. gondii* was isolated from three dogs, from
which sera was not possible to be obtained. Anti-*T.
gondii* antibodies were found in nine of the 22 (40.9 %)
dogs analyzed. *T. gondii* was isolated in five muscle
samples from dogs with positive serology, but not in
four animals with positive serology or in animals with
negative serology (Table I).

Of the 28 muscle samples from free range chickens,
11 (39.3 %) *T. gondii* isolates were classified into CH1
to CH11 (Table II). Anti-*T. gondii* antibodies were found
in 15 (53.6 %) chickens. The parasite was isolated in
nine chicken hearts with *T. gondii* antibodies and two
other chickens without specific antibodies (chickens 3
and 4). *T. gondii* was not isolated in six chickens with
positive serology or in samples obtained from indus-
trialized chicken farms in Belo Horizonte.

Results on virulence in infected BALB/c mice inocu-
lated with different concentrations of tachyzoites from
*T. gondii* isolates are showed in Table III. Virulence
experiments were done in duplicate and were repro-
ducible. The isolates D5, D6, CH4, CH5 and CH10 were
classified into virulent strains in mice, once all the infec-
ted animals died regardless of the tachyzoite concen-
tration. Mice death occurred between six and 23 days
after infection. The isolate D8 was classified into non-
virulent, as all mice survived 30 days of experiment.
All surviving mice showed to have anti-*T. gondii* anti-
bodies (IFAT ≥ 1:16). Except for two animals inocu-
lated with a concentration of 10⁸ tachyzoites, brain
cysts were found in two other animals inoculated with
10⁷ tachyzoites and in five other mice inoculated at a
concentration of 10⁶ tachyzoites. Other isolates (D1,
D2, D3, D4, D7, CH1, CH2, CH3, CH6, CH7, CH8, CH9
and CH11) showed to have intermediate virulence
traits among the phenotype of virulent strains, such as
the strain RH (100 % mortality), and non-virulent strains
as ME49 (100 % survival).

Out of the 22 DNA samples directly extracted from
chicken hearts, a number of 21 showed amplification
products of 241 bp for the region 5’ of the *T. gondii*
SAG2 locus and 19 showed an expected amplification
product of 221 bp for the region 3’ at the same locus.
The isolation was positive in eight (38.1 %) of the 21
samples, in which *T. gondii* was identified through
nested-PCR. For the three samples with no amplifica-
tion of the region 3’, nested-PCR was repeated at dif-
ferent DNA dilutions, that is, 1:2, 1:10, 1:20 e 1:40, but
there was no DNA amplification, precluding RFLP char-
acterization. Of the 21 *T. gondii*-positive samples, 17
(80.9 %) were type I and one (4.8 %) type III. Three
samples (14.3 %), with no region 3’ amplification, were
digested only with the restriction enzyme Sau3AI,
being characterized as type I or II (Fig. 1). The virulent
isolates CH4, CH5 and CH10 were type I. Out of the

<table>
<thead>
<tr>
<th>Chicken n°</th>
<th>IFAT titer</th>
<th>Isolation/Isolate name</th>
<th>Month/Year of isolation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1:64</td>
<td>Yes/CH1</td>
<td>11/2000</td>
</tr>
<tr>
<td>2</td>
<td>1:256</td>
<td>Yes/CH2</td>
<td>11/2000</td>
</tr>
<tr>
<td>3</td>
<td>&lt; 1:16</td>
<td>Yes/CH3</td>
<td>12/2000</td>
</tr>
<tr>
<td>4</td>
<td>&lt; 1:16</td>
<td>Yes/CH4</td>
<td>04/2003</td>
</tr>
<tr>
<td>5</td>
<td>1:1024</td>
<td>Yes/CH5</td>
<td>04/2005</td>
</tr>
<tr>
<td>6</td>
<td>1:64</td>
<td>Yes/CH6</td>
<td>04/2003</td>
</tr>
<tr>
<td>7</td>
<td>1:16</td>
<td>Yes/CH7</td>
<td>05/2003</td>
</tr>
<tr>
<td>8</td>
<td>1:256</td>
<td>Yes/CH8</td>
<td>05/2003</td>
</tr>
<tr>
<td>9</td>
<td>1:1024</td>
<td>Yes/CH9</td>
<td>05/2003</td>
</tr>
<tr>
<td>10</td>
<td>1:1024</td>
<td>Yes/CH10</td>
<td>05/2003</td>
</tr>
<tr>
<td>11</td>
<td>1:1024</td>
<td>Yes/CH11</td>
<td>05/2003</td>
</tr>
<tr>
<td>12</td>
<td>1:16</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>13</td>
<td>1:64</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>14-16</td>
<td>1:256</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>17</td>
<td>1:1024</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>18-28</td>
<td>&lt; 1:16</td>
<td>No</td>
<td>–</td>
</tr>
</tbody>
</table>

Yes: positive isolation; No: negative isolation.

Table II. – Indirect fluorescent antibody test (IFAT) for toxoplasmosis
in free range chickens sera from Minas Gerais, Brazil, and *T. gondii*
isolates obtained from muscle samples.

<table>
<thead>
<tr>
<th>Dog n°</th>
<th>IFAT titer</th>
<th>Isolation/Isolate name</th>
<th>Month/Year of isolation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ND</td>
<td>Yes/D1</td>
<td>02/2000</td>
</tr>
<tr>
<td>2</td>
<td>ND</td>
<td>Yes/D2</td>
<td>04/2000</td>
</tr>
<tr>
<td>3</td>
<td>ND</td>
<td>Yes/D3</td>
<td>04/2000</td>
</tr>
<tr>
<td>4</td>
<td>1:1024</td>
<td>Yes/D4</td>
<td>06/2000</td>
</tr>
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<td>5</td>
<td>1:64</td>
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<td>07/2000</td>
</tr>
<tr>
<td>6</td>
<td>1:1024</td>
<td>Yes/D6</td>
<td>07/2000</td>
</tr>
<tr>
<td>7</td>
<td>1:256</td>
<td>Yes/D7</td>
<td>07/2000</td>
</tr>
<tr>
<td>8</td>
<td>1:64</td>
<td>Yes/D8</td>
<td>08/2000</td>
</tr>
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<td>9-10</td>
<td>1:16</td>
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<td>–</td>
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<tr>
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<td>1:64</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>12</td>
<td>1:256</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>13-25</td>
<td>&lt; 1:16</td>
<td>No</td>
<td>–</td>
</tr>
</tbody>
</table>

ND: not done; Yes: positive isolation; No: negative isolation.

Table I. – Indirect fluorescent antibody test (IFAT) for toxoplasmosis
in dog sera from Minas Gerais, Brazil, and *T. gondii* isolates obtained
from muscle samples.
BRANDÃO G.P., FERREIRA A.M., MELO M.N. & VITOR R.W.A.

**Table III.** – Virulence comparison for BALB/c mice inoculated by intraperitoneal injection with tachyzoites of different *T. gondii* isolates from the State of Minas Gerais, Brazil.

<table>
<thead>
<tr>
<th>Isolates</th>
<th>SAG2</th>
<th>10⁰</th>
<th>10¹</th>
<th>10²</th>
<th>10³</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>D/S</td>
<td>D/S</td>
<td>D/S</td>
<td>D/S</td>
</tr>
<tr>
<td>D1</td>
<td>I*</td>
<td>0/4**</td>
<td>1/24</td>
<td>5/0</td>
<td>16.0</td>
</tr>
<tr>
<td>D2</td>
<td>I*</td>
<td>2/14</td>
<td>16.0</td>
<td>5/0</td>
<td>9.8</td>
</tr>
<tr>
<td>D3</td>
<td>I*</td>
<td>0/5</td>
<td>-</td>
<td>0/5</td>
<td>-</td>
</tr>
<tr>
<td>D4</td>
<td>I*</td>
<td>4/1</td>
<td>15.5</td>
<td>0/5</td>
<td>10.5</td>
</tr>
<tr>
<td>D5</td>
<td>I*</td>
<td>1/0</td>
<td>12.6</td>
<td>5/0</td>
<td>8.8</td>
</tr>
<tr>
<td>D6</td>
<td>I*</td>
<td>5/0</td>
<td>10.4</td>
<td>5/0</td>
<td>8.6</td>
</tr>
<tr>
<td>D7</td>
<td>III*</td>
<td>0/4**</td>
<td>4/1</td>
<td>1/4</td>
<td>21.0</td>
</tr>
<tr>
<td>D8</td>
<td>III*</td>
<td>0/5</td>
<td>-</td>
<td>0/5</td>
<td>-</td>
</tr>
<tr>
<td>CH1</td>
<td>III*</td>
<td>0/5</td>
<td>-</td>
<td>1/4</td>
<td>14.0</td>
</tr>
<tr>
<td>CH2</td>
<td>III*</td>
<td>1/0</td>
<td>-</td>
<td>1/4</td>
<td>14.0</td>
</tr>
<tr>
<td>CH3</td>
<td>III*</td>
<td>0/5</td>
<td>-</td>
<td>2/3</td>
<td>18.5</td>
</tr>
<tr>
<td>CH4</td>
<td>I</td>
<td>1/0**</td>
<td>18.0</td>
<td>5/0</td>
<td>18.5</td>
</tr>
<tr>
<td>CH5</td>
<td>I</td>
<td>5/0</td>
<td>19.8</td>
<td>5/0</td>
<td>16.2</td>
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<tr>
<td>CH6</td>
<td>III</td>
<td>0/4**</td>
<td>-</td>
<td>1/3**</td>
<td>17.0</td>
</tr>
<tr>
<td>CH7</td>
<td>I</td>
<td>4/1</td>
<td>24.8</td>
<td>3/2</td>
<td>25.7</td>
</tr>
<tr>
<td>CH8</td>
<td>I</td>
<td>2/2**</td>
<td>13.0</td>
<td>3/1**</td>
<td>13.0</td>
</tr>
<tr>
<td>CH9</td>
<td>I</td>
<td>0/4**</td>
<td>-</td>
<td>3/2</td>
<td>20.0</td>
</tr>
<tr>
<td>CH10</td>
<td>I</td>
<td>5/0</td>
<td>12.2</td>
<td>5/0</td>
<td>10.6</td>
</tr>
<tr>
<td>CH11</td>
<td>I or II</td>
<td>0/5</td>
<td>-</td>
<td>2/3</td>
<td>20.5</td>
</tr>
</tbody>
</table>

*SAG2:* analysis on the SAG2 locus of *T. gondii*; *:* PCR-RFLP analysis according to Ferreira et al. (2006); D/S: dead/surviving mice with positive IFAT; **:* some mice were negative by IFAT; *d:* mean day of death; *a:* three mice inoculated; *b:* four mice inoculated.

Fig. 1. – RFLP of *T. gondii SAG2* locus. (A) region 5' with the restriction endonuclease *Sau3AI* (able to distinguish type III from types I and II); and (B) region 3' using the restriction endonuclease *HhaI* (able to distinguish type II from types I and III). Products were resolved in 5 % silver stained polyacrylamide gels for genotyping chicken tissues using the reference strains RH (type I), ME49 (type II) and VEG (type III). M: molecular size marker (Promega 100 pb); C: negative control (no DNA). The arrow indicates restriction polymorphisms.
five isolates of intermediate virulence, one was type III (CH6), three were type I (CH7, CH8 and CH9) and one was type I or II (CH11).

DISCUSSION

*T. gondii* was not isolated from pig tissues. Serology was carried out using tissue liquids showing to be negative for all animals under study. Such data are not in accordance with the hypothesis that pigs, in Europe and the USA, are the major source of *T. gondii* human infection (Tenter *et al.*, 2000). Recently, investigations have shown that due to the improvements in breeding conditions, including hygiene measures, close confinement and disease prevention, prevalence of *T. gondii* infections in pigs have significantly decreased in the last decade worldwide, including Brazil (Tenter *et al.*, 2000). Those measures may explain the fact that there were no pig isolates in the current work.

*T. gondii* was isolated from eight out of 25 dogs and 40.9 % showed *T. gondii* antibodies (IFAT ≥ 1:16). Mineo *et al.* (2001) reported a prevalence of 36 % of *T. gondii*-seropositive dogs, out of 163 dogs in Uberlândia (MG). In Brazil, dog is not a human food source, but the infection rates may indicate that the parasite and man have been circulating in the same environment (Geramino *et al.*, 1985). Moreover, it has been suggested that dogs may act in *T. gondii* mechanical transmission by transporting sporulated oocysts in their fur (Lindsay *et al.*, 1997).

*T. gondii* was isolated from 11 out of 28 free range chickens and 53.6 % showed *T. gondii* antibodies (IFAT ≥ 1:16). Dubey *et al.* (2002), investigating *T. gondii* seroprevalence in free range chickens in São Paulo, Brazil, through Modified Agglutination Test (MAT), reported a rate of 39.02 % and Silva *et al.* (2003), in a similar investigation, reported a *T. gondii* seroprevalence of 64 % for the State of Rio de Janeiro. According to Dubey & Beattie (1988), chickens do not produce *T. gondii* antibodies at levels that can be detected by routine tests, which might explain the fact that, in the present work, *T. gondii* was isolated from two serologically negative chickens. Similarly, Silva *et al.* (2003) obtained *T. gondii* isolates from chicken tissues with negative serology through MAT. High prevalence of *T. gondii* in free range chickens may be explained by their habit to move around freely in direct contact with oocyst-contaminated ground, easily accessed by free-living cats (Literák & Hejlícek, 1993). Our results have shown that, differently from free range chickens, samples from industrialized chicken farms revealed that these animals do not pose a risk to man regarding *T. gondii* infection. Such data are in accordance with those described by Literák & Hejlícek (1993). The absence of toxoplasmosis infection in animals from industrialized chicken farms is possibly due to the fact that they are close confined under hygiene measures and prevention of diseases, including coccidiosis.

In the current investigation, *T. gondii* isolates were divided into three groups according to the virulence phenotype in BALB/c mice: virulent; non-virulent; and intermediate virulent. The isolates D5, D6, CH4, CH5 and CH10 were classified into virulent, as they led to death of 100 % of mice, regardless of the inoculated concentration, and also produced brain cysts. As a general rule, animals inoculated with such isolates showed a greater survival length as compared to those inoculated with the strain RH. The isolate D8 was classified into non-virulent, similar to the strain ME49, as it caused no death to mice. The great variation observed among the other isolates (D1, D2, D3, D4, D7, CH1, CH2, CH3, CH6, CH7, CH8, CH9 and CH11) has not allowed us to classify them into virulent and non-virulent, being, thus, classified into intermediate virulent. Intermediate virulent strains are believed to be transition strains from the virulent to the non-virulent phenotype (Literák *et al.*, 1998). Guo *et al.* (1997) reported that intermediate virulent strains occasionally occur in nature. However, in the present work, most isolates obtained were classified into intermediate virulent in BALB/c mice.

Out of the 22 DNA samples from chicken heart specimens, a number of 21 specimens had the region 5’ of the *SAG2* locus amplified and 19, the region 3’. By genotyping 68 samples using the *SAG2* locus, Fuentes *et al.* (2001) have also remarked difficulties in amplifying regions 3’ or 5’ for some samples. It is interesting to note that DNA samples extracted from chicken hearts, from which the parasite was isolated, showed to have a greater amount of *T. gondii* DNA, once more intensively stained bands were detected when compared to those samples with no isolates. This little DNA amount has led us to believe that there were few parasites in the tissues; hence, less chance had the mice to be infected by the inoculated material. In accordance with Fuentes *et al.* (2001), nested-PCR using DNA directly extracted from tissue samples has shown to be an accurate and fast method to detect *T. gondii*, which considerably precludes the use of time-consuming techniques, requiring parasite culture. From the results obtained in the present investigation, we may state that the nested-PCR technique is more sensitive to detect the presence of *T. gondii* in tissue samples than the method of inoculation in mice.

Through RFLP of the *T. gondii SAG2* locus using DNA directly extracted from chicken heart, 17 samples was identified as type I, one as type III; and three samples as type I or II. Three virulent strains (CH4, CH5 and CH10) in mice were type I and one (CH11) was type I or II. Our results are in agreement with those by
Dubey et al. (2002, 2003a), who observed a predominance of T. gondii isolates as being type I in chickens obtained in the States of São Paulo and Rio de Janeiro. On the other hand, other reports have revealed that T. gondii isolates from chickens obtained in Argentina (Dubey et al., 2003c), Egypt (Dubey et al., 2003d) and the US (Dubey et al., 2003e) were mostly type III, suggesting a different epidemiological prevalence of T. gondii genotypes in Brazil, as compared to other countries. Further studies on samples isolated in other Brazilian states are necessary in order to either support or reject such hypothesis.

ACKNOWLEDGEMENTS

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REFERENCES


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