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Toxoplasma gondii Seroprevalence and Risk Factors in Feline Patients Admitted to a Small Animal Veterinary Teaching Hospital in Central Germany over 11 Years (2012–2023)

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Abstract: *Toxoplasma gondii* is a protozoan parasite in cats that can be a cause of zoonotic concern due to sheding infectious and highly resistant oocysts. Clinical toxoplasmosis is rare in cats and can occur with mild to severe clinical signs or even fatal outcomes. *T. gondii* immunoglobulin M (IgM) or IgG is an indicator of ongoing or prior infection. Information about *T. gondii* occurrence in the domestic cat population, primary clinical concerns about affected cats, and potential risk factors is scarce in Germany. This retrospective study explored data from 174 feline patients (≥12 weeks old) presented to a tertiary veterinary care facility in central Germany between 2012 and 2023. *T. gondii* serological status, determined as part of the cats' routine diagnostic evaluation, was assessed in relation to patient characteristics, environmental factors, medical history data, and clinical diagnosis. Neurological (57%), locomotor (25%), and ocular signs (11%) were common, and older age was associated with the presence of IgG ($p = 0.008$). Sex had no significant effects on *T. gondii* antibody positivity ($p = 0.865$). More outdoor cats than indoor-only cats were *T. gondii* IgG-positive ($p = 0.021$), and IgG-seropositive animals received a canned/wet diet more often than a dry kibble diet ($p < 0.0001$). Clinical representations aligned with previous studies, and age, choice of food, and outdoor living ($p = 0.021$) were significant predisposing factors.

Keywords: toxoplasmosis; serology; zoonosis; cat; feline health; veterinary parasitology

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1. Introduction

Pet fitness and its imperative veterinary care are pivotal yet still neglected areas in public health. This is particularly the case with zoonotic pathogens, whose transmission to humans can be facilitated through the closeness of pet–owner interactions. Parasites can be examples of such zoonotic transmission [1].

One very popular pet is the domestic cat (*Felis catus*). Due to its predatory nature, the cat is a successful definitive host for several heteroxenous zoonotic parasites, such as the cosmopolitan *Toxoplasma gondii* [1,2]. *T. gondii* is an apicomplexan parasite that can

virtually infect any homeothermic vertebrate, including humans. Only in Felidae, however, can *T. gondii* sexually reproduce and, consequentially, shed unsporulated oocysts [2–4]. Aerated conditions trigger the sporulation of these oocysts, generating a highly resistant oocyst (with four sporozoites) within 1 to 5 days. Infection of warm-blooded intermediate hosts after oral ingestion of the sporulated oocysts can affect any host organ or tissue. Cell invasion and tissue colonization by *T. gondii* culminate in slowed metabolism and conversion to bradyzoites within somatic cysts [4]. These cysts can infect other hosts (intermediate or definitive) that feed on the cyst-infected animals. Felidae commonly contract *T. gondii* infection through this route [5], but oral ingestion of sporulated oocysts can also cause infection [4]. Asexual multiplication of *T. gondii* in the cat's enterocytes—occasionally with migration to other organs—concludes with a phase of sexual reproduction. Here, oocysts are produced and shed in the cat's feces between 1 and 3 weeks after infection [4]. Re-shedding of oocysts in previously infected cats has been reported with immunosuppression [6] and in immunocompetent cats under experimental conditions [2,3,6],

T. gondii in cats occurs, in most cases, as an asymptomatic infection [5,7,8]. Clinical toxoplasmosis, although rare, can be present with mild to severe clinical signs [2], and cases with rapid fatal development have been reported [9,10]. While *T. gondii* can colonize several organs in cats, most of the reported clinical cases implicate respiratory [9,11], ocular [12], or neurological [13,14] disease. The clinical presentation can include signs like fever, vomiting, diarrhea, weight loss, anorexia, lethargy, icterus, dyspnea, myalgia, enlarged lymph nodes, and/or hypothermia [2,5,7,8,15]. Immunosuppressive therapies are considered a risk factor in the development of clinical toxoplasmosis as they can lead to the activation of bradyzoite cysts [5,7,9,10,14].

Diagnostic investigation for *T. gondii* in feline patients can be via direct detection of oocysts in feces, histopathological identification of bradyzoite cysts in tissue followed by immunohistopathological staining, visualization of intracellular tachyzoites in mononuclear cells in blood or other fluids, or PCR-based detection of fecal, blood, or tissue DNA [2,5]. Seropositivity through the detection of *T. gondii* immunoglobulin M (IgM) or IgG antibodies can be used as an indicator of previous or ongoing infection in late stages [8,16].

Human infection can occur through different routes: ingestion of undercooked meat from infected intermediate hosts; contamination of food and/or water with oocysts; congenital transmission; blood transfusion and organ transplantation; and direct contact with sporulated oocysts shed in cat feces. Human toxoplasmosis is of particular importance during pregnancy and for immunosuppressed patients. In the former, it can lead to congenital abnormalities or fetal death [17]. In the latter, it could cause severe or fatal disease [18].

In the geographic area of Germany, little information regarding the occurrence and exposure of *T. gondii* in the domestic cat population and the primary clinical concerns of affected cats and potential risk factors (i.e., patient characteristics, environmental, and lifestyle factors) exists [5,19–21]. The data available from this geographic area is mainly derived from fecal sample analyses with a focus on parasite typing (detecting mostly genotype II) [19,20] and/or rates of *T. gondii* oocyst shedding (determined as 0.1–1.6% depending upon the population studied and the cats' age) [20–23]. A single cross-sectional study found several patient signalment criteria to affect *T. gondii* serological results [21]. Thus, the objective of this study was to investigate possible risk factors in feline patients seropositive for *T. gondii* admitted to a small animal veterinary teaching hospital in the central part of Germany over an 11-year period.

2. Materials and Methods

2.1. Medical Record Review and Patient Selection Criteria

The digital medical records of feline patients that were presented at the Small Animal Veterinary Teaching Hospital (VTH), the Department for Small Animals at Leipzig University, Germany, between 2012 and 2023 were considered to be used for this retrospective study. Only feline patients that were kept as pet cats and underwent testing for *T. gondii* antibodies (IgG and/or IgM) as part of their routine diagnostic evaluation (with varying degrees of suspicion for a *T. gondii* infection) were considered for inclusion. Animals younger than 12 weeks and positive for *T. gondii* IgG titers were excluded from this study, given the possibility of maternal antibodies being detected [24,25]. Besides *T. gondii* serological status, the following patient information was retrieved from the medical records: patient criteria (age, sex, and neuter status), environmental factors (feeding, housing, possible travel, zip code, and the city of the owner's address), medical history data (vaccination and deworming status), and clinical diagnosis. For the detection of both IgG and IgM *T. gondii* antibodies, an indirect immunofluorescence assay (IFA) was conducted at a commercial laboratory (IDEXX Laboratory, Kornwestheim, Germany).

2.2. Statistical Analysis

Summary statistics are presented as medians (continuous data) or counts (n) and percentages (%) with their respective confidence intervals (95% CI) (ordinal data). To evaluate for possible associations between *T. gondii* seropositivity and risk factors (age, sex, husbandry, feeding practices, and clinical signs), logistic regression (continuous data) and a likelihood ratio (X^2) test (ordinal data) or Fisher's exact test (if $n \leq 5$ in any group) were performed ($\alpha = 0.05$), and the corresponding odds ratio (OR) was calculated. Risk factors identified in univariate analysis were entered into a multinomial logistic regression model to determine their effect on *T. gondii* seropositivity. A Wilcoxon rank-sum test served for two-group comparisons of non-parametric data. JMP® v13.0 (SAS, Cary, NC, USA) was used for all statistical analyses and geographical mapping.

3. Results

The records of a total of 174 cats were included and analyzed for this study. Most of the animals (68%) resided in the city of Leipzig (Figure 1). The remainder of the medical records included feline patients from the neighboring districts in the federal states of Saxony and Saxony-Anhalt. Additionally, a small number of cats were located in the states of Brandenburg, Bavaria, and Thuringia (Figure 1). Between 5 and 30 cats (median: 12) included in this study were presented during the same year (Figure 2). The majority of the cats evaluated for *T. gondii* had a positive serology. Overall, 100 animals were seropositive for *T. gondii* (57.5%; CI = 50.0–64.6%). Of those 100 cats, 28 were positive for IgM antibodies (16.4%; CI = 11.6–22.6%), and IgG antibodies were detected in 94 cats (54.3%; CI = 46.9–61.6%) (Table 1).

Various clinical signs were recorded in the *T. gondii* IgM-positive cats at the time of presentation at the small animal VTH. Neurological signs were the most common (n = 16; 57.1%) among these, followed by clinical signs of a locomotor (n = 7; 25.0%) or ocular condition (n = 3; 10.7%) and other non-specific or systemic signs, including fever, cardiovascular, respiratory, gastrointestinal, or dermatologic signs (Table 1).

The sex distribution was nearly balanced, with 81 (46.6%) female and 93 (53.4%) male cats. In total, 46 of the female animals were *T. gondii* antibody-positive (56.8%; CI = 45.9–67.0%); 10 were positive for IgM (12.7%; CI = 7.0–21.8%); and 44 of the cats showed IgG antibodies (54.3%; CI = 43.5–64.78%). The male group comprised 54 individuals that were positive for *T. gondii* immunoglobulin (58.0%; CI = 47.9–67.6%), of which 18 male cats showed IgM

antibodies (19.6%; CI = 12.7–28.8%), and *T. gondii* IgG was detected in 50 of the male cats (54.3%; CI = 44.2–64.1%). No significant association was found between sex and *T. gondii* antibody positivity ($p = 0.865$), *T. gondii* IgM positivity ($p = 0.220$), or *T. gondii* IgG positivity ($p = 0.997$). In total, 54 (66.7%; CI = 55.8–76.0%) female cats were spayed, and 74 (79.6%; CI = 70.3–86.5%) of the male patients were neutered. While sex had no significant effects on *T. gondii* antibody positivity, significantly more neutered cats (males and females combined) were represented in the *T. gondii* IgG-positive group (79.8%; CI = 70.6–86.7%) than among the *T. gondii* IgG-negative cats (65.8%; CI = 54.9–75.3%; $p = 0.039$; OR = 2.05); however, significance for the same disproportion was not reached when analyzing female cats ($p = 0.083$) and male cats ($p = 0.230$) separately.

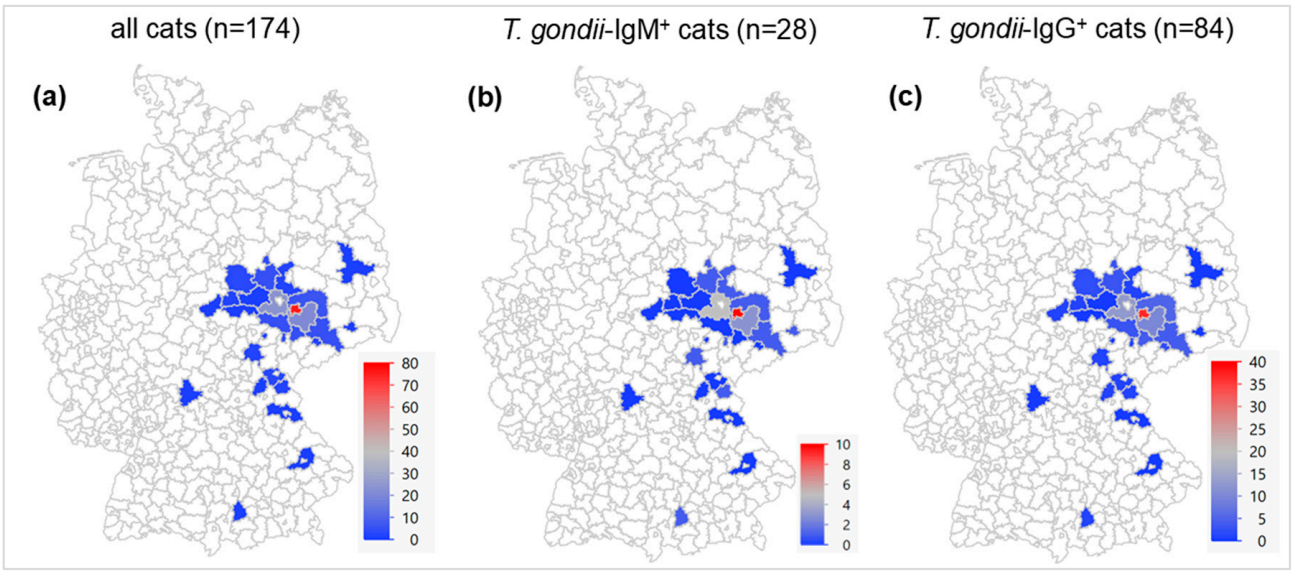


Figure 1. Geographical distribution of feline patients included in this study: (**a**) map of Germany showing the districts from which cats ($n = 174$) were included in this study and the spatial distribution of (**b**) *Toxoplasma gondii* IgM-positive (*T. gondii*-IgM$^+$) cats ($n = 28$) and (**c**) *Toxoplasma gondii* IgG-positive (*T. gondii*-IgG$^+$) cats ($n = 84$). Color indicators (heat map) for the total number of animals are included in the lower right of the maps.

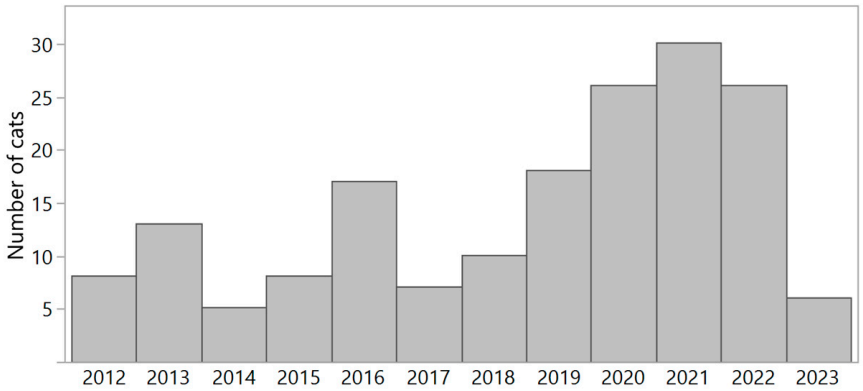


Figure 2. Temporal distribution of feline patients included in this study. The distribution of the cases is shown (cats tested for *Toxoplasma gondii* IgM and/or IgG over the 12 years [2012–2023]).

Table 1. Seroprevalence of *Toxoplasma gondii* by antibody type (IgM and/or IgG) and patient demographics.

Parameter	Group	IgM$^+$	IgG$^+$	Ab [1]
Total animals		28/171 (16.4%)	94/173 (54.3%)	100/174 (57.5%)
Sex	Females	10/28 (35.7%)	44/94 (46.8%)	46/100 (46.0%)
	Males	18/28 (64.3%)	50/94 (53.2%)	54/100 (54.0%)
Age	<1 year	1/28 (3.6%)	8/94 (8.5%)	9/100 (9.0%)
	1–9 years	10/28 (35.7%)	34/94 (36.2%)	34/100 (34.0%)
	≥10 years	17/28 (60.7%)	52/94 (55.3%)	57/100 (57.0%)
Breed	DSH	22/28 (78.6%)	72/94 (76.6%)	75/100 (75.0%)
	Non-DSH	6/28 (21.4%)	22/94 (23.4%)	25/100 (25.0%)
Clinical presentation	Neurological	16/28 (57.1%)	51/94 (54.3%)	54/100 (54.0%)
	Locomotor	7/28 (25.0%)	22/94 (23.4%)	23/100 (23.0%)
	Ocular	3/28 (10.7%)	12/94 (12.8%)	12/100 (12.0%)
	Respiratory	2/28 (7.1%)	6/94 (6.4%)	13/100 (13.0%)
	Cardiovascular	2/28 (7.1%)	13/94 (13.8%)	17/100 (17.8%)
	Gastrointestinal	2/28 (7.1%)	8/94 (8.5%)	8/100 (8.0%)
	other	9/28 (32.1%)	30/94 (31.2%)	33/100 (33.0%)

DSH: Domestic Shorthair. [1] Patients seropositive for either IgM, IgG, or both.

The ages of the cats ranged from 0.3 to 22.7 years (median: 6.5 years). The animals were separated into three age groups: less than 1 year (<1 year) for juveniles; cats were considered to be adults if they were between 1 and 9 years; and cats of 10 years or older (≥10 years) were classified as geriatric. *T. gondii* IgG positivity was associated with a higher age at presentation (median: 7.9 years) than a negative *T. gondii* IgG titer (median: 5.7 years; $p = 0.008$), but a similar difference was not seen between *T. gondii* IgM-positive cats and those without a *T. gondii* IgM titer ($p = 0.296$). Young animals were less frequently seropositive than the other two age groups of cats, with only 1/23 kittens being positive for IgM antibodies (4.4%; CI = 0.7–21.0%), 8/23 kittens being seropositive for IgG (34.8%; CI = 18.8–55.1%), and 9 animals being positive for either Ig or both (39.1%; CI = 22.2–59.2%). A total of 96 cats were categorized as adults, 57 of which (57.6%; CI = 47.7–66.8%) were seropositive for *T. gondii* (IgM, IgG, or both). IgM antibodies were found in 17 adult cats (17.7%; CI = 11.4–26.5%), and 52 cats were positive for IgG (53.1%; CI = 43.3–62.6%) (Table 1). The possible risk of age and sole IgM positivity ($p = 0.296$) was not significant. However, a significant association was seen between age and the presence of IgG ($p = 0.008$; OR = 6.14) or seropositivity for any or both antibodies ($p = 0.024$; OR = 4.60), with geriatric animals being more likely to have *T. gondii* antibodies, followed by adult cats.

The cats were predominantly Domestic (European) Shorthair ($n = 121$; 69.5%); the remaining cats ($n = 53$; 30.5%) were of other breeds (12 different breeds in total), including mixed-breed cats. Information on husbandry and environmental factors was available for 125 cats. These included 81 indoor cats (64.8%; CI: 56.1–72.6%), 8 indoor cats with access to the outside (6.5%; CI = 3.3–12.1%), 32 outdoor cats (25.6%; CI = 18.8–33.9%), and 4 cats that resided in a local shelter (3.2%; CI = 1.3–7.9%). Information on vaccination status was available for 84 cats, of which 50 were regularly vaccinated (59.5%; CI = 48.8–69.4%), 15 cats were irregularly vaccinated (17.9%; CI = 11.1–27.4%), and 19 cats were unvaccinated (22.6%; CI = 15.0–32.7%). The deworming status of 77 cats was recorded: 57 cats were regularly dewormed (74.0%; CI = 63.3–82.5%), 8 were irregularly dewormed (10.4%; CI = 5.4–19.2%), and 12 cats were not dewormed (15.6%; CI = 9.1–25.3%).

The data from the patient history focused on feeding routines/practices, and living or temporarily residing abroad were also included in the evaluation. A thorough dietary

history was available for 34 cats, of which 10 cats (29.4%; CI = 16.8–46.2%) were fed exclusively a dry kibble diet, 7 cats (20.6%; CI = 10.4–36.8%) received exclusively canned (wet) food, and 3 cats (8.8%; CI = 3.1–23.0%) were offered a raw meat-based diet (RMBD). Only 1 of the 47 (2.1%; CI = 0.4–11.1%) recorded travel histories revealed a possible stay outside of Germany; the remaining 46 cats (97.9%; CI = 88.9–99.6%) had never been abroad. Retrovirus status was determined for 20 cats and was negative for all cases.

Vaccination status ($p = 0.728$, $p = 0.242$, and $p = 0.504$), foreign travel history ($p = 0.340$, $p = 0.5340$, and $p = 0.305$), and deworming status ($p = 0.497$, $p = 0.260$, and $p = 0.229$) did not affect *T. gondii* seropositivity or *T. gondii* IgM or *T. gondii* IgG status. However, more outdoor cats (44.1% vs. 24.6%) and fewer indoor-only cats (55.9% vs. 75.4%; $p = 0.021$) were represented among *T. gondii* IgG-positive cats (OR = 2.43). In addition, there was a significant correlation between feeding and *T. gondii* serum positivity. Compared with *T. gondii* IgG-negative cats, *T. gondii* IgG-positive animals were fed more frequently canned (wet) food (87.5%; CI = 52.9–97.8%) than a dry kibble diet (12.5%; CI = 2.2–47.1%; $p < 0.0001$). A similar trend was also observed for *T. gondii* IgM positivity ($p = 0.081$). Multivariate analysis identified neuter status ($p = 0.002$) and diet choice ($p = 0.004$) as significant determinants of a *T. gondii* IgG-positive status.

4. Discussion

This retrospective study evaluated possible risk factors for *T. gondii* infection and seropositivity in a pet cat population that presented to a tertiary veterinary care facility in the geographic area of central Germany. Significant predisposing factors included age, feeding practices, and husbandry.

An increase in the frequency of a positive *T. gondii* IgG titer was documented with a higher age category, which aligns with studies evaluating fecal *T. gondii* shedding [20,26] and one serology study in cats [21] and is most likely attributable to the cumulative risk of (possibly repeated) environmental exposure to *T. gondii* (e.g., from contaminated food/meat or contaminated soil or fresh feces from a cat shedding *T. gondii* oocysts) and the persistence of bradyzoite cysts and, thus, persistent *T. gondii* IgG titers over the lifetime of a cat [2]. Such exposure to *T. gondii* cysts or oocysts can be reasonably expected to be more prevalent in outdoor cats or cats with intermittent outdoor access [5]. This could explain the increased proportion of *T. gondii* IgG-positive cats in the subpopulation allowed to spend time outside, which has also not been previously reported or investigated [19–21,26]. Whether contact with rodent intermediate hosts (e.g., mice) could also present a source for exposure to *T. gondii* oocysts remains to be confirmed as these hosts are prey for cats but typically are not readily eaten or even killed after being caught and trapped [27]. Additional research examining the specific role of prey animals, particularly rodents as intermediate hosts, could provide clearer insight into the environmental transmission dynamics of *T. gondii*.

An unexpected finding of this study was the higher prevalence of *T. gondii* serum positivity in cats fed predominantly wet (canned) food than a dry kibble diet. This risk factor has not been explored by others [19,20,26] and could also be determined by other colinear variables (e.g., the feeding method depending on the type of housing). The possibility of exposure to *T. gondii* tissue cysts in certain meat-containing wet (canned) foods would seem unlikely but cannot be entirely ruled out. While the risk of insufficient or even incorrect processing of the meat in the production process should be very low [28], contamination during the preparation of a cat's meal in a private household (especially when feeding or mixing in undercooked or raw cyst-containing meat or meat products originating from infected animals) is a more likely scenario. This emphasizes the need for continued surveillance and quality control in pet food manufacturing, as well as the

importance of the proper storage, handling, and preparation of cat meals, especially for owners who prepare homemade diets or feed raw meat-based products.

Clinical representations in this study ranged from mild to severe clinical signs, as has been previously reported [2,9,10]. Similar to other reports [9,11–14], this study found the majority of clinical signs in affected cats to be neurological, ocular, and/or respiratory. However, given the retrospective analysis of cases presented to a tertiary veterinary care facility, asymptomatic infections that can occur with *T. gondii* [5,7,8] were likely not represented in this group of cats. However, the causative role of *T. gondii* for the clinical signs displayed, leading to *T. gondii* being considered as a differential diagnosis, is equally challenging to confirm for all cats included in this study.

The findings of this study support possible clinical presentations and risk factors for feline toxoplasmosis. However, several points merit additional discussion. First, the detection of *T. gondii* antibodies does not necessarily confirm active shedding or current infectivity. *T. gondii* IgM positivity may suggest recent exposure, whereas *T. gondii* IgG positivity typically indicates past exposure and a long-term immune response rather than ongoing oocyst shedding. Furthermore, an age cut-off of 12 weeks for positive *T. gondii* IgG titers was used in this study. Maternal antibodies against *T. gondii* in cats are typically transferred through colostrum, and these antibodies wane at around 6–12 weeks of age, depending on the kitten's immune system and level of maternal immunity [24,25]. By 12 weeks, maternal antibodies have declined to undetectable or very low levels in most cats [24], rendering this age a reasonable cut-off to differentiate passive immunity against *T. gondii* from an active *T. gondii* infection. While the decline in maternally-derived antibodies shows some inter-individual variation and can take up until 16 weeks of age to completely disappear in some cats [24], the 12-week cut-off appears to have the best separating ability in a larger population of cats, where antibodies detected after 12 weeks are more likely generated by the kitten's immune response to infection. In addition, the youngest cat included in our study was 17.1 weeks of age. Third, the retrospective design and reliance on cases from a single tertiary veterinary care center may limit the generalizability of these results. Selection biases, incomplete medical records, and limited sample sizes for certain variables may influence the observed associations. Prospective, multicenter studies with standardized diagnostic protocols and comprehensive dietary, environmental, and behavioral data collection would offer further insights. Lastly, geographical variability is an important consideration. While this study provides valuable data for the region studied, *T. gondii* prevalence, strain diversity, and transmission pathways can differ significantly between areas [9,29–33]. Thus, confirming our serology-associated results in other parts of Germany and different countries would strengthen their applicability and inform region-specific preventive strategies. Confirmation of our results, while expected, would also allow us to consider revisiting the application of preventive measures against *T. gondii* exposure and infection in humans, particularly to protect pregnant women and developing fetuses as well as immunodeficient people.

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Institutional Review Board Statement: Ethical review and approval are not applicable to this study. Permission (owner consent) for using anonymized data and also surplus sampling material for research and educational purposes was given by every patient's owner during the signing of the standard hospital admission form of the Department for Small Animals, the College of Veterinary Medicine, Leipzig University, Germany.

Informed Consent Statement: Written owner consent for the use of anonymized patient data was obtained through the hospital admission form. No additional testing was performed for the purpose of this retrospective study.

Data Availability Statement: The anonymized data and further information are available from the corresponding author upon reasonable request.

Conflicts of Interest: The authors declare no conflicts of interest pertaining to this study.

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