



## Effectiveness of ultrasound examination in mono- and mixed infections of dogs

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Protozoan and helminthic gastrointestinal parasites of domestic dogs are very common invasive pathogens. Depending on the parasitic load on the body of a sick animal, the course of the infection varies from asymptomatic to severe, sometimes fatal. In the detection of various pathological conditions of the gastrointestinal tract, an important place belongs to ultrasound examination. It is the fastest, most informative and accessible method of visualization in veterinary medicine, and also allows one to safely assess the state of the abdominal organs and detect pathological changes in good time. The aim of the research was to investigate the informativeness of ultrasound examination of the stomach and small intestine in toxocariasis monoinfection and toxocariasis-cystoisosporiasis co-infection in dogs. It was found that parasitism by *Toxocara canis* and *Cystoisospora canis* led to changes in the metric indicators of the stomach and small intestine wall. Even more significant changes were found in toxocariasis-cystoisosporiasis co-infection than in toxocariasis monoinfection. In the latter case, regardless of the weight category of dogs, the changes were characterized by simultaneous thickening of the stomach wall (by 15.4–46.4%) and duodenum (by 10.3%) in dogs weighing 5–15 kg, and duodenum (by 15.1%) in dogs weighing 15–30 kg. Also, in dogs weighing 5–15 kg the thickness of the jejunum wall was reduced by 18.2–32.1%, that of the ileum as well, (by 8.0%) and the ileum wall was thinner in dogs weighing 15–30 kg (by 12.1%). It was found that all dogs infected with *T. canis* had a thickened submucosal layer of the stomach wall (by 42.9–50.0%) and a thinned mucosal layer of the jejunum wall (by 18.5–35.0%). In dogs weighing 5–15 and 15–30 kg, thickening of the mucous layer of the duodenal wall (by 11.1% and 8.5%, respectively) and thinning of the jejunum wall (by 20.0% and 37.5%, respectively) were detected. In toxocariasis-cystoisosporiasis co-infection, regardless of the weight category of dogs, the changes in the intestines were characterized by thickening of the gastric wall (by 33.3–67.9%), duodenum wall (by 20.9–26.5%) and thinning of the jejunum (by 24.2–39.3%) and ileum wall (by 8.2–15.2%). Changes in the walls occurred due to thickening of all layers of the stomach (by 13.6–80.0%), duodenum (by 12.6–75.0%), as well as thinning of all layers of the jejunum wall (by 20.0–45.0%). Thinning of the ileum wall occurred due to thinning of the mucous layer (by 24.0–40.0%). The obtained data confirm the feasibility of using ultrasound in the assessment of structural and functional changes in the gastrointestinal tract of dogs with mono- and co-infections.

*Keywords: Toxocara canis; Cystoisospora canis; stomach; duodenum; morphometry; imaging method.*

### Introduction

Endoparasites are among the most common pathogens of gastrointestinal diseases in dogs (Grandi et al., 2021; Murnik et al., 2023; Csokai et al., 2024). In addition, dogs can play an active role in the transmission of intestinal parasites to humans, given their cohabitation, making canine gastrointestinal parasitosis a serious public health problem, as it poses a risk to humans, especially children (Souza et al., 2023; Reginaldo et al., 2025; Salant et al., 2025).

Scientists from most countries of the world note that helminths of the species *Toxocara canis* and the protozoan *Cystoisospora* spp. are the most common, due to the high resistance of their exogenous stages of development to adverse environmental factors (Shukullari et al., 2015; Nagamori et al., 2020; Ilić et al., 2021). In particular, in Egypt, the prevalence of *T. canis* in dogs was 19.2%, *C. canis* – 5.1%, *C. ohioensis* – 2.6% (Abbas et al., 2023). In the southeastern USA and North Central Nigeria, *T. canis* was detected in 24.2% and 19.0% of the dogs studied, respectively, *Cystoisospora* spp. – in 3.2% and 25.3% (Ola-Fadunsin et al., 2023; Zhao et al., 2024). In Serbia and Argentina, during coproscopic examination of dogs, *Cystoisospora* spp. was detected in 9.2% and 7.5% of animals, *T. canis* – in 11.5% and 5.0% (Cociancic et al., 2020; Jovanovic et al., 2024). Moreover, there are reports of gastrointestinal co-infections in dogs. In particular, in Mexico, co-infection caused by two or three parasites was detected in 21.3% and 3.1% of dogs, respectively (Rodríguez-Vivas et al., 2011). In Ethiopia, gastrointestinal parasitosis occurred as monoinfection in 39.7% of dogs, and as co-infections in 11.6% of dogs (Ge-

bremedhin et al., 2020). In Serbia, mixed intestinal infections with up to four species of parasites were observed in 44.7% of animals according to the results of coproscopic studies of dogs (Nikolić et al., 2008).

The consequences of such parasitic infestations can vary from subclinical to severe, sometimes leading to the death of animals. Helminths can cause growth and development retardation in animals, disorders of the gastrointestinal tract, development of immunodeficiency and low resistance to other concomitant diseases (Kirkova et al., 2005; Cervone et al., 2024). The presence of parasites in the animal body causes local and general changes in the various organs in which they parasitize or transit during their life cycle, which leads to certain pathological changes (Pinto et al., 2011; Salih & Albayati, 2025).

Ultrasound sonography (USG) plays an important role in the detection of various pathological conditions of the gastrointestinal tract, including gastrointestinal endoparasites in animals. This method is highly effective, non-invasive, allowing visualization of the layers of the intestinal wall, assessment of peristalsis, detection of intussusception, helminths, and inflammatory processes (Arsenopoulos & Fthenakis, 2017; Corda et al., 2022). In particular, USG is an alternative method for diagnosing *T. canis* infection in puppies during the prepatent period (Corda et al., 2019). USG is also useful for detecting signs of pulmonary arterial hypertension, various pathological heart conditions caused by dirofilariasis (Arita et al., 2003; Kim et al., 2019). Other studies of Mesocestoididae infection in dogs have shown ascites with multiple cystic structures floating freely in the

fluid and attached to the surface of the abdominal organs and omentum (Venco et al., 2005). An ultrasound examination of a dog infected with *Metorchis conjunctus* has been described. The changes were characterized by multiple liver abscesses, dilatation of the bile ducts and thickening of their walls, and the presence of echogenic sediment in the gallbladder and ducts (Lemetayer et al., 2016).

Therefore, ultrasound imaging plays an important role in diagnostics, determining the stages of pathogenesis, and monitoring the treatment process for parasitic diseases in veterinary medicine. Therefore, it is relevant to establish the features of changes in the gastrointestinal tract of dogs in different forms of parasitosis.

The aim of the research was to investigate the informativeness of USG examination of the stomach and small intestine in cases of toxocariasis monoinfection and toxocariasis-cystoisosporiasis co-infection in dogs.

## Materials and methods

The study was conducted in the private veterinary clinic “Dovira” (Kharkiv) and the parasitology laboratory of the Poltava State Agrarian University (Poltava) in 2024–2025.

Three groups of dogs were formed: a control group (clinically healthy dogs) and two experimental groups (animals from one group were infected with *T. canis*, dogs of another group were infected with *T. canis* and *C. canis*) with different weight categories of animals: < 5 kg (n = 6), 5–15 kg (n = 7), 15–30 kg (n = 6). Parasitism of *T. canis*

and *C. canis* was confirmed by copro-ovoscopic examination of dogs and detection of *Toxocara* eggs and *Cystoisospora* oocysts. USG examination of the stomach and various parts of the small intestine (duodenum, jejunum, ileum) in dogs was performed using the Chison Qbit 5 device (Chison, China) with linear (11.5–16.5 MHz) and microconvex (6.5–11.7 MHz) sensors. Echocardiographic assessment was implemented to evaluate total wall thickness and its layers (mucosal, submucosal, muscular) of the stomach, duodenum, jejunum, ileum (mm) in dogs of the experimental and control groups.

Standard deviation (SD) and average values (x) were calculated. Differences between the values of the groups were determined using the Tukey test, the differences were considered significant at  $P < 0.05$ .

## Results

Ultrasound studies revealed that parasitization by *T. canis* and *C. canis* led to changes in the metric indicators of the stomach and intestinal wall. Moreover, more significant changes were found in toxocariasis-cystoisosporiasis infection than in toxocariasis alone. In particular, in dogs weighing < 5 kg with toxocariasis monoinfection, the stomach wall thickened (by 46.4%,  $P < 0.05$ ) and the jejunum wall thinned (by 32.1%,  $P < 0.05$ ) compared to clinically healthy animals. In the case of toxocariasis-cystoisosporiasis co-infection in dogs, the walls of the stomach thickened (by 67.9%) and duodenum (by 26.5%) ( $P < 0.05$ ), and the walls of the jejunum and ileum thinned (by 39.3% and 8.2%, respectively;  $P < 0.05$ , Table 1).

**Table 1**

Thickness parameters of the wall segment and individual layers of the stomach and small intestine in dogs weighing < 5 kg with either toxocariasis monoinfection or toxocariasis-cystoisosporiasis co-infection (x ± SD, n = 6)

Parameters of segments of gastrointestinal tract, mm	Clinically healthy dogs	Dogs with <i>T. canis</i> infection	Dogs with <i>T. canis</i> and <i>C. canis</i> co-infection
<b>Stomach</b>			
Thickness of the wall	2.77 ± 0.63 <sup>a</sup>	4.07 ± 0.34 <sup>b</sup>	4.65 ± 0.22 <sup>b</sup>
Thickness of the mucous layer	2.17 ± 0.05 <sup>a</sup>	2.22 ± 0.08 <sup>a</sup>	2.53 ± 0.19 <sup>b</sup>
Thickness of the submucosal layer	0.47 ± 0.05 <sup>a</sup>	0.82 ± 0.24 <sup>b</sup>	0.92 ± 0.21 <sup>b</sup>
Thickness of the muscular layer	0.67 ± 0.05 <sup>a</sup>	1.03 ± 0.08 <sup>b</sup>	1.20 ± 0.11 <sup>c</sup>
<b>Duodenum</b>			
Thickness of the wall	3.42 ± 0.08 <sup>a</sup>	3.60 ± 0.17 <sup>a</sup>	4.32 ± 1.17 <sup>b</sup>
Thickness of the mucous layer	2.52 ± 0.12 <sup>a</sup>	2.50 ± 0.13 <sup>a</sup>	2.92 ± 0.13 <sup>b</sup>
Thickness of the submucosal layer	0.43 ± 0.05 <sup>a</sup>	0.48 ± 0.08 <sup>a</sup>	0.68 ± 0.08 <sup>b</sup>
Thickness of the muscular layer	0.53 ± 0.05 <sup>a</sup>	0.62 ± 0.08 <sup>ab</sup>	0.72 ± 0.08 <sup>b</sup>
<b>Jejunum</b>			
Thickness of the wall	2.77 ± 0.15 <sup>a</sup>	1.93 ± 0.12 <sup>b</sup>	1.67 ± 0.18 <sup>c</sup>
Thickness of the mucous layer	1.97 ± 0.05 <sup>a</sup>	1.30 ± 0.14 <sup>b</sup>	1.10 ± 0.11 <sup>c</sup>
Thickness of the submucosal layer	0.37 ± 0.05 <sup>a</sup>	0.30 ± 0.09 <sup>ab</sup>	0.25 ± 0.05 <sup>b</sup>
Thickness of the muscular layer	0.45 ± 0.05 <sup>a</sup>	0.33 ± 0.08 <sup>b</sup>	0.32 ± 0.08 <sup>b</sup>
<b>Ileum</b>			
Thickness of the wall	2.07 ± 0.05 <sup>a</sup>	2.05 ± 0.08 <sup>a</sup>	1.90 ± 0.09 <sup>b</sup>
Thickness of the mucous layer	0.43 ± 0.05 <sup>a</sup>	0.38 ± 0.08 <sup>a</sup>	0.27 ± 0.05 <sup>b</sup>
Thickness of the submucosal layer	1.27 ± 0.05 <sup>a</sup>	1.28 ± 0.04 <sup>a</sup>	1.28 ± 0.08 <sup>a</sup>
Thickness of the muscular layer	0.37 ± 0.05 <sup>a</sup>	0.38 ± 0.04 <sup>a</sup>	0.35 ± 0.05 <sup>a</sup>

Note: different letters in the table within each line indicate significant ( $P < 0.05$ ) differences between groups according to Tukey's test results.

When analyzing the thickness indicators of the layers of the stomach and small intestine wall, it was found that with toxocariasis monoinfection, the thickness of the submucosal and muscular layers of the stomach wall increased ( $P < 0.05$ , by 60.0% and 47.1%), and the thickness of the mucous and muscular layers of the jejunum wall decreased ( $P < 0.05$ ) compared to clinically healthy animals. In toxocariasis-cystoisosporiasis co-infection, the changes were more significant and characterized by an increase ( $P < 0.05$ ) in the thickness of all layers of the stomach wall (by 13.6–80.0%), duodenum (by 16.0–75.0%), a decrease ( $P < 0.05$ ) in the thickness of all layers of the jejunum wall (by 37.5–45.0%) and the thickness of the ileum mucosa (by 25.0%).

During ultrasound examination of dogs weighing 5–15 kg, it was found that with toxocariasis monoinfection, the thickness of the stomach wall and all parts of the intestine changed ( $P < 0.05$ ) compared with clinically healthy animals. In particular, in dogs infected with *T. canis*, walls of the stomach and duodenal were thicker (by 15.4% and 10.3%), and the jejunum and ileum walls were thinner (by 18.2% and 8.0%). With toxocariasis-cystoisosporiasis co-infection, the chan-

ges were more pronounced and characterized by thickening of the stomach and duodenum walls (by 33.3% and 23.1%), as well as thinning of the jejunum and ileum walls (by 24.2% and 12.0%, Table 2).

Metrics of the thickness of the stomach wall layers in toxocariasis monoinfection are characterized by thickening only of the submucosal layer (by 42.9%,  $P < 0.05$ ), and in toxocariasis-cystoisosporiasis co-infection – of the mucous (by 26.1%), submucosal (by 44.3%) and muscular (by 44.4%) layers compared to clinically healthy animals ( $P < 0.05$ ). In the small intestine, in dogs with *T. canis* parasitism, thickening of the mucous layer of the duodenal wall (by 11.1%,  $P < 0.05$ ), thinning of the mucous layer of the jejunum and ileum intestines was noted (by 18.5% and by 20.0%, respectively;  $P < 0.05$ ). As for the association of *T. canis* and *C. canis*, more pronounced changes were detected, which are characterized by thickening of all layers of the duodenal wall (by 12.6–50.0%,  $P < 0.05$ ), thinning of all layers of the jejunum wall (by 20.0–33.3%,  $P < 0.05$ ), and thickness of the ileum mucosa (by 40.0%,  $P < 0.05$ ).

In dogs weighing 15–30 kg, it was found that the walls of the stomach and duodenum thickened (by 19.5% and 15.1%, respective-

ly,  $P < 0.05$ ) in the case of toxocarosis monoinfection. Also, thinning of the walls of the jejunum and ileum was detected (by 23.7% and 12.1%, respectively,  $P < 0.05$ ). With toxocarosis-cystoisosporiasis co-infection, the changes are characterized by more pronounced thic-

kening ( $P < 0.05$ ) of the walls of the stomach and duodenum (by 34.1% and 20.9%), as well as thinning ( $P < 0.05$ ) of the walls of the jejunum and ileum (by 36.8% and 15.2%, Table 3).

**Table 2**

Thickness parameters of the wall segment and individual layers of the stomach and small intestine in dogs weighing 5–15 kg with either toxocarosis monoinfection or toxocarosis-cystoisosporiasis co-infection ( $x \pm SD$ ,  $n = 7$ )

Parameters of segments of gastrointestinal tract, mm	Clinically healthy dogs	Dogs with <i>T. canis</i> infection	Dogs with <i>T. canis</i> and <i>C. canis</i> co-infection
<b>Stomach</b>			
Thickness of the wall	3.89 ± 0.11 <sup>a</sup>	4.46 ± 0.33 <sup>b</sup>	5.17 ± 0.17 <sup>c</sup>
Thickness of the mucous layer	2.26 ± 0.08 <sup>a</sup>	2.47 ± 0.36 <sup>a</sup>	2.89 ± 0.17 <sup>b</sup>
Thickness of the submucosal layer	0.73 ± 0.08 <sup>a</sup>	0.97 ± 0.14 <sup>b</sup>	1.01 ± 0.13 <sup>b</sup>
Thickness of the muscular layer	0.90 ± 0.08 <sup>a</sup>	1.01 ± 0.12 <sup>a</sup>	1.27 ± 0.14 <sup>b</sup>
<b>Duodenum</b>			
Thickness of the wall	3.87 ± 0.19 <sup>a</sup>	4.34 ± 0.18 <sup>b</sup>	4.08 ± 0.33 <sup>c</sup>
Thickness of the mucous layer	2.71 ± 0.23 <sup>a</sup>	3.00 ± 0.16 <sup>b</sup>	3.04 ± 0.22 <sup>b</sup>
Thickness of the submucosal layer	0.57 ± 0.17 <sup>a</sup>	0.63 ± 0.05 <sup>a</sup>	0.84 ± 0.08 <sup>b</sup>
Thickness of the muscular layer	0.59 ± 0.07 <sup>a</sup>	0.71 ± 0.12 <sup>a</sup>	0.91 ± 0.11 <sup>b</sup>
<b>Jejunum</b>			
Thickness of the wall	3.34 ± 0.22 <sup>a</sup>	2.74 ± 0.13 <sup>b</sup>	2.49 ± 0.12 <sup>c</sup>
Thickness of the mucous layer	2.23 ± 0.14 <sup>a</sup>	1.69 ± 0.19 <sup>b</sup>	1.67 ± 0.17 <sup>b</sup>
Thickness of the submucosal layer	0.59 ± 0.07 <sup>a</sup>	0.54 ± 0.15 <sup>ab</sup>	0.41 ± 0.13 <sup>b</sup>
Thickness of the muscular layer	0.53 ± 0.11 <sup>a</sup>	0.51 ± 0.07 <sup>ab</sup>	0.40 ± 0.08 <sup>b</sup>
<b>Ileum</b>			
Thickness of the wall	2.54 ± 0.13 <sup>a</sup>	2.31 ± 0.18 <sup>b</sup>	2.16 ± 0.16 <sup>b</sup>
Thickness of the mucous layer	0.54 ± 0.10 <sup>a</sup>	0.41 ± 0.09 <sup>b</sup>	0.31 ± 0.09 <sup>b</sup>
Thickness of the submucosal layer	1.49 ± 0.16 <sup>a</sup>	1.41 ± 0.15 <sup>a</sup>	1.39 ± 0.15 <sup>a</sup>
Thickness of the muscular layer	0.51 ± 0.07 <sup>a</sup>	0.49 ± 0.09 <sup>a</sup>	0.46 ± 0.08 <sup>a</sup>

Note: see Table 1.

**Table 3**

Thickness parameters of the wall segment and individual layers of the stomach and small intestine in dogs weighing 15–30 kg with either toxocarosis monoinfection or toxocarosis-cystoisosporiasis co-infection ( $x \pm SD$ ,  $n = 6$ )

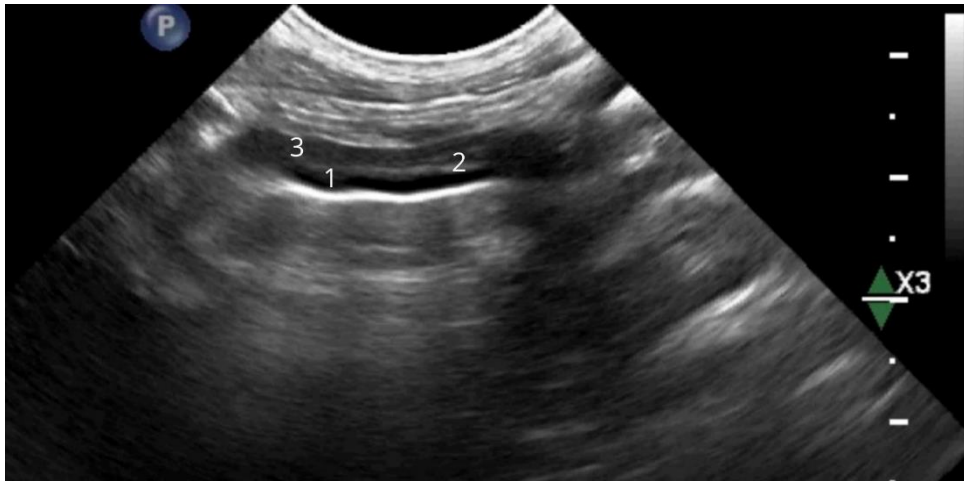
Parameters of segments of gastrointestinal tract, mm	Clinically healthy dogs	Dogs with <i>T. canis</i> infection	Dogs with <i>T. canis</i> and <i>C. canis</i> co-infection
<b>Stomach</b>			
Thickness of the wall	4.12 ± 0.12 <sup>a</sup>	4.85 ± 0.41 <sup>b</sup>	5.47 ± 0.27 <sup>c</sup>
Thickness of the mucous layer	2.45 ± 0.10 <sup>a</sup>	2.70 ± 0.32 <sup>a</sup>	3.13 ± 0.27 <sup>b</sup>
Thickness of the submucosal layer	0.72 ± 0.08 <sup>a</sup>	1.05 ± 0.14 <sup>b</sup>	1.08 ± 0.16 <sup>b</sup>
Thickness of the muscular layer	0.95 ± 0.14 <sup>a</sup>	1.10 ± 0.09 <sup>ab</sup>	1.25 ± 0.24 <sup>b</sup>
<b>Duodenum</b>			
Thickness of the wall	4.28 ± 0.21 <sup>a</sup>	4.95 ± 0.12 <sup>b</sup>	5.23 ± 0.29 <sup>b</sup>
Thickness of the mucous layer	2.95 ± 0.19 <sup>a</sup>	3.23 ± 0.14 <sup>b</sup>	3.38 ± 0.21 <sup>b</sup>
Thickness of the submucosal layer	0.63 ± 0.18 <sup>a</sup>	0.83 ± 0.10 <sup>ab</sup>	0.92 ± 0.16 <sup>b</sup>
Thickness of the muscular layer	0.70 ± 0.09 <sup>a</sup>	0.88 ± 0.08 <sup>b</sup>	0.98 ± 0.08 <sup>b</sup>
<b>Jejunum</b>			
Thickness of the wall	3.82 ± 0.24 <sup>a</sup>	2.88 ± 0.19 <sup>b</sup>	2.43 ± 0.12 <sup>c</sup>
Thickness of the mucous layer	2.50 ± 0.20 <sup>a</sup>	1.72 ± 0.19 <sup>b</sup>	1.53 ± 0.15 <sup>b</sup>
Thickness of the submucosal layer	0.63 ± 0.12 <sup>a</sup>	0.55 ± 0.16 <sup>ab</sup>	0.38 ± 0.08 <sup>b</sup>
Thickness of the muscular layer	0.68 ± 0.12 <sup>a</sup>	0.62 ± 0.10 <sup>ab</sup>	0.52 ± 0.10 <sup>b</sup>
<b>Ileum</b>			
Thickness of the wall	3.28 ± 0.12 <sup>a</sup>	2.88 ± 0.18 <sup>b</sup>	2.82 ± 0.24 <sup>b</sup>
Thickness of the mucous layer	0.78 ± 0.10 <sup>a</sup>	0.52 ± 0.12 <sup>b</sup>	0.48 ± 0.10 <sup>b</sup>
Thickness of the submucosal layer	1.88 ± 0.15 <sup>a</sup>	1.77 ± 0.15 <sup>a</sup>	1.75 ± 0.19 <sup>a</sup>
Thickness of the muscular layer	0.62 ± 0.17 <sup>a</sup>	0.60 ± 0.09 <sup>a</sup>	0.58 ± 0.12 <sup>a</sup>

Note: see Table 1.

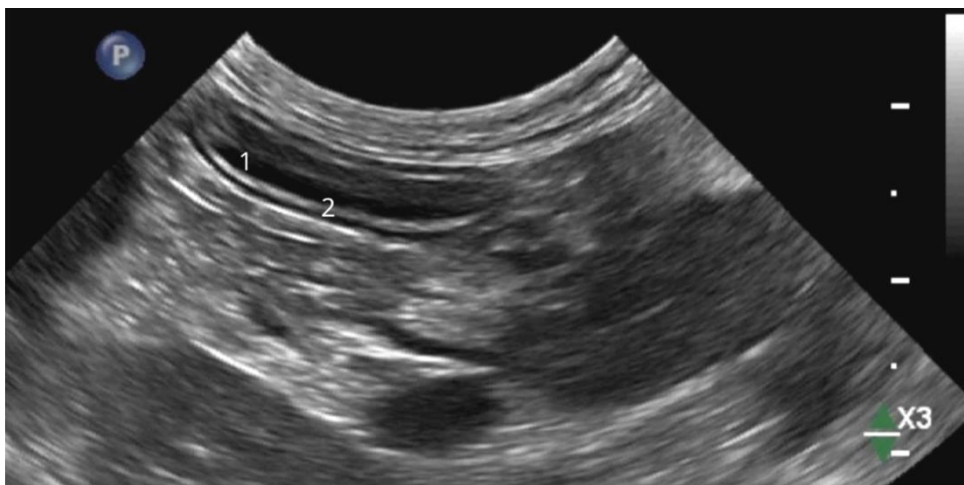
With toxocarosis alone, only the submucosal layer thickened in the stomach (by 50.0%,  $P < 0.05$ ), and with toxocarosis-cystoisosporiasis co-infection, the mucous (by 24.0%), submucosal (by 54.3%) and muscular (by 36.8%) stomach layers were thickened compared to clinically healthy animals. In the small intestine, with toxocarosis monoinfection, thickening ( $P < 0.05$ ) of the mucous and muscular layers of the duodenal wall (by 8.5% and 28.6%, respectively), as well as thinning ( $P < 0.05$ ) of the mucous layer of the jejunum (by 32.0%) and ileum (by 37.5%) walls were noted. In case of toxocarosis-cystoisosporiasis co-infection, thickening ( $P < 0.05$ ) of all layers of the duodenal wall (by 15.3–50.0%), thinning ( $P < 0.05$ ) of all layers of the jejunum wall (by 28.6–40.0%) and the mucous layer of the ileum (by 24.0%) were observed.

Thus, more significant changes were found during USG examination of dogs co-infected with *T. canis* and *C. canis*, which were characterized by a more pronounced thickening of the stomach wall and its layers (Fig. 1), as well as the duodenal wall and its layers

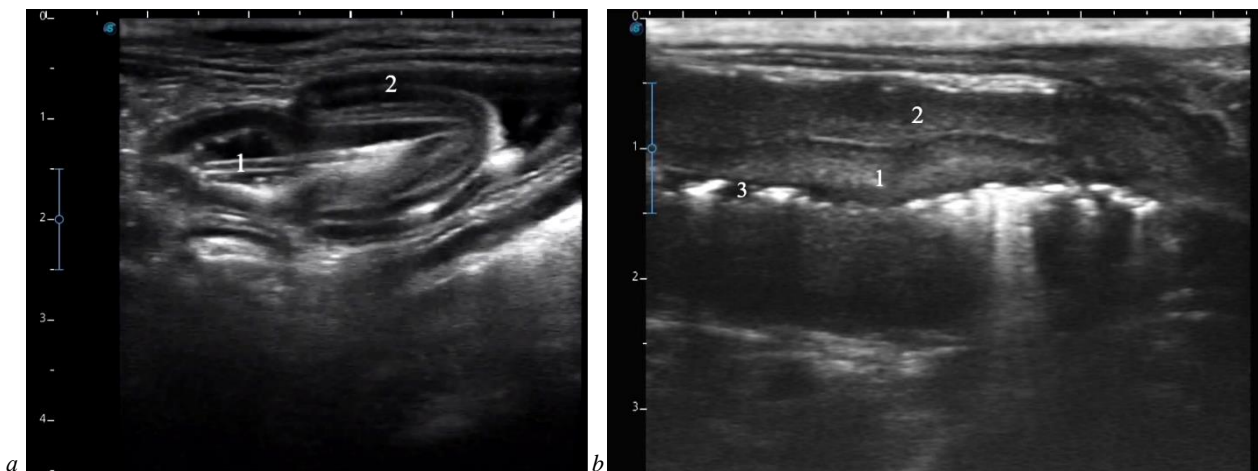
(Fig. 2). The differentiation of the layers was preserved, and the ratio of the layers was changed. The greatest changes were found in the jejunum, where, regardless of the presence of mono- or co-infection, in some cases the presence of *Toxocara* was detected in the intestinal cavity, as well as intussusception of the intestinal wall (Fig. 3a). Comparatively, with toxocarosis-cystoisosporiasis co-infection, a more pronounced thinning of the jejunum wall layers was detected, and the average amount of anechoic content in its cavity is visualized (Fig. 3b). The smallest changes, especially in dogs weighing < 5 kg with *T. canis* parasitization, were found in the ileum. In most cases, the differentiation of the intestinal wall layers was preserved, the ratio of the layers and the surrounding tissues did not change (Fig. 4). In some cases, with toxocarosis alone, a slight thinning of the ileum wall due to its mucous layer was detected in dogs weighing 5–15 and 15–30 kg. With toxocarosis-cystoisosporiasis co-infection, the ileum wall was also thinned due to thinning of the mucous layer in all dogs, regardless of weight category.



**Fig. 1.** Ultrasound image of a stomach segment during ultrasound examination of dogs co-infected with both *T. canis* and *C. canis* (layer differentiation preserved, layer ratio disturbed): thickened mucous layer (1); thickened submucosal layer (2), thickened muscular layer (3)



**Fig. 2.** Ultrasound image of a segment of the duodenum in dogs co-infected with both *T. canis* and *C. canis* (layer differentiation preserved, layer ratio disturbed): thickened mucosal layer (1); thickened submucosa (2)



**Fig. 3.** Ultrasound images of jejunum segments during ultrasound examination of infested dogs: *a* – parasitised by *T. canis* (content is anechoic, not homogeneous, a linear structure with thin hyperechoic walls is visualized); helminth in the intestinal cavity (1); intussusception of the intestinal wall (2); *b* – co-parasitised by *T. canis* and *C. canis*; thinning and increased echogenicity of the mucous layer (1), intestinal cavity, an average amount of anechoic content is visualized (2), increased echogenicity of local fat (3)

## Discussion

Ultrasound is known to be a safe, versatile, readily available, and highly informative imaging modality that is widely used to assess the gastrointestinal tract in domestic animals, including dogs. It provides information on wall thickness, differentiation, and layer thickness, characterization of the intestinal and gastric lumen contents, and assessment of their motility, which allows the assessment of the pres-

ence and nature of pathological changes (Larson & Biller, 2009; Esteves-Monteiro et al., 2026). A deep understanding of the deviations from reference values during ultrasound examination of the gastrointestinal tract in animals significantly increases the accuracy of diagnosis, taking into consideration the changes that occur in various gastrointestinal diseases. These changes, which are commonly observed in dogs, often lead to changes in echogenicity, intestinal wall thickness, and intestinal wall layers (Patsikas et al., 2003; Diana et al., 2009).



**Fig. 4.** Ultrasound image of an ileum segment in dogs infected with *T. canis* (layer differentiation preserved, layer ratio unchanged, surrounding tissues unchanged): mucosal layer (1); submucosa (2); muscular layer (3)

Despite its diagnostic and informative significance, ultrasound examination in dogs with gastrointestinal parasitosis has been covered only in individual publications (Venco et al., 2005; Lemetayer et al., 2016; Corda et al., 2019). Therefore, we conducted an ultrasound examination of the stomach and small intestine in cases of toxocarosis infection and toxocarosis-cystoisosporiasis co-infection in dogs. It was found that both with parasitization of nematodes *T. canis* and the association of *T. canis* and *C. canis*, changes occurred in the thickness of the walls and layers of the stomach and small intestine sections. Moreover, with toxocarosis-cystoisosporiasis infection, more significant changes were found than with toxocarosis alone. In particular, with toxocarosis monoinfection in dogs of varying weight, the changes were characterized by thickening of the stomach wall (by 15.4–46.4%). Also in dogs weighing 5–15 and 15–30 kg, the duodenal wall was thickened (by 10.3% and 15.1%). In all experimental dogs, thinning of the jejunum wall was recorded (by 18.2–32.1%), as well as of the ileum in dogs weighing 5–15 and 15–30 kg (by 8.0% and 12.1%). When analyzing the metric indicators of the thickness of the layers of the stomach wall and small intestine, it was found that with toxocarosis alone, the stomach submucosal layer was thickened (by 42.9–50.0%) in all experimental dogs and the muscular layer (by 47.1%) in dogs < 5 kg. Thinning of the mucous layer of the jejunum wall was detected in all experimental dogs (by 18.5–35.0%), and thinning of the muscular layer (by 40.0%) was found in dogs < 5 kg. Thickening of the mucous layer of the duodenal wall was detected in dogs weighing 5–15 and 15–30 kg (by 11.1% and 8.5%, respectively).

In case of toxocarosis-cystoisosporiasis co-infection, regardless of the weight category of dogs, the changes were characterized by thickening of the stomach wall (by 33.3–67.9%), duodenum (by 20.9–26.5%) and thinning of the jejunum (by 24.2–39.3%) and ileum (by 8.2–15.2%) walls. Also, in all experimental dogs, simultaneous thickening of the mucous, submucosal and muscular layers of the stomach wall (by 13.6–80.0%), duodenum (by 12.6–75.0%), as well as thinning of these layers in the jejunum wall (by 20.0–45.0%) were recorded. Thinning of the ileum wall occurred due to the thinning of the mucous layer (by 24.0–40.0%). Scientists note that most gastrointestinal pathologies alter the thickness and integrity of the intestinal wall layers (serosal, muscular, submucosa, and mucosa), which is a crucial aspect of their assessment, as it provides valuable clinically relevant information (Penninck et al., 2003; Gaschen, 2011; Winter et al., 2014). There are also reports that pathological conditions such as inflammation, chronic enteropathies, neoplastic disorders, ulcers, and ruptures can affect the normal wall thickness of the gastrointestinal organs (Manczur & Vörös, 2000).

According to our studies, the greatest changes were found in the jejunum. All its layers were significantly thinner, which, in our opinion, is due to the most frequent localization of *Toxocara* in this department, which we detected during ultrasound examination, and also due to the pressure of nematodes on the intestinal wall, as well as the occurrence of chronic inflammation and the accumulation of contents

and exudate in the lumen of the intestine, which further leads to thinning of the wall as a whole and all its layers. Thinning of the mucous membrane of the jejunum in parvovirus enteritis according to the results of ultrasound examination is evidenced by researchers who note that such thinning occurs as a result of villi detachment and the inflammatory process (Stander et al., 2010). There is a report of detecting *T. canis* nematode in the small intestine in puppies by ultrasonography (Corda et al., 2019). Other researchers have noted that in dogs infected with *T. canis*, a significant decrease in the height of the villi was observed, which was directly proportional to the degree of infection (Lloyd et al., 1991). The thickening of the wall of the stomach and duodenum that we found indicates their edema and the development of an inflammatory process without stretching their walls with the contents. Some scientists have found by ultrasound that gastritis increases the size of the stomach wall (Mălăncuș et al., 2010).

Our data confirm the feasibility of using ultrasound in the assessment of structural and functional changes in the gastrointestinal tract of dogs with mono- and co-infections.

## Conclusion

Ultrasound examinations of dogs with mono- and co-infections caused by parasitization of nematodes *T. canis* and protozoa *C. canis* revealed changes in the thickness of the walls and their layers of the stomach and small intestine, with more significant changes observed in dogs infected with the association of *T. canis* and *C. canis*. The most frequent changes in dogs with toxocarosis monoinfection, regardless of their weight category, were thickening of the stomach wall (up to 46.4%) and its submucosal layer (up to 60.0%), thinning of the jejunum wall (up to 32.1%) and its mucous layer (up to 35.0%). In dogs weighing from 5 to 30 kg, thickening of the duodenal wall (up to 15.1%) and its mucous layer (up to 11.1%), thinning of the ileum wall (up to 12.1%) and its mucous layer (up to 37.5%) were also detected. In dogs with toxocarosis and cystoisosporiasis, regardless of their weight category, thickening of the stomach and duodenal wall (up to 67.9% and 26.5%, respectively) and thinning of the jejunum and ileum walls (up to 39.3% and 15.2%, respectively) were observed. Such changes occurred due to thickening of the mucous (up to 26.1% and 15.3%, respectively), submucosal (up to 80.0% and 75.0%) and muscular (up to 71.4% and 50.0%) layers in the stomach and duodenum walls, as well as thinning of the mucous (up to 45.0%), submucosal (up to 40.0%) and muscular (up to 40.0%) layers in the jejunum wall, and the mucous layer alone (up to 40.0%) in the ileum wall.

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The authors state that there is no conflict of interest.

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