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False-Positive *Ascaris suum* Egg Counts in Pigs

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Abstract—**Boes J., Nansen P. & Stephenson L. S. 1997. False-positive *Ascaris suum* egg counts in pigs. *International Journal for Parasitology* 27: 833–838. False-positive *Ascaris suum* egg counts in pig faeces are frequently observed under both experimental and natural conditions. Data from 12 experiments with *A. suum* infections in pigs were summarized and showed that the percentage of false-positive faecal samples ranged from 4 to 36%. False-positive egg count values varied greatly between pigs and experiments (range 20–1060 eggs per gram faeces). Indoor experiments with pigs housed groupwise in pens generally produced more and higher false-positive egg counts, which may reflect differences in surface area and hence exposure to infective eggs, compared with pasture experiments. The positive predictive value (the number of pigs diagnosed positive by faecal sample that actually harboured worms) was low for indoor experiments (45%) compared with pasture experiments (89%). Differences in design for indoor experiments, such as floor type and use of bedding material, did not influence the positive predictive value (44–47%). A positive correlation was found ($r = 0.56$, $P < 0.05$) between faecal egg counts of true-positive and false-positive pigs that were penned together. The results of this survey strongly support previous suggestions that false-positive *A. suum* egg counts in pigs are the result of coprophagia in indoor experiments and coprophagia/geophagia in pasture experiments. False-positive *A. suum* egg counts in pig faeces may vary greatly in prevalence and magnitude, and depend in part on management/housing factors. © 1997 Australian Society for Parasitology. Published by Elsevier Science Ltd.**

Key words: Pigs; false-positive egg counts; coprophagia; geophagia; *Ascaris suum*.

INTRODUCTION

The presence of intestinal helminths is commonly demonstrated through detection of eggs in faeces, often expressed as number of eggs per gram of faeces (EPG). However, it can be difficult to relate an indirect measure like EPG to parasite numbers (Anderson, 1993). Several authors have reported a discrepancy between the number of pigs whose faeces proved to be positive for *Ascaris suum* eggs and the number from which adult worms were actually recovered (Bindseil, 1974; Bernardo *et al.*, 1990; Eriksen *et al.*, 1992; Bøgh *et al.*, 1994). Bindseil (1974) found that of 69 slaughtered pigs, 28% had false-positive *A. suum* egg counts (range 30–1020 EPG). Bøgh *et al.* (1994) reported that 6 of 26 pigs that excreted *A. suum* eggs at slaughter were false-positive (range 20–80 EPG).

In experiments with *A. suum* in pigs at our centre, false-positive egg counts have been observed in several studies with different designs with regard to type of infection, housing, group size, etc. The occurrence of false-positive egg counts is an important and neglected observation, complicating especially prevalence studies, as these are usually based on faecal egg counts. Roepstorff (in press), using coprological surveillance to establish the helminth status of commercial pig herds, suggested an arbitrary cut-off value for *A. suum* egg counts, where an EPG $\leqslant 200$ should be regarded as false-positive.

The objective of this paper is to report and discuss the occurrence of false-positive *A. suum* egg counts in pigs and the potential influence of coprophagia, by summarizing and analysing data collected at our

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centre since 1992. In contrast to slaughterhouse surveys, the studies included here provide more detailed information and all have employed standard methods and techniques (e.g., a modified McMaster method for faecal egg counts). Experimental design, type of infection, housing conditions and group size will be compared and the parasitological results discussed.

MATERIALS AND METHODS

Each author who had conducted experiments with *A. suum* infections in pigs at our centre since 1992 filled out a questionnaire that included detailed parasitological data (EPG and worm counts for all experimental animals), and precise descriptions of the experimental set-up. This included type of infection (experimental inoculation or natural infection from contaminated pasture), housing (indoor, groupwise in pens or outdoor on pasture), floor type (solid vs slatted), use of bedding material, indoor group size and total number of experimental animals.

In all 12 experiments included in this survey standardized procedures were used for faecal sample analysis and post mortem worm recovery. The presence of *A. suum* eggs in pig faeces was determined via a modified McMaster method with a sensitivity of 20 EPG (Roepstorff & Nansen, in press). Worms were collected manually from faeces after anthelmintic treatment, or from the small intestine at necropsy. All egg counts and worm burdens included were measured simultaneously either at time of necropsy or after anthelmintic treatment. Overdispersed distributions (egg counts) were transformed to natural logarithms before calculating the Pearson correlation coefficient.

The number of pigs that excreted *A. suum* eggs in the faeces and the number of pigs that harboured intestinal ascarids at time of necropsy or treatment were recorded for each experiment. The number and percentage of pigs with positive faecal egg counts but no worms in the intestine at necropsy or treatment were calculated. Also, the range of the false-positive egg count levels was included. Subsequently, 4 diagnostic categories were defined to describe the reliability of faecal sample analysis as a measure of adult *A. suum* infection in pigs. These were the true-positives (% of *A. suum*-positive pigs that also had positive egg counts), the false-negatives (% of *A. suum*-positive pigs that had negative egg counts), the true-negatives (% of *A. suum*-negative pigs that also had negative egg counts), and the false-positives (% of *A. suum*-negative pigs that had positive egg counts). A positive predictive value was calculated, which expressed the number of pigs diagnosed positive by faecal sample that actually harboured worms. Consequently, a low positive predictive value indicated a high number of false-positive pigs.

RESULTS

When the histories of infection from all 12 studies included in this survey were taken into account, it appeared that false-positive egg counts occurred more often in the course of infection than was expected from treatment or slaughter data alone. To illustrate this, data are reproduced in Table 1 from experimental groups of pigs taken from 2 indoor experiments (Table 1A, B) and 1 pasture experiment (Table 1C). Table 1A shows that low egg counts in indoor pigs ceased when worm-positive penmates were necropsied. Table 1B illustrates that false-positive egg counts in indoor experiments can be quite high, depending on the number of eggs excreted by true-positive penmates. In experiments on pasture contaminated with *A. suum* eggs, false-positive egg counts were found in previously uninfected pigs before infections could have reached patency (Table 1C). However, the results of the analyses listed below are based on data obtained at time of treatment or necropsy.

The data were obtained from 8 studies with experimental and 4 with natural *A. suum* infections, in a total of 437 pigs, and are summarized in Table 2. Six experiments had been conducted indoors, including 220 pigs; the other 6 were pasture experiments including 164 pigs. As pigs kept indoors were housed in pens, the average surface area per pig was limited compared with pasture experiments (Table 2). In the 12 experiments included, 29–76% of all pigs had positive *A. suum* egg counts. The percentage of pigs with positive egg counts but no intestinal worms ranged from 4 to 36%. False-positive egg counts ranged from 20 to 1060 EPG. There was no morphological difference between eggs excreted by true- and false-positive pigs.

The percentage of *A. suum* false-positive pigs and the range of their egg count levels (expressed as EPG) were greater in the indoor experiments, which had relatively small surface areas per pig. To investigate whether the magnitude of the false-positive egg counts in a given pen depended upon the amount of eggs excreted by penmates harbouring worms (cf. Table 1B), a correlation between true-positive and false-positive EPG was calculated for the indoor experiments. Thirteen pens that contained both wormy and false-positive pigs were selected. There was a significant association between the mean EPG of the true-positive and false-positive pigs (Pearson correlation coefficient $r = 0.56$, $P < 0.05$). No correlation could be computed for natural infections, because pasture contamination would act as a major confounder.

Table 3 shows the overall association between *A. suum* worm burden and faecal egg counts in pigs from all 12 experiments. The sensitivity, calculated as the % true-positives, and the specificity, calculated as the % true-negatives, were both low, 77% and 77%, respectively. However, the association between worm burden and egg count was highly significant (χ^2 $P < 0.0001$). The overall positive predictive value was 69% (Table 4), which meant that nearly one-third (31%) of the pigs that were diagnosed as *A. suum*-

Table 1—The occurrence of false-positive *A. suum* egg counts in pigs (possible false-positive faecal egg counts in bold type). (A1–A3) Low *A. suum* egg counts ceased when worm-positive pigs were necropsied. (B) The influence of true-positive egg counts on the magnitude of false-positive *A. suum* egg counts. (C) False-positive *A. suum* egg counts were detected in previously uninfected pigs introduced to infected pasture, before infections reached patency

Pen	Weeks after first inoculation 0–6	7	8	9	10	11	12	13	14	16	Worm burden
A1[a]	0	1760	1480	9040	13 120[d]						51
	0	0	**40**	**40**	**40**	0[d]					0
	0	0	**40**	**20**	**100**	0[d]					0
	0	0	**140**	**40**	**180**	0	0	0[d]			0
A2	0	0	0	0	**20**	0[d]					0
	0	0	**20**	**40**	0	0	**20**[d]				1
	0	**40**	**20**	**20**	0	**20**	**60**	**100**	**60**	**20**[d]	0
	0	3400	5080	7280	8760	9760	12 500	8480	12 960	9400[d]	20
A3	0	0	600	1760	3920[d]						28
	0	80	740	1120	1220[d]						21
	0	0	**660**	**20**	**140**	0	0[d]				0
	0	0	**60**	**20**	**40**	0	0	0	0[d]		0
B[b]	0	120	920	4680	13 080	31 440	32 800[d]				95
	0	0	720	1680	5640	6480	8280[d]				31
	0	0	0	0	20	60	280[d]				5
	0	0	**40**	0	**80**	**240**	**120**[d]				0
	0	0	0	0	**220**	**260**	**240**[d]				0
	0	0	0	0	0	**220**	**280**[d]				0
	0	0	0	0	**120**	**80**	**80**[d]				0
	0	0	0	0	0	**180**	**640**[d]				0
	Weeks after turn-out on pasture 0	2	4	6	8	10					Worm burden
C[c]	0	**20**	0	160	4220	8880[e]					77
	0	0	0	1180	2060	8480[e]					60
	0	0	**20**	560	1820	2960[e]					60
	0	**20**	**20**	20	2680	3500[e]					31
	0	**40**	0	0	520	2260[e]					6
	0	0	0	0	120	300[e]					1
	0	0	**20**	20	20	0[e]					1
	0	0	0	0	0	**20**[e]					0
	0	**20**	0	0	0	**20**[e]					0
	0	0	0	0	**20**	0[e]					0
	0	**20**	0	**20**	0	0[e]					0
	0	0	0	**20**	0	0[e]					0
	0	0	0	0	0	0[e]					0
	0	0	0	0	0	0[e]					0
	0	0	0	0	0	0[e]					0

[a]Reproduced with permission from Eriksen *et al.* (1992); [b]reproduced with permission (Petkevičius *et al.*, 1995); [c]partial results (Boes, in preparation).
[d]In the pig necropsied.
[e]At time of anthelmintic treatment.

positive by egg counts harboured no worms. However, there was considerable variation between experiments; the percentage of false-positive pigs ranged from 5 to 69%. The results of the analysis of the different experimental set-ups are listed in Table 4 and show that indoor experiments generally resulted in more false-positive egg counts (55%), presumably due to higher concentrations of eggs, compared with pasture experiments (11%). The positive predictive value was remarkably consistent (44–47%), regardless of differences in floor type and use of bedding material (Table 4). Note that the positive predictive value is normally a declining function of prevalence; the fact that it does not vary with changing prevalence in the indoor studies and that this relationship is reversed when indoor and pasture experiments are compared (see Table 4), is also evidence that the percentage of false-positives and true-positives are linked.

Table 2—Experimental design and parasitological results in various experiments with *Ascaris suum* infections in pigs

Type of *A. suum* infection	Housing	Floor type	Bedding	Total pigs used	No. pigs per pen	Area per pig (m^2)	Pigs with positive EPG (%)	Pigs with false-positive EPG (%)	Range false-positive EPG (min–max)	Reference
Experimental	Indoor pens	Solid	Straw	40	4	2.5	13 (33)	4 (10)	20–160	Eriksen *et al.* (1992)
Experimental[a]	Indoor pens	Slatted	Straw	36	9	1.1	19 (53)	13 (36)	20–360	Eriksen (unpublished)
Experimental	Indoor pens	Solid	—	32	8	1.3	18 (56)	11 (34)	20–640	Petkevičius *et al.* (1995)
Experimental	Indoor pens	Solid	—	50	10	1.0	16 (32)	11 (22)	20–1060	Petkevičius *et al.* (1997)
Experimental	Indoor pens	Solid	—	28	7	1.4	12 (43)	7 (25)	60–720	Petkevičius (in preparation)
Experimental	Indoor pens	Slatted	—	34	5	2.0	11 (29)	3 (8)	20–740	Roepstorff (in preparation)
Experimental	Pasture	—	Grass	38	—	80	20 (53)	2 (5)	20–40	Boes (in preparation)
Experimental	Pasture	—	Grass	28	—	100	12 (43)	2 (7)	20–1000	Roepstorff & Murrell (1997)
Natural	Pasture	—	Grass	28	—	100	14 (50)	1 (4)	20–20	Roepstorff & Murrell (1997)
Natural[a]	Pasture	—	Grass	50	—	80	15 (30)	3 (6)	20–20	Boes (in preparation)
Natural	Pasture	—	Grass	49	—	80	37 (76)	2 (4)	20–20	Boes (in preparation)
Natural	Pasture	—	Grass	20	—	200	8 (40)	2 (10)	20–240	Petkevičius *et al.* (1996)

[a]Individual worm burdens measured after expulsion following anthelmintic treatment.

Table 3—Association between *A. suum* worm burden and faecal egg counts in experimental pigs

A. suum worm count	Faecal egg counts No. (%) pigs	% negative	% positive
Negative	263 (60)	77	23
Positive	174 (40)	23	77
χ^2 value	120.57[a]		

[a]Significant, $P < 0.0001$.

DISCUSSION

False-positive *A. suum* egg counts in pigs have apparently received little attention in the literature to date. They are, however, an important observation, which can seriously complicate the interpretation of the results of prevalence studies, which usually rely on faecal egg counts. Furthermore, false-positive egg counts could influence decision making on whether or not to perform anthelmintic treatment, as the prevalence of infection will be overestimated. The objective of this survey was to investigate the occurrence of false-positive *A. suum* egg counts in pigs and establish if the available data lend support to the assumption that they are the result of coprophagia.

The results of our survey show that the occurrence of false-positive *A. suum* egg counts in pigs is associated with factors of experimental design, of which housing (indoor or pasture experiments) was the most important factor. Differences in design for indoor experiments, such as floor type and use of bedding material, did not seem to influence the occurrence of false-positive egg counts. The proportion of false-positive pigs and the range of false-positive EPG can vary considerably within and between experiments. False-positive egg counts in indoor experiments were generally higher than in pasture experiments, which may reflect the differences in surface area per pig and thus exposure to faeces containing eggs. However, from the results of this survey it was not possible to define a general cut-off value for *A. suum* EPG, below which coprophagia was presumed to account for all eggs in the faecal sample analysis.

The data included in our survey are egg counts and worm burdens measured simultaneously, either at slaughter or after anthelmintic treatment. This provides a safer method to measure the infection status of the animals compared with faecal egg counts alone. However, low egg counts were recorded once or more frequently during the course of infection in pigs that had no egg count at treatment or slaughter. This finding, first reported by Eriksen *et al.* (1992) (Table 1A), was observed in all other experiments included in this survey. Furthermore, previously uninfected pigs that are introduced to infected pasture excrete low num-

Table 4—Influence of management/housing factors on the association between worm burden and egg counts, and the positive predictive value of faecal sample analysis, in *A. suum* infections in pigs

Analysis	*N*	True positives (%) worms, EPG	False negatives (%) worms, no EPG	True negatives (%) no worms, no EPG	False positives (%) no worms, EPG	Positive predictive value
All experiments	437	77	23	77	23	69
(min–max)		(60–100)	(0–40)	(56–90)	(10–44)	(31–95)
Pasture	213	74	26	86	14	89
Indoor	224	85	15	72	28	45
Solid floor	150	93	7	73	27	44
Slatted floor	74	74	26	71	29	47
Bedding	76	94	6	72	28	47
No bedding	148	81	19	73	27	44

bers of *A. suum* eggs before infections reach patency, i.e. within 6 weeks (Table 1C). Low *A. suum* egg counts (20–40 EPG) have been observed in parasite-naive pigs as early as 3 days after turnout on infected pasture (Boes, unpublished results).

Another interesting observation was that false-positive egg counts were not necessarily seen in every pig that was housed together with one or more worm-positive, egg excreting pigs. This could suggest that there are differences in behaviour, as a result of which certain pigs are not coprophagous. However, weekly or biweekly faecal egg counts only reflect a point in time; thus it seems more likely that false-positive egg counts in these pigs are simply not detected. Furthermore, the available surface area/pig and distribution of eggs could influence both prevalence and magnitude of false-positive egg counts. And finally, the EPG in false-positive pigs correlated positively with the EPG in true-positive pigs. None the less, individual differences could exist in the extent to which pigs practise coprophagia.

Eriksen *et al.* (1992) suggested that *A. suum* egg counts in worm-free pigs may be a result of coprophagia. They observed that (1) such pigs generally showed much lower egg counts than did pigs which harboured worms, (2) they aggregated in pens containing a wormy pig and (3) when the worm-positive pigs had been slaughtered, egg excretion ceased in the remaining pigs (cf. Table 1A). Muff *et al.* (1984) previously suggested that faecal egg counts may give positive findings in pigs that do not bear patent infections if the animals are kept groupwise in pens. Our survey confirmed these observations and strongly suggested that false-positive *A. suum* egg counts in pigs in indoor experiments are the result of coprophagia. In pasture experiments the influence of geophagia, which is normal behaviour in rooting pigs under natural circumstances, cannot be excluded. As soil on pasture is often mixed with faeces, geophagia and coprophagia may both occur. Absence of soil is likely to induce coprophagia in indoor pigs.

Coprophagia is performed by rodents and lagomorphs, and to a lesser degree by piglets, foals, dogs and nonhuman primates and is defined as feeding or eating of dung or excrement that is considered normal behaviour among many animals (Schulze & Haenel, 1969; Soave & Brand, 1991). Coprophagia is also of nutritional significance because it provides a source of vitamins, minerals, amino acids, trace elements and other nutrients that are excreted with the faeces and were not completely utilized in the gastrointestinal tract (Soave & Brand, 1991). Sansom & Gleed (1981) studied coprophagia by newborn piglets and administration of iron to sows as a means of preventing anaemia in piglets. They found that piglets born to sows housed on solid-floored farrowing pens on average ingested 20 g of faeces and bedding daily. In farrowing pens with slatted floors without bedding the average daily intake of faeces by piglets was only 8.5 g (Gleed & Sansom, 1982). Evidence of coprophagia as a source of *Isospora suis* oocysts in pigs has been reported previously (Joyner *et al.*, 1981).

The results of this survey demonstrate that the use of faecal egg counts to determine infection with *A. suum*, and probably other soil transmitted helminths, should be very critically evaluated. Ingestion of faeces or soil that is contaminated with *Ascaris* eggs by pigs and other animals, and in the case of geophagia also by humans, will complicate prevalence studies estimating ascarosis in humans (cf. Guyatt & Bundy, 1993) or pigs (cf. Roepstorff & Jorsal, 1989). A study of children in Jamaica found that geophagia may be closely related to intensity of geohelminth infection in humans (Wong *et al.*, 1988, 1991). A cut-off value for *A. suum* egg counts in pigs as suggested for prevalence studies by Roepstorff (in press), should be considered cautiously under experimental circumstances, because of the overlap with egg counts that are just commencing or egg counts from infections with only 1 male and 1 female *Ascaris* worm. Furthermore, false-positive egg counts can be quite high in indoor experiments (cf. Table 1B). In any case, repeated faecal

sampling seems indicated when egg counts are very low.

In conclusion, the results of this survey support the suggestion that false-positive *A. suum* egg counts in pigs can occur, often due to coprophagia in indoor experiments and coprophagia/geophagia in pasture experiments. The number and range of false-positive *A. suum* egg counts in pigs can be considerable, but vary greatly and depend in part on management/housing. Prevalence studies that reveal many low *A. suum* egg counts in pigs should be interpreted with care.

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REFERENCES

Anderson R. M. 1993. Epidemiology. In: *Modern Parasitology* (Edited by Cox F. E. G.), pp. 75–116. Blackwell Scientific, Oxford.

Bernardo Th. M., Dohoo I. R., Donald A., Ogilvie T. & Cawthorn R. 1990. Ascariasis, respiratory diseases and production indices in selected Prince Edward Island swine herds. *Canadian Journal of Veterinary Research* **54:** 267–273.

Bindseil E. 1974. Observations on the relationship between *Ascaris suum* burdens in pigs and faecal egg counts. *Acta Pathologica et Microbiologica Scandinavica Section B* **82:** 879–884.

Bøgh H. O., Eriksen L., Lawson L. G. & Lind P. 1994. Evaluation of an enzyme-linked immunosorbent assay and a histamine release test system for the detection of pigs naturally infected with *Ascaris suum*. *Preventive Veterinary Medicine* **21:** 201–214.

Eriksen L., Nansen P., Roepstorff A., Lind P. & Nilsson O. 1992. Response to repeated inoculations with *Ascaris suum* eggs in pigs during the fattening period. *Parasitology Research* **78:** 241–246.

Gleed P. T. & Sansom B. F. 1982. Ingestion of iron in sow's faeces by piglets reared in farrowing crates with slotted floors. *British Journal of Nutrition* **47:** 113–117.

Guyatt H. L. & Bundy D. A. P. 1993. Estimation of intestinal nematode prevalence: influence of parasite mating patterns. *Parasitology* **107:** 99–106.

Joyner L. P., Gregory M. W., Norton C. C., Done J. T. & Wells G. W. H. 1981. Coccidiosis and coprophagy in pigs. *The Veterinary Record* **108:** 264–265.

Muff F., Koch W. & Wolff K. 1984. Zur Epizootologie des Askaridenbefalles beim Schwein. *Schweizer Archive für Tierheilkunde* **126:** 409–428.

Petkevičius S., Bach Knudsen K. E., Nansen P. & Roepstorff A. 1996. The influence of diet on infections with *Ascaris suum* and *Oesophagostomum dentatum* in pigs on pasture. *Helminthologia* **33:** 173–180.

Petkevičius S., Bach Knudsen K. E., Nansen P., Roepstorff A., Skjøth F. & Jensen K. 1997. The impact of diets varying in carbohydrates resistant to endogeneous enzymes and lignin on populations of *Ascaris suum* and *Oesophagostomum dentatum* in pigs. *Parasitology* **114:** 555–568.

Petkevičius S., Bjørn H., Roepstorff A., Nansen P., Bach Knudsen K. E., Barnes E. H. & Jensen K. 1995. The effect of two types of diet on populations of *Ascaris suum* and *Oesophagostomum dentatum* in experimentally infected pigs. *Parasitology* **111:** 395–402.

Roepstorff A. Helminth surveillance as a prerequisite for anthelmintic treatment in intensive sow herds. *Veterinary Parasitology*. In press.

Roepstorff A. & Jorsal S. E. 1989. Prevalence of helminth infections in swine in Denmark. *Veterinary Parasitology* **33:** 231–239.

Roepstorff A. & Murrell K. D. 1997. Transmission dynamics of helminth parasites of pigs on continuous pasture: *Ascaris suum* and *Trichuris suis*. *International Journal for Parasitology* **27:** 563–572.

Roepstorff A. & Nansen P. *The Epidemiology, Diagnosis and Control of Helminth Parasites in Swine. A Methodological Guideline*. FAO, Rome. In press.

Sansom B. F. & Gleed P. T. 1981. The ingestion of sow's faeces by suckling piglets. *British Journal of Nutrition* **46:** 451–456.

Schulze J. & Haenel H. 1969. Beziehungen zwischen Koprophagie, Darmflora und Vitaminen bei Versuchstieren. *Zeitschrift für Versuchstierkunde* **11:** 190–206.

Soave O. & Brand C. D. 1991. Coprophagy in animals: a review. *The Cornell Veterinarian* **81:** 357–364.

Wong M. S., Bundy D. A. P. & Golden M. H. N. 1988. Quantitative assessment of geophagous behaviour as a potential source of exposure to geohelminth infection. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **82:** 621–625.

Wong M. S., Bundy D. A. P. & Golden M. H. N. 1991. The rate of ingestion of *Ascaris lumbricoides* and *Trichuris trichiura* eggs in soil and its relationship to infection in two children's homes in Jamaica. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **85:** 89–91.