EQUINE

Effects of pulmonary abscesses on racing performance of horses treated at referral veterinary medical teaching hospitals: 45 cases (1985–1997)

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Objective—To determine whether results of physical or radiographic examination or biochemical analyses in adult racehorses with primary lung abscesses were associated with ability to race following treatment.

Design—Multiple-center retrospective study.

Animals—25 Standardbreds and 20 Thoroughbreds.

Procedure—Medical records of horses with a primary lung abscess that were admitted to any of 4 veterinary teaching hospitals were reviewed. Results of physical examination, laboratory testing, and thoracic radiography were reviewed. Racing performance after treatment was compared with performance before illness and with performance of the general population of racehorses of similar age, sex, and breed.

Results—23 of 25 Standardbreds and 13 of 20 Thoroughbreds raced after diagnosis and treatment of a lung abscess. Most horses had a solitary abscess in the dorsal to caudodorsal lung fields. Results of initial physical examination, biochemical analyses, and culture and identification of the microbial isolate were not associated with whether a horse returned to racing. For horses that had raced prior to the illness, race performance after treatment of the lung abscess was not significantly different from performance before the illness.

Conclusions and Clinical Relevance—On the basis of racing performance in those horses that resumed racing after treatment, long-term residual lung damage did not develop in horses with primary lung abscesses that were treated appropriately. It is not known whether horses that recovered would be more likely to bleed from the site of a prior infection when resuming strenuous exercise and whether lung abscesses contributed to a failure to resume racing. (*J Am Vet Med Assoc* 2000;216:1282–1287)

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Athletic horses subjected to intense exercise are considered at risk for the development of infectious lung disorders such as pneumonia, pleuropneumonia, and primary lung abscesses.[1,2] Until recently, the repercussions of infectious pulmonary disorders on long-term athletic success were unclear. In a report in 1994,[3] 4 of 7 adult Standardbred horses with primary lung abscesses subsequently raced. Similarly, other investigators examined the outcome of 51 athletic horses with pneumonia, lung abscesses, or pleuritis and found that 3 horses with primary lung abscesses recovered and resumed athletic function.[4] These reports suggest that residual lung damage was not a sequela (although only a few horses were monitored after the recuperative period, precluding statistical analysis). Clearly, a larger study population needed to be examined.

In the multiple-center retrospective study reported here, we sought to determine the percentage of surviving horses that resumed racing, the physical examination and laboratory findings predictive of resumption of racing, the influence of the bacterial isolate on resumption of athletic performance, the most common location of pulmonary abscesses, and the effects of the pulmonary disorder on subsequent athletic performance. We hypothesized that adult racehorses, when treated appropriately for pulmonary abscesses, would not develop long-term residual lung damage that would negatively impact on subsequent athletic ability.

Criteria for Selection of Cases

Medical records of all Thoroughbred and Standardbred racehorses with primary pulmonary abscesses that were treated at any of 4 veterinary medical teaching hospitals (Cornell University, University of Guelph, University of Pennsylvania, University of Montreal) between 1985 and 1997 were reviewed. Horses that had clinical signs of pneumonia, radiographic or ultrasonographic evidence of pulmonary abscesses, and findings supportive of a pulmonary infection during cytologic examination of a transtracheal aspirate were included in the study. Horses with pleuropneumonia and horses that developed pulmonary abscesses secondary to aspiration of feed materials or medicants (eg, attributable to pharyngeal neuropathies or medicating accidents) or secondary to thoracic trauma or neoplasia were excluded. In addition, horses that were euthanized or died were not included in the study.

Information examined for horses that met the criteria for inclusion in the study included age, duration of illness, rectal temperature, heart rate, respiratory rate, WBC count, counts of segmented neutrophils, lymphocytes, and monocytes, PCV, and total protein and fibrinogen concentrations at the time of initial examination at the teaching hospital. In addition, the number of days that each horse was hospitalized and the number of days that elapsed from initial diagnosis to the horse's first race after treatment were determined.

Procedures

Imaging studies—Thoracic radiographs and ultrasonographic images were examined by a board-certified radiologist (AEY) to determine the number and location of pulmonary abscesses. Radiographic features that were considered diagnostic of an abscess, in conjunction with clinical and clinicopathologic findings, included the following: focal, oval-shaped soft-tissue opacity, lack of air bronchograms within the soft-tissue opacity, demarcated margin relative to surrounding parenchyma, and detection of cavitation.[5]

Abscesses were assigned to 1 of 4 anatomic quadrants obtained by drawing a vertical line through the carina and a horizontal line caudally from the ventral aspect of the trachea to the ventral edge of the caudal vena cava. Thus, the 4 regions were craniodorsal, cranioventral, caudodorsal, and caudoventral.

Assessment of racing performance—Information on racing performance was obtained from databases of the Jockey Club and the United States Trotting Association. A horse was considered to have raced if it started in ≥ 1 race. The number of starts and average earnings per start for the year(s) preceding the horse's illness and for the first and subsequent years following the horse's resumption of racing were examined. In addition to recording number of starts and earnings per start, the Jockey Club calculates **starts percentile rank (SPR)** for each Thoroughbred in its database. The SPR ranges from 0 to 99 and represents a given horse's average earnings per start relative to average earnings per start of similar horses, thus controlling for effects of sex, age, and race year. For example, an SPR of 80 indicates that the average earnings per start for that horse are greater than that of 80% of similar horses. The average (or median) SPR of a population of healthy racehorses[6] is 50. An SPR can be calculated annually or for lifetime performance. The United States Trotting Association currently does not calculate this statistic, but SPR were calculated for the Standardbreds in this study, using the method previously described.[6] For Thoroughbred and Standardbred racehorses, the SPR was calculated for the year(s) preceding their illness, the year immediately following their illness after they had resumed racing, and for all years after they resumed racing.

Statistical Analyses

Comparisons were made between horses that raced after the illness and those that did not. A Wilcoxon rank sum test (2-tailed with $\alpha = 0.05$, unless otherwise specified) was used for continuous data, and a χ^2 or Fischer exact test was used for discrete data. Nonparametric analyses were used for continuous data, because examination of box and whiskers plots of many variables clearly indicated skewed distributions. For selected proportions, exact binomial 95% **confidence intervals (CI)** were calculated.[a] For comparisons of SPR before and after a lung abscess was diagnosed and treatment instituted, a Wilcoxon signed rank test (1-tailed) was used. Analyses were conducted separately for Thoroughbreds and Standardbreds, except when indicated, because substantial differences in management and racing expectations existed for the 2 breeds.

Results

Horses—Medical records and racing performances of 45 horses were examined, consisting of 20 Thoroughbreds (12 males, 8 females) and 25 Standardbreds (16 males, 9 females). Five horses (3 Standardbreds, 2 Thoroughbreds) that were euthanatized or died were excluded from the study.

Medical history, physical examination, and laboratory analyses—The initial medical history and results of physical examination and clinicopathologic analyses were examined to determine whether alterations in these variables were predictive of a resumption of subsequent athletic performance. Because 23 of 25 (92%; 95% CI, 74 to 99%) of the Standardbreds raced following treatment of their illness, insufficient power existed to detect notable differences between the horses that raced and those that did not race; nevertheless, the 23 Standardbreds that raced again following treatment were younger and had lower fibrinogen concentrations than the 2 Standardbreds that did not race again (**Table 1**). Even though only 13 of 20 (65%; 95% CI, 41 to 85%) of the Thoroughbreds eventually raced following treatment of the lung disease, notable differences were not detected in the medical history or results of physical examination and clinicopathologic analyses between the horses that raced following treatment and those that did not race (**Table 2**). Sex did not significantly influence whether Standardbreds (P = 0.54) or Thoroughbreds (P = 0.90) resumed racing; females were as likely to resume racing as males. In general, horses that resumed racing had been ill for 2 to 4 weeks prior to referral, were tachypneic at the time of admission, had a neutrophilic leukocytosis, and remained hospitalized for a period of 1 week.

Microbial isolates—Bacterial isolates were obtained from transtracheal aspirates in 14 of 20 (70%; 95% CI, 45 to 89%) Thoroughbreds and 18 of 25 (72%; 95% CI, 50 to 88%) Standardbreds. The most commonly isolated gram-positive organism was *Streptococcus equi* subspecies *zooepidemicus* (24 of 45 [53%] samples; 95% CI, 34 to 71%), and the most commonly cultured gram-negative organism was *Actinobacillus* spp (17 of 45 [38%] samples; 95% CI, 21 to 57%). Polymicrobial infections were detected in 7 of 18 (39%; 95% CI, 17 to 64%) Standardbreds and 6 of 14 (43%; 95% CI, 18 to 71%) Thoroughbreds, but in neither breed was this finding significantly (P = 0.39 and P = 0.07 for Standardbreds and Thoroughbreds,

Table 1—Medical history and results of initial physical and laboratory examinations in Standardbreds with a pulmonary abscess that were treated at any of 4 veterinary medical teaching hospitals, 1987 to 1995

	Raced after treatment				Failed to race after treatment			
Variable	n	25th percentile	Median	75th percentile	n	25th percentile	Median	75th percentile
Age on admission (y)	23	2	3*	3	2	5	7	9
No. of days ill before admission	21	7	14	30	1	NA	1	NA
No. of days hospitalized	23	0	5	12	2	9	10	11
Rectal temperature (C)	22	37.2	38.2	38.7	2	37.5	38.0	38.5
Heart rate (beats/min)	22	40	45	52	2	36	36	36
Respiratory rate (breaths/min)	21	20	24	38	2	16	18	20
PCV (%)	21	32	35	41	2	26	30	33
Total protein (g/dl)	14	6.8	7.0	7.8	2	6.9	7.7	8.5
WBC count (10^3 cells/μl)	21	9.3	13.9	18.8	2	6.3	7.8	9.3
Neutrophils (10^3 cells/μl)	21	6.3	9.0	15.6	2	4.6	5.6	6.7
Lymphocytes (10^3 cells/μl)	21	1.2	3.1	4.8	2	1.2	1.7	2.1
Monocytes (10^3 cells/μl)	19	0.2	0.4	0.9	2	0.2	0.4	0.5
Fibrinogen (mg/dl)	19	300	380*	600	2	900	930	960

*Values were significantly ($P < 0.05$) different from values of horses that failed to race after treatment.
NA = Not available.
n = Number of horses for which data were available for that variable. For horses that failed to race, the 25th and 75th percentiles are minimum and maximum values for that variable.

Table 2—Medical history and results of initial physical and laboratory examinations in Thoroughbreds with a pulmonary abscess treated at any of 4 veterinary medical teaching hospitals, 1987 to 1995

	Raced after treatment				Failed to race after treatment			
Variable	n	25th percentile	Median	75th percentile	n	25th percentile	Median	75th percentile
Age on admission (y)	13	3	3	4	7	3	3	7
No. of days ill before admission	12	11	30	60	7	5	12	150
No. of days hospitalized	13	1	10	15	7	5	9	12
Rectal temperature (C)	13	37.9	38.2	38.6	7	38.4	38.7	39.1
Heart rate (beats/min)	12	36	44	48	7	40	48	64
Respiratory rate (breaths/min)	11	20	28	30	7	16	24	30
PCV (%)	8	27	30	38	3	31	36	38
Total protein (g/dl)	8	6.9	7.4	7.8	3	6.6	8.0	8.0
WBC count (10^3 cells/μl)	8	9.0	12.0	15.6	5	6.8	9.0	16.2
Neutrophils (10^3 cells/μl)	8	5.8	9.0	12.7	5	3.7	6.1	13.1
Lymphocytes (10^3 cells/μl)	8	1.7	2.5	3.0	5	2.1	2.9	3.3
Monocytes (10^3 cells/μl)	6	0.1	0.7	1.5	3	0.0	0.8	0.8
Fibrinogen (mg/dl)	9	155	642	830	4	468	496	534

n = Number of horses for which data were available for that variable.
See Table 1 for key.

respectively; both tests 1-tailed) associated with a decreased chance for resumption of racing. Anaerobic organisms were cultured from 2 of 18 (11%; 95% CI, 1 to 35%) tracheal aspirates obtained from Standardbreds and from 5 of 16 (31%; 95% CI, 11 to 59%) tracheal aspirates obtained from Thoroughbreds; however, in both breeds, isolation of an anaerobic bacteria did not significantly ($P = 0.78$ and $P = 0.35$ for Standardbreds and Thoroughbreds, respectively; both tests 1-tailed) decrease the chances for resuming racing. For the Standardbreds that raced following treatment, an anaerobic or polymicrobial infection was not significantly ($P = 0.38$ and $P = 0.10$, respectively) detrimental to their cumulative racing performance (SPR for all years after they had resumed racing). Thoroughbreds with an anaerobic or polymicrobial infection had significantly ($P = 0.04$ and $P = 0.03$, respectively) greater cumulative SPR than those Thoroughbreds that did not have these types of infections.

Treatment—Information on the antimicrobial regimen was available for 23 Standardbreds and 19 Thoroughbreds. A combination of penicillin and an aminoglycoside was used in 6 Standardbreds and 9 Thoroughbreds. Ceftiofur was used in 10 Standardbreds and 3 Thoroughbreds. Trimethoprim-sulfamethoxazole or -sulfadiazine were used alone or in combination with other drugs in 9 Standardbreds and 10 Thoroughbreds, and metronidazole was used in combination with the aforementioned drugs in 7 Standardbreds and 8 Thoroughbreds.

Location of abscess—In 15 of 25 (60%; 95% CI, 38 to 79%) Standardbreds, abscesses were located in the caudodorsal lung field, whereas in 14 of 20 (70%; 95% CI, 45 to 89%) Thoroughbreds, abscesses were located in the caudodorsal lung field (**Fig 1**). Remaining abscesses were located in the caudoventral region alone or in both the caudoventral and caudodorsal regions. With the exception of 8 horses, all abscesses appeared as a single mass. In 18 of 25 (72%) Standardbreds and 14 of 20 (70%) Thoroughbreds, abscesses were cavitated. In 22 of the 45 (49%) horses, it was not possible to determine whether the abscess

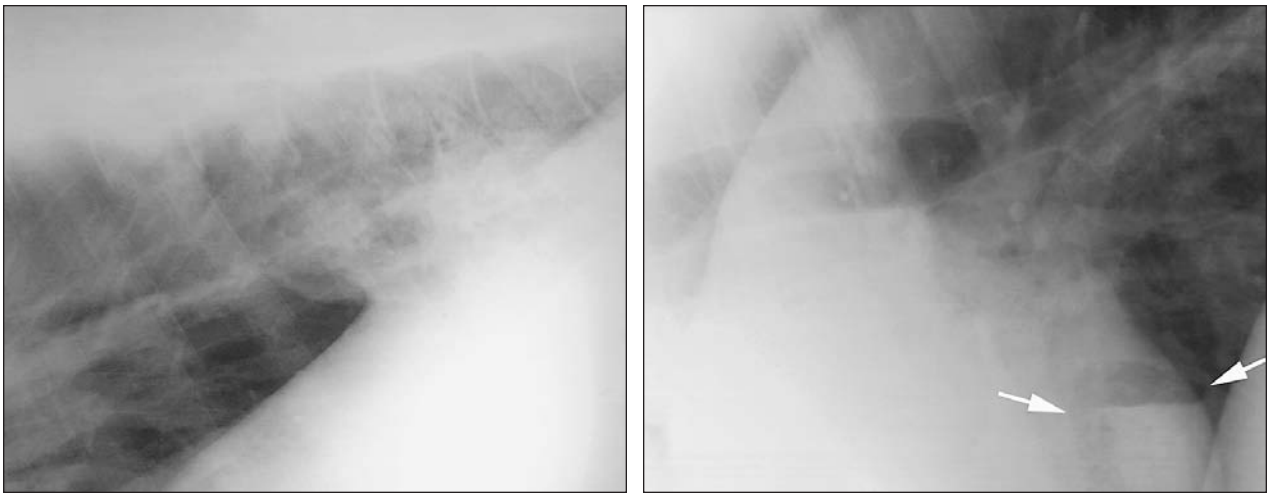

Figure 1—Lateral radiographic views of the thorax of a horse with pulmonary abscesses located in the caudodorsal (left) and caudoventral (right) lung fields. Notice the fluid-air interface (arrows) for the abscess in the caudoventral lung field.

Table 3—Starts percentile rank (SPR) for racehorses before and after diagnosis and treatment of lung abscesses at any of 4 veterinary medical teaching hospitals, 1987 to 1995

	Standardbreds				Thoroughbreds			
SPR	n	25th percentile	Median	75th percentile	n	25th percentile	Median	75th percentile
Before treatment	6	44	57	88	17	36	55	90
Year one after treatment	23	32	74	91	13	61	78	92
Cumulative after treatment	23	44	50	81	13	49	61	82

The SPR before treatment represents the year(s) immediately preceding the diagnosis and treatment of the lung abscess, year one after treatment represents the performance for the first year of racing after treatment, and cumulative SPR after treatment represents the cumulative performance for all years after the horse resumed racing following treatment. All median values were similar to those of the population of horses of similar age, breed, and sex (SPR of the population[6] is 50).

was located predominantly on the left or right side because of its proximity to the mediastinum. For abscesses in which the location was known, there was a similar prevalence on the left and right sides (Standardbreds, $P = 0.48$; Thoroughbreds, $P = 0.59$).

Racing outcome—Following discharge from the referral hospital, a median of 174 days (25th percentile, 85 days; 75th percentile, 262 days) elapsed before Standardbreds resumed racing, whereas a median of 201 days (25th percentile, 135 days; 75th percentile, 269 days) elapsed before Thoroughbreds resumed racing. Prior to admission to the referral hospital, 16 of 25 (64%; 95% CI, 43 to 82%) Standardbreds and 17 of 20 (85%; 95% CI, 62 to 97%) Thoroughbreds had raced; therefore, various SPR for the horses were compared (**Table 3**).

For the 14 Standardbreds that had raced both prior to and after their illness, median SPR for the year(s) preceding the illness was not significantly greater than the median SPR for the year immediately following the illness ($P = 0.42$) or the cumulative SPR for all years after the illness ($P = 0.38$). For the 12 Thoroughbred horses that had raced prior to and after their illness, median SPR for the year(s) preceding the illness was not significantly greater than the median SPR for the year immediately following the illness or the cumulative SPR for all years after the illness ($P = 0.18$ and $P = 0.53$, respectively).

Discussion

In the study reported here, which focused on Standardbreds and Thoroughbreds that were examined at referral centers for the diagnosis and treatment of primary lung abscesses, we determined that abnormalities recorded in the medical history or results of physical or clinicopathologic examinations during initial admission were not associated with resumption of racing. Most pulmonary abscesses were located in the caudodorsal lung field and had a similar prevalence on the left and right side of the thorax. Types of bacterial organisms isolated from a transtracheal aspirate were not associated with subsequent racing performance. The racing performance of horses treated for pulmonary abscesses and discharged was not significantly different from the racing performance before the illness or from the expected average racing performance (SPR = 50) of the general population of racehorses of the same age, sex, and breed.[6]

These conclusions were applicable specifically to those horses with primary lung abscesses that were referred to veterinary teaching hospitals for evaluation and care and that survived and were discharged to the owners. It would have been valuable to determine whether historical, physical, or laboratory variables of all horses referred with primary pulmonary abscesses were predictive of survival; however, we specifically excluded those horses that were euthanatized or died, because this outcome may have been influenced by

numerous confounding factors. For example, financial or emotional considerations, clinical experience of the attending veterinarian, development of secondary complications such as laminitis, colitis, or phlebitis, or the existence of other factors not adequately detailed in the medical records may have influenced the decision to euthanatize a horse.

Nonetheless, use of data from multiple referral hospitals and restricting analysis to survivors enabled us to analyze the outcome of a larger number of cases than in previous reports and to determine whether horses were able to resume a similar level of racing performance in the short- or long-term period after the illness. For Standardbreds and Thoroughbreds, median SPR for short-term (the first year after resumption of racing) or cumulative racing performance was not significantly different from the median SPR for the same horses prior to the diagnosis and treatment of the pulmonary disorder. Clearly, a much larger case series would have allowed testing for even smaller differences than was detected in our study, but horses meeting our selection criteria were rather rare (45 cases in 13 years from 4 institutions). Although horses responding successfully to treatment were not routinely radiographed after initial admission, we believe that lung damage capable of compromising performance did not develop and affect future athletic capacity. Understandably, nobody can know the future athletic capacity for these horses had they not become ill, but their prior performance and the expected population means were used as estimates. Still, a penalty was inferred (at least for the Thoroughbreds) from lung abscesses, because 7 of 20 Thoroughbreds failed to race after treatment and discharge. Our findings mirror those of a previous multiple-center study[6] in which investigators evaluated future athletic performance of foals treated for *Rhodococcus equi* pneumonia. Unfortunately, comparable longitudinal studies of adult athletic dogs with pulmonary abscesses have not yet been performed, precluding extrapolations from another species used for a similar purpose.

It has been documented in studies of sedentary humans receiving antimicrobial treatment for pulmonary abscesses that within 1 month after initiation of treatment, there is an 80% reduction in size of cavities < 3 cm in diameter[7] and that 70% of cavitary lesions resolve completely by 3 months.[8-10] Although the total duration of antimicrobial treatment of horses in our study was not completely known, it was assumed that a treatment period similar to that in human studies was implemented, because the elapsed median time from hospital discharge to first race was 5.8 months for Standardbreds and 6.7 months for Thoroughbreds.

One surprising finding in our study was that the type of microbial infection (polymicrobial or anaerobic) failed to affect the resumption of racing and subsequent racing performance. Anaerobic organisms reside in the oral cavity and respiratory tract and are detectable in aspirated secretions. Typically, they proliferate in tissues in which exposure to host defenses is reduced or in which the oxidation-reduction potential of the tissue has been altered because of trauma, ischemia, surgical manipulation, or shock. These bacteria produce a number of exotoxins including heparinases and collagenases, which contribute to tissue damage and promote abscess formation.[11]

Anaerobic organisms are the most common microbial isolates in humans with primary lung abscesses.[12,13] Interestingly, the isolation of an aerobic organism (gram-positive or -negative), rather than an anaerobic organism, worsens the prognosis for survival in human patients.[12,13] These findings contrast with data for horses with pleuropneumonia in which it has been documented that the isolation of an anaerobic organism from the tracheal aspirates or pleural effusions of affected horses was a major prognostic indicator. Only 30 to 40% of horses with such isolates recovered, compared to those horses lacking anaerobic isolates.[14,15] The percentage of horses that recovered and went on to successful racing careers was not determined in those studies. Nevertheless, horses with anaerobic parapneumonic effusions appear to be capable of resuming athletic careers.[16]

Despite the large number of horses with pulmonary abscesses evaluated in our study, we were limited in that only 11% of the samples obtained from the Standardbreds and 31% of the samples obtained from the Thoroughbreds yielded anaerobic organisms. Whether the low number of anaerobic isolates reflected our selection bias (only surviving horses were examined) or reflected difficulties in obtaining material for anaerobic culture[11] could not be ascertained. Additional studies are required to fully determine whether the isolation of anaerobic organisms from pulmonary secretions in horses with lung abscesses affects racing ability. At this time, clinicians must exercise caution when giving owners and trainers an unfavorable prognosis for resumption of racing in horses with anaerobic isolates.

In this study, it was found that two-thirds of the abscesses were detected in the caudodorsal lung field, the site most commonly involved in exercise-induced pulmonary hemorrhage.[17] It can only be speculated that the combination of extravasated blood and exercise-induced immunosuppression[1,2] permitted aspirated bacteria to become firmly established within the lung field. One intriguing question is whether horses that have resumed athletic training are more susceptible to greater degrees of exercise-induced pulmonary hemorrhage because of potential alterations in pulmonary parenchyma. Until methods of quantifying pulmonary hemorrhage become firmly established, the effect of prior injury in the caudodorsal lung field on the degree of subsequent exercise-induced pulmonary hemorrhage remains unanswered.

Horses in this study appear to have been successfully treated, as determined on the basis of resumption of racing. However, because of the costs incurred during diagnostic testing, treatment, and the recuperative period, and because of the amount of time lost for income generation, primary pulmonary abscesses negatively impact short-term racing performance.

[a]Epi Info 6, version 6.04b, Centers for Disease Control and Prevention, Atlanta, Ga.

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Correction: Toxicosis in cats erroneously treated with 45 to 65% permethrin products

In "Toxicosis in cats erroneously treated with 45 to 65% permethrin products" (*JAVMA*, Vol 215, pp 198-203), the second sentence in the first paragraph on page 199 should read, "In 11 of the 12 cats, the product was intentionally, although not maliciously, applied to the animal by the owner."