

Performance and mortality of farmed hares

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Performance and mortality of hares were evaluated for 2 consecutive years in a large farm in Veneto Region (Northern Italy). On average, fertile reproductive pairs ($n = 318$) gave birth 4.8 times and produced 11.4 live leverets, weaned 8.4 leverets and produced 7.0 growing hares (60 days) every year. Mean mortality was 3.6%, 22.9%, 9.7% and 2.5% in newborn (0 to 2 days of age), suckling (3 to 25 days), growing (26 to 60 days) and sub-adult (61 days until sale) hares, respectively. The main causes of mortality were enteric diseases (75.5%, 75.9% and 12.1% in suckling, growing and sub-adult hares, respectively), followed by respiratory diseases (3.4%, 8.0% and 36.2% in suckling, growing and sub-adult hares, respectively), starvation (11.3% and 8.8% in suckling and growing hares, respectively) and trauma (7.1%, 2.3% and 30.2% in suckling, growing and sub-adult hares, respectively). In reproducing hares, mortality was 24.7% and 15.4% in 2010 and 2011, respectively. Respiratory diseases (34.8%) and ulcerative pododermatitis (18.9%) were the most common pathological changes detected in reproducing hares. Farmed hares seem to be affected by diseases resembling those of rabbits reared under intensive conditions. It seems necessary to improve the husbandry of hares to reach satisfactory technical standards and to preserve their health.

Keywords: hares, performance, mortality, pathology, enteric disease

Implications

Nearly 40% of reared hares die before they are sold or reach reproductive age. The main causes of mortality are enteric disorders in young hares (suckling and growing). The most frequently involved bacteria are *Clostridium perfringens* and *Escherichia coli*, followed by *Clostridium spiroforme*. Hares are affected by diseases resembling those of rabbits reared under intensive conditions. It is necessary to improve the husbandry of hares to reach satisfactory technical standards and to preserve their health.

Introduction

Since World War II, the European brown hare (*Lepus europaeus* Pallas, 1778) population across Italy has decreased because of hunting, agriculture, urbanization and introduction of new pathogens, as well as natural predation (Spagnesi and Trocchi, 1992; Paci and Bagliacca, 2003). Included within minor farmed species, hares had started to be farmed by 1964 thanks to the activity of the National Institute for Wildlife (now the Institute for Environmental Protection and Research, Rome) to restore

local populations and to limit imports from other countries (Trocchi and Riga, 2005).

Difficulties in rearing this animal have been significant, and even today, efforts are less than successful. Open-air cages have been proven to be the most successful rearing system, but technical standardization is still quite low. In particular, farms have low productive dimensions (i.e. low number of pairs) and results may vary greatly (Romboli *et al.*, 1984; Spagnesi and Trocchi, 1992). As a consequence, knowledge about productive performance and, more importantly, diseases of farmed hares is lacking (Trocchi and Riga, 2005; Sánchez-García *et al.*, 2012). In contrast, a number of studies are available on free-living European brown hares in Italy and in Europe (Strauß *et al.*, 2008; Ferretti *et al.*, 2010); these mainly focus on local wild populations or farmed hares released into the wild.

The present study was aimed at evaluating the performance and the causes of mortality of hares reared in a large farm in Veneto Region (Northern Italy) during 2010 and 2011.

Material and methods

Farm and animals

The farm was located in the province of Venice (Municipality of Santa Maria di Sala) at 13 m a.s.l. The farm housed a total

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of 720 cages, of which 320 were for reproducing animals and the remaining for the other hare categories, over an area of 4500 m². A total of 318 pairs (in their 1st, 2nd and 3rd year of reproductive activity) per year were monitored during 2010 and 2011. The pairs were kept outside in roofed cages (1 m long, 1.60 m wide and 70 to 80 cm high), with wooden side and back walls and a wire net front side and floor. Cages were equipped with feeders for the manual distribution of feed and automatic nipple drinkers.

All animals were fed with a unique commercial diet specifically formulated for hares (dry matter: 89.3%; CP: 15.2%; crude fibre: 19.1%; ether extract: 3.2%). In addition, fresh alfalfa (*Medicago sativa*) during April to October and savoy cabbage (*Brassica oleracea*) during the other months were administered *ad libitum* to all animals. Litters were weaned at 24 to 25 days of age, moved into group cages and then sold or maintained on the farm for reproductive restocking.

Animals were vaccinated against staphylococcosis, pasteurellosis and European brown hare syndrome with aluminium hydroxide adjuvant autogenous vaccines produced by the Istituto Zooprofilattico Sperimentale della Lombardia e dell'Emilia Romagna (Brescia, Italy).

On-farm recordings

Data on performance and mortality were collected from January 2010 to December 2011 on six hare categories: newborn leverets (0 to 2 days of age), suckling leverets (3 to 25 days), growing hares (26 to 60 days), sub-adult hares (61 days until sale), reproducing hares (animals kept for reproduction) and culled hares (spent reproducing hares). The term 'born hares' includes all of the categories of hares (i.e. newborn, suckling, growing and sub-adult hares) born during the year.

The number of litters, leverets born alive, weaned leverets (at 25 days) and sub-adult hares per litter were recorded. Data are given as litters per present reproductive pair, litters per present fertile reproductive pair, leverets born alive per fertile reproductive pair, weaned leverets per fertile reproductive pair and sub-adult hares per fertile reproductive pair.

Sterility was estimated as the ratio between the number of hare does that never littered during the year and the total number of present hare does. The culling rate of hare does was calculated as the number of does replaced per year out

of the total of present does. Hare does were replaced because of health (e.g. respiratory diseases, mastitis, sore hocks or mortality of litters due to enteritis) and behavioural problems (e.g. cannibalism of litters) or mortality.

Health monitoring and necropsy

Health status of hares was monitored daily, and dead animals were promptly removed from cages. During 2010 and 2011, a total of 2839 animals belonging to the different hare categories died, and 87% of them were necropsied by veterinarians at the laboratories of the Department of Comparative Biomedicine and Food Science (BCA).

Laboratory analyses

Samples of pathological tissues were collected for histopathological ($n = 83$ animals), parasitological ($n = 67$ animals), bacteriological ($n = 307$ animals) and virological ($n = 55$ animals) examinations to confirm the suspected diagnosis. Histopathology and parasitology were performed at the BCA Laboratories, whereas bacteriology and virology were carried out at the Clinical Diagnostic Laboratory, Istituto Zooprofilattico Sperimentale delle Venezie, Legnaro PD, Italy. Bacteriological examinations were performed according to the procedures previously described by Agnoletti *et al.* (2004), Pisoni *et al.* (2007) and Cocchi *et al.* (2008). All of the other laboratory analyses were carried out according to the standard techniques in use at the laboratories.

Statistical analysis

The performance data of the 2 years were analysed by a one-way ANOVA using the GLM procedure (SAS Institute Inc., Cary, NC, USA). Differences in mortality rate and occurrence of pathological changes between the years were analysed by χ^2 -test. Differences among means with $P < 0.05$ were accepted as representing statistically significant differences.

Results

Reproductive performance and mortality

The performances of the reproductive pairs during the 2 years were very similar (Table 1). Sterility ranged from 8.2% in 2010 to 8.8% in 2011. The fertile pairs littered on average 4.8 times

Table 1 Reproductive performance of hares per year during 2010 and 2011

| | Year | | Probability |
|---|-------|-------|-------------|
| | 2010 | 2011 | |
| Litter per fertile reproductive pair (n) | 4.76 | 4.85 | ns |
| Leverets born alive per present reproductive pair (n) | 10.49 | 10.41 | ns |
| Leverets born alive per fertile reproductive pair (n) | 11.42 | 11.41 | ns |
| Weaned leverets (25 days) per fertile reproductive pair (n) | 8.02 | 8.76 | ns |
| Sub-adult hares per fertile reproductive pair | 6.97 | 7.02 | ns |
| Sterility (%) | 8.2 | 8.8 | ns |
| Culling rate of hare does (%) | 81.1 | 69.2 | <0.001 |

per year, and the average yearly number of live leverets was 10.5 per reproductive pair and 11.4 per fertile pair. The fertile pairs weaned 8.4 leverets (25 days of age) per year, and the number of growing hares (60 days of age) per reproductive pair was 7.0. The culling rate of reproducing females significantly decreased from 81.1% during 2010 to 69.2% during 2011.

Total mortality of born hares was 39.0% and 38.5% in 2010 and 2011, respectively (Table 2). Most animals died during the suckling period with an average value of 22.9%. Neonatal mortality was 3.6% on average. A rather significant percentage of mortality (9.7%) was recorded in growing hares, whereas sub-adult hares showed the lowest mortality rate (2.5%). Although the total mortality of born hares was similar, in 2010 losses were significantly higher in newborn and suckling leverets and lower in growing and sub-adult hares than 2011. Reproducing hare mortality was also higher during 2010 compared with 2011 (24.7% v. 15.4%; $P < 0.001$).

Table 2 Mortality rate of hares during 2010 and 2011

| | Year | | Probability |
|-----------------------------|------|------|-------------|
| | 2010 | 2011 | |
| Mortality of born hares (%) | 39.0 | 38.5 | ns |
| Newborn leverets | 5.1 | 2.1 | <0.001 |
| Suckling leverets | 24.7 | 21.1 | <0.001 |
| Growing hares | 7.1 | 12.2 | <0.001 |
| Sub-adult hares | 2.0 | 3.0 | 0.02 |
| Reproducing hares (%) | 24.7 | 15.4 | <0.001 |

Causes of mortality

In most cases, no evident pathological changes were detected at necropsy in newborn leverets. On average, the most common causes of mortality in suckling leverets were enteric diseases (75.5% of dead leverets), followed by starvation (11.3%), traumas (7.1%) and respiratory diseases (3.4%) (Table 3). The most frequent pathological findings associated with enteric disease were catarrhal (76.3% of leverets dead for enteric diseases) and catarrhal-haemorrhagic (16.2%) entero-typhlitis with gut distension, yellow to dark brown watery gut content and undigested milk in the stomach (Table 4). *Clostridium perfringens* and *Escherichia coli* were the most commonly isolated bacteria, followed by *Clostridium spiroforme* (Table 5).

On average, in the 2 years, enteric diseases represented 75.9% of the causes of mortality in growing hares, followed by starvation (8.8%), respiratory diseases (8.0%) and traumas (2.3%) (Table 3). The main pathological changes associated with enteric disease were catarrhal entero-typhlitis (61.8%) and coprostitis (14.4% in 2010 and 24.0% in 2011; $P < 0.05$) (Table 4). Coprostitis was characterized by abdominal distension with caecal impaction and gaseous watery content in the stomach and in the intestine. Sporadically, mucous colon content, splenomegaly, bladder distension and aspiration pneumonia were observed at necropsy. Either *C. perfringens* or *E. coli* or both bacteria were isolated from intestinal contents (Table 5). During autumn 2011, coccidiosis was diagnosed in two hares affected by coprostitis.

In sub-adult hares, the predominant causes of mortality were respiratory diseases (36.2%), followed by trauma (30.2%), enteric diseases (12.1%) and purulent otitis (8.1%)

Table 3 Causes of mortality in hares during 2010 and 2011 (the value within parenthesis is the percentage of animals affected on the total of animals submitted to necropsy)

| | | | Animals showing lesions of | | | | |
|--|--------------|---------------------------|----------------------------|----------------------|------------|-------------------------|--------------------|
| | Dead animals | Not submitted to necropsy | Enteric diseases | Respiratory diseases | Starvation | Traumatic lesions | Other ^a |
| Year 2010 | | | | | | | |
| Newborn leverets (<i>n</i>) | 170 | — | — | — | — | — | 170 (100%) |
| Suckling leverets (<i>n</i>) | 824 | 116 | 531 (75.0%) | 18 (2.5%) | 90 (12.7%) | 47 (6.6%) | 22 (3.1%) |
| Growing hares (<i>n</i>) | 238 | 84 | 111 (72.1%) | 15 (9.7%) | 12 (7.8%) | 3 (1.9%) | 13 (8.4%) |
| Sub-adult hares (<i>n</i>) | 67 | 7 | 6 (10%) | 19 (31.7%) | — | 25 (41.7%) | 10 (16.7%) |
| Reproducing animals (<i>n</i>) | 157 | 27 | 3 (2.3%) | 33 (25.4%) | — | 11 (8.5%) ^b | 83 (63.8%) |
| Total (<i>n</i>) | 1456 | 234 | 651 (53.3%) | 85 (7.0%) | 102 (8.3%) | 86 (7.0%) | 298 (24.4%) |
| Year 2011 | | | | | | | |
| Newborn leverets (<i>n</i>) | 70 | — | — | — | — | — | 70 (100%) |
| Suckling leverets (<i>n</i>) | 699 | 41 | 500 (76.0%) | 28 (4.3%) | 65 (9.9%) | 50 (7.6%) | 15 (2.3%) |
| Growing hares (<i>n</i>) | 405 | 73 | 258 (77.7%) | 24 (7.2%) | 31 (9.3%) | 8 (2.4%) | 11 (3.3%) |
| Sub-adult hares (<i>n</i>) | 99 | 10 | 12 (13.5%) | 35 (39.3%) | — | 20 (22.5%) | 22 (24.7%) |
| Reproducing animals (<i>n</i>) | 98 | 5 | 3 (3.2%) | 41 (44.1%) | — | 17 (18.3%) ^b | 32 (34.4%) |
| Culled animals (<i>n</i>) ^c | 12 | — | — | 1 (8.3%) | — | 1 (8.3%) | 10 (83.3%) |
| Total (<i>n</i>) | 1383 | 129 | 773 (61.6%) | 129 (10.3%) | 96 (7.7%) | 96 (7.7%) | 160 (12.8%) |

^aNo evident pathological change, purulent otitis, diaphragmatic hernia, hepatitis, cystitis, ulcerative pododermatitis, chronic nephropathy, mastitis, reproductive diseases and pericarditis.

^bStatistically significant difference ($P < 0.05$) between 2010 and 2011.

^cCulled animals: spent reproducing hares.

Table 4 Main pathological changes associated with mortality of hares (the value within parenthesis is the percentage of animals showing the lesion on the total of animals affected)

| Pathological change | Suckling leverets | | Growing hares | | Sub-adult hares | | Reproducing animals | |
|---|-------------------|------------|------------------------|------------------------|-----------------|-----------|---------------------|-----------------|
| | 2010 | 2011 | 2010 | 2011 | 2010 | 2011 | 2010 | 2011 |
| Enteric lesions | | | | | | | | |
| Catarrhal enteritis | 2 (0.4) | 3 (0.6) | 2 (1.8) | 2 (0.8) | — | — | — | — |
| Catarrhal typhlitis | 48 (9.0) | 19 (3.8) | 8 (7.2) | 1 (0.4) | — | — | — | — |
| Catarrhal-haemorrhagic enteritis | 1 (0.2) | — | — | 1 (0.4) | 2 (33.3) | — | — | — |
| Catarrhal-haemorrhagic typhlitis | 2 (0.4) | 2 (0.4) | 1 (0.9) | — | — | — | — | — |
| Catarrhal entero-typhlitis | 401 (75.5) | 386 (77.2) | 74 (66.7) | 154 (59.7) | 2 (33.3) | 4 (33.3) | 3 (100.0) | 1 (33.3) |
| Catarrhal-haemorrhagic entero-typhlitis | 77 (14.5) | 90 (18.0) | 10 (9.0) | 38 (14.7) | — | 1 (8.3) | — | 2 (66.7) |
| Coprostasis | — | — | 16 (14.4) ^a | 62 (24.0) ^a | 2 (33.3) | 7 (58.3) | — | — |
| Total (n) | 531 | 500 | 111 | 258 | 6 | 12 | 3 | 3 |
| Respiratory lesions | | | | | | | | |
| Purulent rhino-sinusitis | 2 (11.1) | 3 (10.7) | 6 (40.0) | 9 (37.5) | 2 (10.5) | — | — | — |
| Pneumonia/pleuropneumonia | 14 (77.8) | 19 (67.9) | 5 (33.3) | 8 (33.3) | 2 (10.5) | 2 (5.7) | 3 (9.0) | 1 (2.4) |
| Fibrinous pneumonia/pleuropneumonia | — | 3 (10.7) | — | 3 (12.5) | 1 (5.3) | 5 (14.3) | 5 (15.2) | 17 (41.5) |
| Purulent pneumonia/pleuropneumonia | 2 (11.1) | 2 (7.1) | 3 (20) | 3 (12.5) | 13 (68.4) | 28 (80.0) | 20 (60.6) | 23 (56.1) |
| Lung oedema | — | 1 (3.6) | 1 (6.7) | 1 (4.2) | 1 (5.3) | — | 5 (15.2) | — |
| Total (n) | 18 | 28 | 15 | 24 | 19 | 35 | 33 ^b | 41 ^b |
| Other | | | | | | | | |
| Purulent otitis | — | 1 | — | 1 | 6 | 6 | 1 | 4 |
| Ulcerative pododermatitis | — | — | — | — | — | — | 42 ^c | 5 ^c |
| Chronic nephropathy | — | — | — | — | — | — | 10 | 12 |
| Mastitis | — | — | — | — | — | — | 7 | 2 |
| Pericarditis | — | — | — | — | — | 1 | 2 | 1 |
| Reproductive lesions ^d | — | — | — | — | — | — | 4 | 4 |
| Total (n) | 22 | 15 | 13 | 11 | 10 | 22 | 83 | 32 |

^aStatistically significant difference ($P < 0.05$) between 2010 and 2011.^bStatistically significant difference ($P < 0.01$) between 2010 and 2011.^cStatistically significant difference ($P < 0.001$) between 2010 and 2011.^dPyometra, dystocia and uterine prolapse in females; orchitis in males.

(Table 3). The most common respiratory pathological change was purulent pleuropneumonia (75.9%), consisting of apical and bilateral pneumonia with the presence of purulent exudate in the bronchi and alveoli and sometimes in the trachea, as well as exudative pleuritis and, in more chronic cases, pulmonary abscesses (Table 4). The aetiological agent associated with these lesions was always *Pasteurella multocida* (Table 5). Hares affected by purulent otitis showed head tilt, balance disorders with falling and rolling, and sometimes inappetence. At necropsy, accumulation of purulent exudate was observed in the inner and medium ear. *P. multocida*, *Staphylococcus aureus* and *Pseudomonas aeruginosa* were isolated at bacteriology (Table 5). Sub-adult hares ($n = 24$) were found positive for coccidia during autumn 2011.

Although causes of mortality were rather similar in most of the hare categories over the 2 years, in reproducing hares two main differences were recorded. Respiratory diseases affected 25.4% of dead reproducing hares during 2010 and 44.1% during 2011 ($P < 0.01$; Table 3). The most common pathological change was purulent pleuropneumonia (58.1% on average; Table 4). Ulcerative pododermatitis was detected in 32.3% and 5.4% of the specimens ($P < 0.001$) in 2010 and 2011, respectively (Table 4). Multifocal visceral

purulent lesions, such as renal and splenic abscesses, thrombo-embolic pneumonia and/or fibrinous pleuritis were also observed in association with metatarsal ulcerations. These lesions were caused by *S. aureus* (Table 5) and, in more chronic cases, AA-amyloid was observed histologically in sections of the liver, kidneys and spleen stained with Congo red. Furthermore, enteric diseases (2.7%), traumas (8.5% in 2010 and 18.3% in 2011, $P < 0.05$) and purulent otitis (2.2%) were observed in reproducing hares (Table 3). Other pathological findings were purulent mastitis caused by *P. multocida* or *S. aureus* and reproductive disorders, such as pyometra, dystocia, uterine prolapse and orchitis (Tables 4 and 5). Three reproducing hares were affected by *Taenia pisiformis*.

Discussion and conclusions

In *Lepus europeus*, reproductive activity and length are regulated by the photoperiod rather than by the environmental conditions (Trocchi and Riga, 2005). Under captivity, the reproductive performance of hares seems to be similar to that observed in free-ranging hares, that is, 7.8 to 11.3 growing hares yearly produced per doe (Trocchi and Riga, 2005). In the

Table 5 Results of bacteriological examinations of tissue samples taken at necropsy

| | Suckling leverets | | Growing hares | | Sub-adult hares | | Reproducing hares | |
|--|-------------------|------|---------------|------|-----------------|------|-------------------|------|
| | 2010 | 2011 | 2010 | 2011 | 2010 | 2011 | 2010 | 2011 |
| Intestine samples (n) | 55 | 49 | 12 | 26 | 2 | 6 | 1 | 2 |
| <i>Escherichia coli</i> | 6 | – | 6 | 6 | 1 | 2 | – | – |
| <i>C. perfringens</i> | 18 | 17 | 3 | 4 | 1 | 1 | – | – |
| <i>E. coli</i> + <i>C. perfringens</i> | 6 | – | – | 4 | – | 1 | – | – |
| <i>Clostridium spiroforme</i> ^a | 1 | 9 | – | – | – | – | – | 2 |
| <i>Clostridium sordelli</i> | 1 | 1 | – | – | – | – | – | – |
| <i>Klebsiella</i> spp. | 3 | – | – | – | – | – | – | – |
| <i>Staphylococcus aureus</i> | 1 | – | 3 | – | – | – | – | – |
| Other spp. ^b | 8 | 7 | – | 8 | – | – | – | – |
| Negative | 11 | 15 | – | 4 | – | 2 | 1 | – |
| Lung samples (n) | 7 | 11 | 4 | 5 | 10 | 21 | 17 | 28 |
| <i>Pasteurella multocida</i> | 5 | 8 | 3 | 4 | 5 | 18 | 7 | 20 |
| <i>S. aureus</i> | – | – | – | – | 1 | – | 2 | – |
| <i>Bordetella bronchiseptica</i> | – | – | – | – | – | – | 2 | – |
| Negative | 2 | 3 | 1 | 1 | 4 | 3 | 6 | 8 |
| Nasal cavity samples (n) | – | – | 4 | 7 | 1 | – | – | – |
| <i>P. multocida</i> | – | – | – | 2 | 1 | – | – | – |
| <i>S. aureus</i> | – | – | 4 | 2 | – | – | – | – |
| Negative | – | – | – | 3 | – | – | – | – |
| Ear samples (n) | – | – | – | – | 4 | 4 | 1 | – |
| <i>Pseudomonas aeruginosa</i> | – | – | – | – | 1 | 1 | – | – |
| <i>S. aureus</i> | – | – | – | – | 1 | 1 | 1 | – |
| <i>Staphylococcus coagulase</i> – | – | – | – | – | 1 | – | – | – |
| <i>P. multocida</i> | – | – | – | – | – | 2 | – | – |
| Negative | – | – | – | – | 1 | – | – | – |
| Limb samples (n) | – | – | – | – | – | – | 21 | 2 |
| <i>S. aureus</i> | – | – | – | – | – | – | 19 | 2 |
| Negative | – | – | – | – | – | – | 2 | – |
| Breast samples (n) | – | – | – | – | – | – | 5 | 2 |
| <i>S. aureus</i> | – | – | – | – | – | – | 5 | – |
| <i>P. multocida</i> | – | – | – | – | – | – | – | 2 |
| Negative | – | – | – | – | – | – | – | – |

^aIdentified by both bacteriological and bacterioscopic examination.^b*Enterococcus* spp., *Bacillus* spp., *Pseudomonas aeruginosa*, *Streptococcus* spp.

present study, the average number of growing hares (7.0) produced per fertile reproductive pair was higher than that previously reported in farmed hares (5.3) (Spagnesi and Trocchi, 1992), whereas the yearly number of litters per fertile pair (4.8) and the number of live leverets (10.5) were comparable to published data (from 1.3 to 5 litters/year and 4.0 to 11.1 live leverets annually) (Romboli *et al.*, 1984; Nioli, 2006).

Sterility of reproducing hares was lower than that reported in other studies (8.5% v. 11% to 25% according to Tocchini *et al.*, 2000 and Mantovani, 1996). The yearly culling rate (75%) was higher compared with previous findings (40% to 55%; Spagnesi and Trocchi, 1992; Mantovani, 1996) because the hare does were replaced as soon as health or behavioural problems emerged.

Mortality of born hares was very high, as nearly 40% of hares died before sale or before reaching reproductive age. These findings are consistent with Tocchini *et al.* (2000) (mortality rate: 26% to 39%) and Miragoli (2007) (48%).

Romboli *et al.* (1984) documented a mortality of young hares (until 30 days of age) of 17.9% to 28.6%, which was significantly affected by the type of cage and whether it offered less or more protection from climatic and stressing factors.

The main causes of mortality in born hares were enteric disorders, affecting primarily young animals (i.e. suckling leverets and growing hares), which confirms the previously documented susceptibility of hares to digestive problems (Spagnesi and Trocchi, 1992). In both years, more than 50% of born hares showed pathological changes attributable to enteric disorders. Among these, coprostatitis affected 21.1% of growing hares, in contrast to Miragoli (2007) who reported 2%. Although a similar disease named Leporine Dysautonomia has been documented in free-living hares by Griffiths and Whitwell (1993), the pathological condition affecting our hares rather resembled the epizootic rabbit enteropathy of farmed rabbits (Licois *et al.*, 2006).

In the present study, the bacteria most frequently involved in enteric disorders were *C. perfringens* and *E. coli*, followed by *C. spiroforme*. These pathogens have been previously documented as a common cause of enteritis in farmed lactating (Ducluzeau *et al.*, 1975) and growing hares (Miragoli, 2007).

On the basis of our observations and those from other studies (Ducluzeau *et al.*, 1975; Miragoli, 2007), bacterial enteric diseases seem to be a common health problem in young captively bred hares in contrast to free-living hares (Spagnesi and Trocchi, 1992). As in rabbits, the suckling and weaning periods in young hares are critical for the development of a balanced gut microbiota: any disruption of the digestive equilibrium may promote the growth of potentially pathogenic bacteria, such as clostridia and coliforms (Peeters, 2000; De Blas *et al.*, 2012). In free-living hares, the development of intestinal microbiota is favoured by contact with soil, whereas in farmed hares, the development and composition of the gut microflora depends on the contact with parents (Spagnesi and Trocchi, 1992), as well as on the feeding regime.

Respiratory diseases were the second most common cause of mortality (~10% in both years), as previously documented (7.5% of the total mortality) (Miragoli, 2007) and affected mainly older animals (sub-adult and reproducing hares). Respiratory diseases were observed during autumn and winter, when sudden variations in temperature occurred, and were caused mainly by *P. multocida*, which was able to cause disease in older animals (Spagnesi and Trocchi, 1992). Furthermore, rhino-sinusitis was observed in growing hares whose parents were affected by pododermatitis or mastitis. Accordingly, transmission of *P. multocida* and *S. aureus* from reproducing hares to their progeny may be hypothesized, similarly to rabbits (Coudert *et al.*, 2006; Vankraeynest *et al.*, 2006).

A number of other pathological conditions were detected in the present study. Starvation in lactating and growing hares and trauma in born hares accounted for ~10% of mortality, as already observed (15% at 25 to 75 days of age; Paci *et al.*, 1998). Ulcerative pododermatitis associated with multifocal visceral purulent lesions and generalized AA-amyloidosis was observed only in reproducing hares and was caused by *S. aureus*, most likely predisposed by mechanical injury from the cage wire floor, as evidenced in rabbits (Rosell and de la Fuente, 2013). To our knowledge, ulcerative pododermatitis has never been documented in hares, but generalized AA-amyloidosis has been reported in free-living hares affected by chronic inflammation (Geisel and Linke, 1988).

Purulent otitis, affecting mainly sub-adult hares, was caused by *P. multocida*, *Staphylococcus* spp. and *P. aeruginosa*, well-known aetiological agents of suppurative otitis in rabbits (Coudert *et al.*, 2006; Vankraeynest *et al.*, 2006). Parasitic diseases, that is, coccidiosis and cysticercosis, were occasionally diagnosed and attributed to the administration of potentially contaminated fresh alfalfa or savoy cabbage, as previously reported (Spagnesi and Trocchi, 1992).

In conclusion, losses due to mortality were important and causes of mortality resembled those recognized in the intensive rearing system of rabbits. As for rabbits, most of the diseases cannot be attributed only to a specific aetiological agent but also to some important weaknesses in the farming (i.e. stocking density, protection offered by cages), feeding and management systems. Because an improvement of health status would permit owners to greatly increase their farm profitability as well as the animals welfare, further studies are needed for standardizing husbandry techniques and for defining and controlling the predisposing factors to disease.

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