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Association of lameness with milk yield and lactation curves in Chios dairy ewes

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1 **Association of lameness with milk yield and lactation curves in Chios**
2 **dairy ewes**

3

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11 **Lameness and milk yield in dairy ewes**

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18 **SUMMARY**

19 The objective of the study was twofold: (i) to quantify the differences in daily
20 milk yield (DMY) and total milk yield (TMY) between lame and non-lame dairy ewes
21 and (ii) to determine the shape of lactation curves around the lameness incident. The
22 overall study was a prospective study of lameness for the surveyed sheep population,
23 with a nested study including the selection of matching controls for each lame ewe
24 separately. Two intensively reared flocks of purebred Chios ewes and a total of 283

25 ewes were used. Data, including gait assessment and DMY records, were collected on
26 a weekly basis during on-farm visits across the milking period. A general linear model
27 was developed for the calculation of lactation curves of lame and non-lame ewes,
28 whereas one-way ANOVAs were used for the comparisons between lame ewes and
29 their controls. Lameness incidence was 12.4% and 16.8% in Farm A and B,
30 respectively. Average DMY in lame ewes was significantly lower (213.8 g, $P<0.001$)
31 compared with the rest of the flock, where DMY averaged at 1.340 g. The highest
32 DMY reduction in lame ewes was observed during the 16th week of the milking
33 period ($P<0.001$), whereas, the reduction of DMY, for lame ewes, remained
34 significant at $P<0.001$ level from the eighth to the 28th week of milking. The
35 comparisons between lame and controls revealed that at the week of lameness
36 diagnosis a significant DMY reduction ($P\leq 0.001$) was observed in lame ewes (about
37 32.5%), which was maximized one week later (35.8%, $P\leq 0.001$) and continued for
38 several weeks after recovery, resulting in 19.3% lower TMY for lame ewes for the
39 first 210 days of milking period ($P<0.01$). Moreover, at flock level, TMYs for non-
40 lame and lame ewes, as calculated by the general linear model, were 318.9 kg and
41 268.0 kg, respectively. The results of this study demonstrate an evidence of significant
42 financial losses in dairy sheep due to lameness, which though, need to be accurately
43 estimated in further, more detailed, analyses.

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46

47 **KEYWORDS:** lameness, dairy sheep, milk yield, lactation curves

48 INTRODUCTION

49 Lameness is a departure from normal gait, caused by disease or injury in some
50 part of limbs or trunk, usually accompanied with pain (Boden, 1998). The aetiology
51 can be broadly classified as either genetic, congenital, physical injury or infection
52 (Coulon *et al.* 1996; Green *et al.* 2002; Winter, 2004). The notion is that lameness is
53 one of the most important health problems in sheep, related not only to impaired
54 animal welfare but to production losses, as well. Most of the available information on
55 sheep lameness relates to meat/wool producing breeds, with well documented
56 evidence of the causes, prevalence, incidence and economic consequences (Green &
57 George, 2008; Kaler & Green, 2008), which include weight loss, reproductive failure
58 and reduced wool production (Stewart *et al.* 1984; Marshall *et al.* 1991; Eze, 2002).
59 However, it is dairy sheep production that is the major industry in Greece and most
60 Mediterranean countries (De Rancourt *et al.* 2006; Gelasakis *et al.* 2012), with its
61 renowned culinary specialties, like Feta and Roquefort cheeses. Therefore, detailed
62 information regarding the effect of lameness on sheep milk production is warranted.

63 In dairy sheep, lameness incidence has been found to show high variability
64 depending on both physiological and environmental factors (Gelasakis *et al.* 2013).
65 Moreover, in the majority of cases and irrespective of the problem's magnitude within
66 the flocks, farmers underestimate lameness incidence and tend to disregard the
67 negative effects of lameness on milk production (Gelasakis *et al.* 2010). This attitude
68 bears a striking resemblance with that of dairy cow farmers (Espejo *et al.* 2006; Leach
69 *et al.* 2010). It is well established, though, that lameness is associated with a
70 significant reduction in milk yield in this species (Warnick *et al.* 2001; Green *et al.*
71 2002; Bicalho *et al.* 2008). Further research is expected to facilitate the better

72 understanding of the significance of the problem in dairy sheep as in the case of dairy
73 cows (Huxley, 2013).

74 Besides the welfare issues, one factor that could raise dairy sheep farmers'
75 awareness on lameness is to demonstrate its cost. In this respect, the quantification of
76 lameness impact on milk production is a prerequisite. Moreover, as with all diseases,
77 early detection is crucial for timely intervention and successful treatment; visual
78 identification (locomotion scoring) of lame ewes is a subjective, time consuming and
79 difficult method to apply (Kaler *et al.* 2009, Phythian *et al.* 2013) considering the
80 natural tendency of most sheep to congregate at the sight of humans observing or
81 approaching them. An objective variable would be very useful, especially if it could
82 alert farmers early in the course of the disease.

83 Hence, the objective of the present study was twofold. First, to quantify the
84 differences in daily milk yield (DMY) and total milk yield (TMY) between lame and
85 non-lame ewes and secondly, to determine the shape of lactation curves around the
86 lameness incident in order to explore the possibility to use milk recording data as an
87 early diagnostic tool.

88

89 **MATERIALS AND METHODS**

90 Two intensively reared flocks of purebred Chios ewes were used for the study.
91 Flock monitoring and data collection pertained to the period 2008-2009. The study
92 has been approved by the ethics review committee of the School of Veterinary
93 Medicine, Aristotle University of Thessaloniki.

94

95 *Animals and management*

96 A total of 170 and 113 ewes that lambed from October through December
97 2008, on Farms A and B, respectively, were considered for the study. Both farms
98 were located in Northern Greece (Farm A: 20m above sea level, latitude 40°17'18'',
99 longitude 23°09'29'' and Farm B 107 m above sea level, latitude 39°22'43'',
100 longitude 22°51'37''). A sheep shed providing a floor area of 2 m²/ewe and a volume
101 of about 10 m³/ewe was available on Farm A, but ventilation was moderate. On Farm
102 B, a shed providing a floor area of 2 m²/ewe and a volume of 12 m³/ewe was available
103 while ventilation was adequate in this case; fans were installed and operated when
104 necessary. Barley straw was used as bedding on both farms. During winter, fresh
105 bedding was added every other day; in spring and summer periods this interval was
106 extended to 5-10 days, depending on bedding condition. The bedding was removed
107 and premises were disinfected twice a year on Farm A and three times per year on
108 Farm B, using a combination of commercial disinfectants and lime. Ewes had access,
109 year round, to an earthen exercise paddock (2.5 m²/ewe).

110 On Farm A lambing started at the end of October and peaked in late
111 November. Lambs were kept with their dams for about eight weeks. On Farm B,
112 oestrus synchronization with intravaginal sponges resulted in a short lambing period
113 of about 10 days, in mid November. Lambs were artificially reared for eight weeks.

114 Ewes were machine-milked three times per day for three months and
115 thereafter, twice a day until the end of the milking period, which lasted about eight
116 months. Milking parlours were equipped with automatic milk recording systems for
117 individual ewes (SAE Afikim – Afimilk and Alpro - De Laval, for Farms A and B,
118 respectively).

119 On Farm A, feeding of ewes during the experimental period was based on
120 alfalfa hay (1.0-1.6 kg/ewe/day), barley straw (0.2-0.5 kg/ewe/day) and concentrates
121 (0.7-1.5 kg/ewe/day) comprising of corn grain (35.0%), barley grain (32.5%), soybean
122 meal (30.0%) and a mineral/vitamin supplement (2.5%). The amount of ration offered
123 was adjusted to group milk yield and pasture availability. Rations were offered in
124 troughs allowing sufficient space (0.3 m/ewe), to enable access of all ewes at the same
125 time. A five-hectare sown irrigated pasture (*Lolium perenne* + *Trifolium repens*) was
126 available for grazing from March until September. On Farm B, feeding of ewes was
127 based on alfalfa hay (0.8-1.4 kg/ewe/day), barley straw (0.1-0.4 kg/ewe/day), corn
128 silage (1.0-2.0 kg/ewe/day) and concentrates (0.7-1.3 kg/ewe/day) comprising of corn
129 grain (37.0%), barley grain (23.0%), soybean meal (16.0%), wheat bran (10.0%),
130 sunflower cake (10.0%) and a mineral/vitamin supplement (4.0%). The amount of
131 ration offered was adjusted to group milk yield. Rations were offered on a feeding belt
132 (0.33 m/ewe) which enabled access of all ewes at the same time.

133 A well-designed vaccination protocol against Brucellosis (*Brucella melitensis*
134 vaccine, strain Rev. 1), Clostridial diseases (Covexin 8A; Schering-Plough Animal
135 Health), Contagious agalactia (Agalax; CEVA), Chlamydial abortion (Enzovax;
136 Intervet International B.V.) and Paratuberculosis (Gudair Vaccine; Provet) was
137 strictly followed in both flocks. Regarding parasites, ewes were treated with
138 ivermectin (0.2 mg/kg Valaneq; Merial) and fenbendazole (Farm A, 10 mg/kg
139 Panacur; Intervet) or netobimin (Farm B, 10 mg/kg Hapadex; Schering-Plough
140 Animal Health) at the third month of gestation and at lambing, respectively. All ewes
141 were treated with an intramammary antibiotic preparation (Nafpenzal Dry Cow;
142 Intervet International) at dry-off (extra-label use). Routine foot trimming was carried
143 out once a year, at lambing. After the diagnosis of lameness, lame ewes were treated

144 using a single intramuscular injection of long acting Oxytetracycline (Alamycin LA;
145 Norbrook) at a dose rate of 20 mg/kg.

146 *Experimental design*

147 The overall study was a prospective study of lameness for the surveyed sheep
148 population. For the implementation of the study, the same veterinarian visited the
149 farms once a week throughout the entire milking period resulting in a total of 34 visits
150 per flock. Milk yield was electronically recorded daily for individual ewes in both
151 flocks. For the subsequent statistical analyses seven-day average milk yields were
152 used representing the average DMVs for the week of visit. Average DMVs, also,
153 enabled the calculation of lactation curves and enabled the comparisons between lame
154 ewes and the selected controls regarding milk yield for the pre- and postlameness
155 period.

156 Ewes were observed twice daily (in the milking parlour) by the farm owners or
157 the personnel for any abnormal behaviour. On both farms, a passageway that allowed
158 ewes to enter the milking parlour in single line was constructed to allow gait
159 observation of individual ewes. Ewes showing signs of disease or a sudden reduction
160 in DMV were clinically examined by the veterinarian at the next visit. When a ewe
161 was found lame, then a healthy one of the same age, same number and stage of
162 lactation, similar milk potential (previous lactation records) and average DMV at the
163 beginning of current milking period was chosen as a control. The selection was based
164 on data from the farm's electronic records. Both animals were colour-marked to help
165 identify them after milking for further testing. Clinical examination, microbiological
166 examination of milk samples and parasitological examination of faeces were
167 performed both on lame and control ewes in order to identify and exclude from the

168 study ewes either showing clinical signs of diseases or with subclinical mastitis and/or
169 high levels of parasitic infestation. The examinations and tests performed are
170 summarized below:

171 **(i) Clinical examination:** It comprised inspection (head, body, limbs, feet and
172 conjunctivae), palpation of udder and joints, as well as auscultation of lungs and heart.
173 Heart rate, breathing rate and body temperature were recorded. Also, body condition
174 score (BCS) was assessed using the five-point scale, from 1 (emaciated) to 5 (obese),
175 proposed by Russel *et al.* (1969).

176 **(ii) Locomotion Score (LS) and lameness:** Locomotion assessment was based on the
177 following four-point scale scoring system (Hill *et al.* 1997): 1= Normal gait, 2= No
178 obvious lameness when standing, abnormal gait when walking, 3= Shifting stance and
179 obvious lameness when walking, 4= Unwilling to bear weight on one foot when
180 standing or walking. Ewes with a locomotion score higher than 1 at least once
181 throughout the milking period were considered to be lame. All other ewes were
182 considered non-lame for the purposes of this study. The cause of lameness was
183 assessed during the clinical examination by an experienced veterinarian. Lamé feet
184 were inspected through observation and palpation in order to localize possible
185 abnormalities, injuries, lesions or painful sites. Afterwards, a detailed foot-trimming
186 was performed in order to reveal any lesions underneath the hoof wall; final diagnosis
187 of foot lameness was set on the basis of the lesions and the clinical manifestation of
188 the hoof disease.

189 **(iii) Milk sampling and assessment:** Milk samples were taken for California Mastitis
190 Test (CMT, Bovi-Vet; Kruuse) and bacteriological examination, to test for subclinical
191 mastitis (Fthenakis *et al.* 1991).

192 **(iv) Parasitological examination:** Faecal samples were collected directly from the
193 rectum and were examined for faecal egg counts (FECs) using the modified
194 McMaster method (Ministry of Agriculture, Fisheries and Food, 1986).

195 Examination and testing of case and control animals continued throughout the
196 milking period. On Farm A, 21 out of 170 ewes were found lame due to foot lesions;
197 four of them were excluded from the analysis due to subclinical or clinical mastitis of
198 either the lame or the control ewe, at some point of the study. On Farm B, lameness
199 was diagnosed in 19 out of 113 ewes. Seven of them were excluded from analysis due
200 to health problems (subclinical mastitis, metritis, hernia) or insufficient data. Finally,
201 17 and 12 lame ewes from Farms A and B, respectively, were used in the subsequent
202 statistical analysis.

203 **Data management and statistical analysis**

204 **(i) Descriptive statistics**

205 Initially, descriptive statistics were calculated including means and standard errors of
206 means for DMY and for TMY of the first 210 days of milking period, of lame ewes
207 and their selected controls.

208 **(ii) Lactation curve calculation**

209 A general linear model was developed for the calculation of lactation curves of lame
210 and non-lame ewes across milking period using ASReml (Model 1). In each flock, the
211 first lameness event during milking period was used for each ewe.

$$212 \text{TDM}_{\text{abcdkghj}} = \mathbf{m} + \mathbf{F}_a + \mathbf{YM}_b + \mathbf{LA}_c + \mathbf{MY}_d + \mathbf{W}_k + \mathbf{E}_g \mathbf{W}_k + \mathbf{L}_h + \mathbf{S}_j + \mathbf{e}_{\text{abcdkghj}}$$

213 **(Model 1)**

214 Where:

215 $TDM_{abcdkghj}$ = average DMY for the g^{th} ewe of the a^{th} flock measure on the k^{th} week of
216 milking period (kg),
217 m = overall mean,
218 F_a = fixed effect of the a^{th} flock (2 levels),
219 YM_b = fixed effect of the b^{th} interaction between lambing year and lambing month,
220 LA_c = fixed effect of the c^{th} interaction between the number of lactation and age at
221 lambing (in months),
222 MY_d = fixed effect of the d^{th} interaction between the month and the year DMY was
223 calculated,
224 W_k = fixed effect of the k^{th} week of milking period when DMY was assessed (a
225 second order polynomial was used in order milk yield curves and covariances for
226 repeated measures of the same ewe to be considered),
227 $E_g W_k$ = random effect of the interaction between the g^{th} ewe and the k^{th} week of
228 milking period when DMY was assessed (a second order polynomial was, also, used
229 for the same reasons described above),
230 L_h = fixed effect of the h^{th} lameness status (2 levels, 1= non-lame ewes, 2= lame
231 ewes),
232 S_j = fixed effect of the j^{th} week postlambing,
233 $e_{abcdkghj}$ = random residual.

234 DMYs of lame ewes and their selected controls (adjusted for number and
235 week of lactation) were compared using one-way analysis of variance (one-way

236 ANOVA); comparisons were performed per week for the period initiated four weeks
237 before lameness onset and were completed eight weeks after it.

238

239 **Effect of lameness on TMY**

240 TMY was calculated for all ewes based on the average weekly DMY solutions
241 produced by model 1. Moreover, one way ANOVAs were used in order to compare
242 TMY between lame ewes and their controls.

243

244 **RESULTS**

245 Lameness incidence on Farms A and B was 12.4% and 16.8%, respectively.
246 The majority of lameness cases were diagnosed during the first four months of
247 lactation both on Farm A (82.4%) and B (66.7%). Aetiology and duration of lameness
248 are presented in Table 1. White line abscesses (WLA) were the major causes of
249 lameness (70.6% and 58.3% of cases on Farms A and B, respectively) followed by
250 footrot, pedal joint abscesses (PJA) and injuries. Locomotion score was equal to 2 for
251 most of the WLA cases (66.7%) on Farm A and footrot and PJA were associated with
252 severe lameness (LS=4). On Farm B, the majority of cases were assigned a
253 locomotion score equal to 3, regardless the cause of lameness. Duration of lameness
254 was longer than a week in 83.3% and 47.1% of cases on Farms A and B, respectively
255 (Table 1) (Table 1 near here). In the same table, it is obvious that irrespective of the
256 etiology, most of the lameness cases occurred from January to April. In particular,
257 white line lesions were most prevalent in January and February, whereas, all of the
258 footrot cases were observed between February and April. At the end of the lactation
259 period (in July) no cases of lameness were observed.

260 All factors fitted in Model 1, including lameness, had a significant effect on
261 DMY ($P<0.05$). Average DMY in lame ewes was significantly lower (213.8 g,
262 $P<0.001$) compared with the rest of the flock, where DMY averaged at 1.340 g (a
263 reduction of about 16%). Figure 1 shows the DMY curves for non-lame and lame
264 ewes across the milking period. Mean DMY was 1.89 ± 0.107 kg and at 1.86 ± 0.061 in
265 the beginning of the milking period for lame and non-lame ewes, respectively
266 ($P>0.05$). Afterwards, DMY reduction rate tended to be higher in lame ewes, which
267 finally resulted in a significantly reduced DMY during the sixth ($P<0.05$, 1.78 ± 0.034
268 kg and 1.63 ± 0.063 kg of DMY for non-lame and lame ewes, respectively) and the
269 seventh week of milking period ($P<0.01$, 1.76 ± 0.029 kg and 1.58 ± 0.055 kg of DMY
270 for non-lame and lame ewes, respectively). The highest DMY reduction was observed
271 during the 16th week of milking period ($P<0.001$, 1.49 ± 0.013 kg vs. 1.16 ± 0.020 kg of
272 DMY for non-lame and lame ewes, respectively). The reduction of DMY, for lame
273 ewes, remained significant at $P<0.001$ level from the eighth to the 28th week of
274 milking. The reduction of DMY slowed down from the 29th week of milking period
275 ($P<0.01$, 0.83 ± 0.034 kg and 0.65 ± 0.063 kg of DMY for non-lame and lame ewes,
276 respectively) to the 34th week of milking period (end of lactation), when the
277 differences were not significant (0.49 ± 0.061 kg and 0.48 ± 0.107 kg of DMY for non-
278 lame and lame ewes, respectively).

279 Figure 2 shows the mean DMY of lame ewes and their controls per week, after
280 adjusting for number and week of lactation, initiating from the fourth week before
281 onset of lameness up to the eighth week afterwards. DMY tended to be lower for lame
282 ewes, two weeks before lameness diagnosis (10.8%, $P=0.052$, 1.66 vs. 1.48 kg for
283 control and lame ewes, respectively) and 16.1% lower one week before lameness
284 diagnosis ($P\leq 0.001$, 1.62 kg vs. 1.36 kg for control and lame ewes, respectively). At

285 the week of lameness diagnosis a significant milk yield reduction ($P \leq 0.001$) was
286 observed in lame ewes (about 32.5%), which was maximized one week later (35.8%,
287 $P \leq 0.001$). Figure 2 shows that for eight successive weeks after lameness diagnosis,
288 DMY of lame ewes continued to be significantly lower compared with controls at the
289 $P \leq 0.001$ level.

290 TMYs for non-lame and lame ewes, as calculated by the weekly solutions of
291 DMY produced by model 1, were 318.9 kg and 268.0 kg, respectively (a reduction of
292 about 16% for lame ewes). TMY, for the first 210 days of milking period, was 53.7 kg
293 lower (19.3%) in lame ewes compared with controls ($P < 0.01$, Table 2) (Table 2 near
294 here).

295 **DISCUSSION**

296 This is a follow up study of a previously published work (Gelasakis *et al.*,
297 2010). In the aforementioned study, part of the data from Farm A was forced into a
298 different general linear model in order to calculate the effect of lameness on total milk
299 production; a reduction of about 20% was found. In the present study, a significant
300 decrease in lame ewes' DMY was observed when comparison was made both at flock
301 level on the prospective study and on individual ewe level (nested case-control study).
302 This is not surprising as a reduction on milk yield has also been reported in meat
303 sheep breeds (Winter, 2004) but research is limited.

304 Milk yield in dairy cows has been found to be lower (Rajala-Schultz *et al.*
305 1999; Warnick *et al.* 2001, Bicalho *et al.* 2008), equal (Martin *et al.* 1982) or even
306 higher (Dohoo and Martin, 1984) in cases of lameness. In lame, high yielding cows,
307 although a significant reduction in milk yield is expected, the latter remains at the
308 same or higher levels compared with herd average (Green *et al.* 2002), making rather

309 difficult the accurate assessment of the effect of lameness on milk yield (Huxley,
310 2013). This problem can be overcome by calculating lactation curves of lame cows
311 and comparing them both with the average herd lactation curve and with those of
312 appropriately selected controls (Barkema *et al.* 1994). A similar approach was used in
313 our study.

314 The present study revealed that, DMY reduction started about four weeks
315 before lameness diagnosis. The difference between lame ewes and their controls
316 (Figure 2) became significant the week preceding diagnosis and continued for several
317 weeks after recovery, which is in accordance with the long-term effect of lameness on
318 milk yield in cows as reported by Green *et al.* (2002). This finding provides a
319 reasonable explanation for the significant reduction both on DMY and on TMY
320 observed in lame dairy ewes, even when the duration of lameness is short (less than a
321 week). A loss of 50 kg of milk per lactation represents an income loss of about 45€
322 per case (mean price for sheep milk was 0.9€/kg during the 2013-2014 milking period
323 in Greece). A treatment cost of 15-20€ per case must also be added. Obviously,
324 depending on lameness incidence, losses can add up quickly and represent a
325 significant financial burden for farmers.

326 Most cases of lameness in the present study were attributed to white line
327 abscesses for which the aetiopathology remains unclear (Winter and Arsenos, 2009),
328 although some evidence of genetic influences on the occurrence of white line lesions
329 have recently been reported by Conington *et al.* (2010). Generally, increasing parity
330 and herd size are considered probable risk factors of white line lesions (Barker *et al.*
331 2009); nutrition and other predisposing factors or stressors (for example, inadequate
332 housing conditions) can have a direct effect on milk production but at herd or flock
333 level, these are usually common to all animals. Farm-specific epidemiologic

334 investigation is needed to reveal differences related to management issues that pertain
335 mainly to lame ewes. Moreover, the seasonal pattern of lameness occurrence within
336 dairy sheep flocks needs to be further investigated and specified for the different
337 causes of lameness. This could facilitate hypotheses making procedures regarding
338 possible risk factors associated either with the productive cycle of dairy sheep or with
339 the environmental conditions.

340 Is high milk production predisposing dairy ewes to lameness? The majority of
341 cases in this study were diagnosed during the first four months postlambing, when
342 milk yield was highest. However, due to the seasonal pattern of milk production the
343 high lameness incidence coincided with the season that environmental humidity levels
344 were also high (January to April). This is a major predisposing factor (Gelasakis *et al.*
345 2009) and may confound our results. Results reported in Table 2 imply that ewes
346 selected as controls had higher milk production than the other non-lame ewes; they
347 had an advantage of 53.7 kg of milk over lame ewes in the first 210 days of milking
348 period whereas all non-lame ewes (controls included) had an advantage of 50.9 kg for
349 the entire milking period (34 weeks). This is an indication that high milk production is
350 indeed associated to lameness which is, also, supported by results from research on
351 cows (Oikonomou *et al.* 2013).

352 The partial effects of different causes of lameness on milk yield were not
353 possible to be estimated given the low number of cases per causative agent.
354 Estimating the latter effects forms an interesting research topic for future studies on
355 dairy sheep. However, the notion is that the negative effect of lameness on milk
356 production could be due to the fact that stress and pain result in lower feed
357 consumption. This is considered the major factor associated with decreased milk yield
358 in meat sheep breeds, where, chronic lameness has been proved to have a significant

359 negative effect on body condition (Stewart *et al.* 1984; Marshall *et al.* 1991). Lamé
360 ewes may be underfed at pasture consuming a low quality and quantity of grass. This
361 situation is certainly prevalent in animals covering their nutritional demands partially
362 or totally from grazing. In our study, this scenario doesn't seem viable as the
363 nutritional demands were covered by daily provision of an adequate ration in the shed.
364 On Farm A, the grazing ground was very close to the shed and pasture quality was
365 always very good. In any case, the highest prevalence of lameness and the subsequent
366 reduction in milk yield were mainly observed during the winter months, when ewes
367 didn't graze. A more reasonable hypothesis would be that lame ewes are not able to
368 compete for a place at the feeding trough, which results on the consumption of lower
369 quantity and, eventually, quality of feed. This scenario seems more viable in our case,
370 even though feeding troughs provided, in theory, sufficient space for each ewe. In
371 order to prove it, though, the behavioural pattern of intensively reared lame ewes
372 should be assessed, using observational techniques, which forms an important subject
373 of future research. Lower feed consumption could also result from the presence of
374 inflammatory factors (e.g. cytokines interleukin-1 and interleukin-6); some of these
375 factors are known to cause anorexia in laboratory animals (Harden *et al.* 2008).

376 The fact that milk production is already significantly lower one week prior to
377 lameness diagnosis is a very promising observation. If it is confirmed in future studies
378 it could become the basis for the development of an algorithm that could potentially
379 warn farmers very early in the course of the disease. Similar approaches are already
380 investigated in dairy cows (Machado *et al.* 2011; Van Hertem *et al.* 2013). Of course,
381 automated milk recording is still an exception on dairy sheep farms but they are
382 expected to become more common in the future. Early disease diagnosis is one
383 potential way to justify the investment. In any case, though, the specificity of

384 monitoring milk yield as an early diagnostic tool for the identification of specific
385 diseases needs to be assessed.

386 **CONCLUSION**

387 The results of this study demonstrate an evidence of significant financial
388 losses in dairy sheep due to lameness, which though, need to be accurately estimated
389 in further, more detailed, analyses. A large scale survey should now be considered in
390 order to assess the effects of different causes of lameness, on different breeds of
391 sheep, raised under different environments and management schemes.

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506 **FIGURE LEGENDS**

507 **Figure 1.** Average lactation curves for i) non lame and ii) lame ewes (ewes diagnosed
508 lame at least once across milking period) (95% confidence interval for the mean is
509 used as measure of dispersion).

510 **Figure 2.** Mean DMY and lactation curves (adjusted for stage and number of
511 lactation) of lame and control ewes for the period between four weeks before lameness
512 diagnosis and eight weeks after it.