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Correlated response in body condition and energy mobilisation in rabbits selected for litter size variability

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Short title: Correlated Response in Body Condition in Rabbits

Abstract

A divergent selection experiment on litter size variability (high and low lines) was performed in rabbits over seven generations. The aim of this study was to evaluate the correlated responses to selection in body condition and fat reserves mobilisation. Litter size variability was estimated as phenotypic variance of litter size within female after correcting for the year-season and the parity-lactation status effects. A total of 226 females were used in this study, of which 158 females were used to measure body condition and energy mobilisation. Body condition was measured as body weight and perirenal fat thickness. Females were stimulated with the adrenergic isoproterenol. Mobilisation capacity of fat reserves was measured by the lipolytic potential, defined as the increment in non-esterified fatty acids levels from basal concentration until adrenergic stimulation at mating, delivery and 10 d after delivery

of the second reproductive cycle. Females were classified as survivor or non-survivor when they were culled for sanitary reasons or died before the third kindling. Data were analysed using Bayesian methodology. Survivor females presented higher body weight than the non-survivor females at delivery (238 g, P=1.00) and 10 d after delivery (276 g, P=1.00). They also showed higher perirenal fat thickness at 10 d after delivery (0.62 mm, P=1.00). At delivery, basal non-esterified fatty acids levels (NEFA) was lower in survivor than non-survivor females (-0.18 mmol/l, P=1.00), but their lipolytic potential (∆NEFA) was higher (0.08 mmol/l, P=0.94). Body weight was similar between lines in survivor females. Perirenal fat thickness was lower in the high line than in the low line at delivery (-0.23 mm, P=0.90) and 10 d after delivery (-0.28 mm, P=0.92). The high line exhibited higher NEFA (0.10 mmol/l, P=0.93) and lower ∆NEFA (-0.08 mmol/l, P=0.92) than the low line at delivery. The low line showed a favourable correlated response to selection on body condition and fat reserves mobilisation. In conclusion, the low line selected for litter size variability seems to adapt better to adverse conditions, as it has a greater capacity to mobilise energy reserves at delivery than the high line. Females that adequately manage their body reserves and perform energy mobilisation correctly have a lower risk of dying or being culled.

Keywords: Body reserves, non-esterified fatty acids, perirenal fat thickness, phenotypic variance of litter size, survival

Implications

It is important in livestock production assessing animals in management of their body reserves, in the face of environmental challenges and adverse sanitary conditions.

This study evaluates the correlated response to selection in body reserves management and energy mobilisation of two lines divergently selected for litter size variability. Females selected for litter size homogeneity showed a better adaptation to energy challenges than females selected for litter size heterogeneity by increasing body reserves and mobilised fat reserves at delivery and lactation, leading to females that are more resilient.

Introduction

In commercial rabbit breeding, culling and mortality are important from the production and financial viewpoint (Rosell and de la Fuente, 2016). Rosell and de la Fuente (2009) estimate the mortality or culling females before third delivery by 50%. These females are still growing and they often overlap lactation and gestation. This situation involves high nutritional requirements (Martinez-Paredes et al., 2012). An energy deficit for gestation, lactation and maintenance will have a deteriorating effect on body condition of the females, and will increase the susceptibility to disease or death if reproduction continues under such conditions (Friggens, 2003). Animals balance their energy budget among energy-requirements functions such as reproduction, growth or immune function; therefore, a reduction in mobilisation of their body energy reserves may result in a weaker immune response and consequently in a poorer welfare (Pilorz *et al*., 2005; Amat *et al.*, 2007).

Mobilisation of adipose tissue can be measured by the blood concentration of non-esterified fatty acids (Fortun *et al*., 1994); a negative energy balance is associated with an increase of their levels in blood (Fortun-Lamothe, 2006). This mobilisation changes the animal body condition (Garnsworthy and Wiseman, 2006), which can

affect its survival (Roche *et al*., 2009). Body condition is a common tool for assessing the body energy stores of dams in animal production (review by Chilliard, 1993). Perirenal fat thickness is used to measure body condition in rabbits, as it is their main fat deposit and is highly correlated with animal energy content (Pascual *et al.*, 2000).

In prolific species such as rabbits, variability in litter size during the female's lifespan has been related to disease incidence (García *et al.*, 2012) and immune response (Blasco *et al*., 2018). A divergent selection experiment for litter size variability is currently being carried out in rabbits, with the homogenous line showing 45% lower litter size variability than the heterogeneous line (Blasco *et al*., 2017). The aim of this study was to analyse the correlated response to selection for the lines in body condition and fat reserves mobilisation.

Material and methods

Animals

Animals came from the seventh generation of a divergent selection experiment. The selection criterion was litter size variability at birth. Variability of litter size was estimated as phenotypic variance of litter size at birth within female taking into account all parities, after correcting litter size for the effects of year-season and parity-lactation status (see more details in Blasco *et al*., 2017). A total of 126 and 102 females of the high line (homogeneous) and the low line (heterogeneous) respectively constituted the seventh generation of selection and they were used to estimate response to selection and correlated response in litter size 1st and 2nd parity, and survival rate. Females were classified as survivor or non-survivor when they

were culled for sanitary reasons or died before the third kindling. The causes of culling or mortality were determined by observation and were the following: obstetric disorders, ulcerative podermatitis, diarrohea, mastitis and coryza. Obstetric disorders included the cases of death at delivery, mummified foetuses, prolapse and infertility. Elimination for infertility was considered when a doe had 4 consecutive non-fertile mating or 7 consecutive refusals to the male.

A subset of 82 females from the high line and 76 females from the low line were used to measure body condition and energy mobilisation. Females were primiparous at the beginning of the study.

All animals were kept on the farm at the Miguel Hernández University, Elche (Spain). Rabbits were fed a standard commercial diet (16.5% crude protein, 15.8% fiber, 4% fat, 36% NDF, 18.5% ADF, 12% IDF and 2.400 kcal digestible energy; Cunilactal, Nutreco). Food and water were provided ad libitum. Females were housed in individual cages (37.5 x 33 x 90 cm) under a constant photoperiod of 16 h continuous light: 8 h continuous darkness and controlled ventilation throughout the experiment. They were first mated at 18 wk of age and at 10 d after parturition thereafter. Litters were not standardised.

Traits

Litter size of all parities was recorded. Litter size variability with all parities, after correcting litter size for the effects of year-season and parity-lactation status was estimated for all females of seventh generation.

Body weight, body fat reserves and energy mobilisation were recorded at three different physiological stages; second mating, delivery and 10 d after delivery. Perirenal fat thickness was measured by ultrasound imaging to evaluate body fat reserves, as described by Pascual *et al*. (2004), using Justvision 200 SSA-320A Toshiba ultrasound equipment. Basal non-esterified fatty acids (NEFA) were measured to evaluate energy mobilisation. Lipolytic potential of fat reserves was estimated as the increase of blood non-esterified fatty acids (ΔNEFA) after injection of isoproterenol, an adrenergic agent that increases lipolysis (Therilgaard *et al.*, 2005). Blood was sampled before and 7.5 min after injection of 50 µg of isoproterenol per kg of body weight (Sigma 15627). This time interval and concentration of isoproterenol were established as appropriate by Theilgaard *et al*. (2005) for assessing the lipolytic potential in rabbits. Four ml of blood samples were obtained from the central ear artery early in the morning, before feed was distributed, in order to prevent the effect of feeding, as proposed by Theilgaard *et al*. (2005). The samples were drawn into tubes containing EDTA and centrifuged immediately after sampling (4,000 r.p.m., 4 ºC, 15 min) and plasma was stored at -20ºC until further analysis. Plasma NEFA concentrations were determined using the *in vitro* enzymatic colorimetric methodology prepared by the NEFA test Wako C (Wako Pure Chemical Industries, Ltd, Osaka, Japan). Samples were analysed with a UV spectrophotometer (Hewlett Packard Model 8453), measured at 550 nm. The sensitivity of the assay was 0.01 mmol/L and the intra- and inter assay coefficients of variation were both < 5%.

Statistical Analysis

Litter size variability was analysed using a model with a single group effect with four levels (survivor females at third delivery of the high line and of the low line, and non-survivor females at third delivery of the high line and of the low line). Litter size at 1st parity was analysed using a model with season effect and the same group effect as the former model. Litter size at 2nd parity was analysed with the same model as first parity, including lactation status effect with two levels (lactating and non-lactating at mating). Body weight, perirenal fat thickness, NEFA and ΔNEFA after isoproterenol injection were analysed at second mating, delivery and 10 d after delivery using the same model as litter size at second parity, and repeating the same analyses including the covariate litter size at first parity for traits measured at mating, and litter size at second parity for traits measured at delivery and 10d after delivery. Correlated responses to selection were estimated at the differences between high and low line.

All analyses were performed using Bayesian methodology (Blasco, 2017). Bounded uniform priors were used for all effects. Residuals were a priori normally distributed with mean $\mathbf{0}$ and variance $\mathbf{I}\sigma^2_e$. The prior for the variance was also bounded uniform. Features of the marginal posterior distributions for all unknowns were estimated using Gibbs sampling. The Rabbit program developed by the Institute for Animal Science and Technology (Valencia, Spain) was used for all procedures. We used a chain of 60,000 samples, with a burn-in period of 10,000. Only one out of every 10 samples was saved for inferences. Convergence was tested using the Z criterion of Geweke (Sorensen and Gianola 2002) and Monte Carlo sampling errors were computed using time-series procedures described in Geyer (1992).

Results

The main causes of mortality or culling before the third delivery were obstetric disorders (27%), ulcerative pododermatitis (17%), diarrhoea (15%), mastitis (11%) and coryza (7%). Forty-four percent of the females died or were culled between the last week of gestation and the first week of lactation.

Features of the estimated marginal posterior distribution of the differences between survivor and non-survivor females are presented in table 1. Table 1 offers the probability of these differences being greater than zero if D>0 or lower than zero if D<0. Notice that in Bayesian statistics these probabilities can be in some cases equal or higher than 0.95 even when the confidence intervals at 95% probability include zero (see Blasco, 2017). Also notice that in a Bayesian context there is no "significance", but the actual probabilities of the differences being higher or lower than zero are estimated instead.

Litter size at 1st parity was higher in survivor females than in non-survivor females (D=0.50; P=0.91). No differences were found for litter size at 2nd parity. Survivor females presented higher body weight at delivery and at 10 d after delivery, around 0.6 SD for both traits (P=1.00). We observed similar perirenal fat thickness at mating and delivery in both females, but at 10 d after delivery survivor females showed a large difference (0.6 SD of this trait, P=1.00). At delivery, a substantial difference between survivor and non-survivor females was found for NEFA (0.6 SD of this trait, -0.18 mmol/l, P=1.00, Table 2). However, the difference for ΔNEFA was lower (0.2 SD of this trait, 0.08 mmol/l, P=0.94). Similar results were obtained when the covariate litter size was included in the analyses (data not shown).

Table 3 summarises the features of marginal posterior distributions of the differences between lines for litter size variability and litter size at 1st and 2nd parity. As the environmental effects are the same for both lines, the differences between lines (D) are genetic differences, so they estimate the response and correlated responses to selection. Response to selection was obtained in the 7th generation. The high line showed higher litter size variability than the low line for both survivor (D=1.33; P=0.99) and non-survivor females (D=1.65; P=0.97). Survival rate did not have a correlated response to selection (38/88 vs 29/73; $P(\chi^2)$=0.53). Survivor females from both lines showed similar litter size at 1st parity, but the high line showed higher litter size at 2nd parity than the low line (D=−0.89, P=0.94). Non-survivor females of the high line showed higher litter size in both parities than the low line.

Survivor females from both lines had similar body weight at all stages (Table 3). At mating, perirenal fat thickness was also similar in both lines, but the high line showed lower perirenal fat thickness than the low line at delivery (-0.23 mm, P=0.90) and this difference was consolidated 10 d after (-0.28 mm, P=0.92). This difference was moderate (around 0.3 SD of this trait). At delivery, the difference between lines was also moderate for NEFA (0.3 SD of this trait, 0.10 mmol/l, P=0.93; Table 4) and low for ΔNEFA (0.2 SD of this trait, -0.08 mmol/l, P=0.92). No differences in the high and low lines were found at mating and 10 d after delivery for either trait. Non-survivor females of both lines showed similar body condition and NEFA (Tables 3 and 4). Lipolytic potential (ΔNEFA) was higher in the high line at mating and 10 d after delivery (0.26 mmol/l and 0.14 mmol/l respectively), and lower at delivery (-0.14 mmol/l, P=0.92) than in the low line. These differences were relevant (between 0.4 and 0.7 SD of these

traits). Similar results were obtained when the covariate litter size was included (data not shown), except for body weight at mating in non-survivor females (D=-145; $HPD_{95\%}$= -370, 65; P=0.90) and ∆NEFA at mating in survivor females (D=0.11; $HPD_{95\%}$ = -0.02, 0.25; P=0.95).

Discussion

Response to selection was obtained in the 7th generation, agreeging with the results of the whole experiment (Blasco et al., 2017), and correlated responses are expected. Regardless of the line, around 30% of the females were non-survivor before the third delivery. The highest mortality in the females occurred during the last week of pregnancy and the 1st seven days of lactation, which agrees with Rosell and De la Fuente (2016). We used body weight and perirenal fat thickness as indicators of body condition, NEFA as indicators of actual energy mobilisation, and ∆NEFA as indicator of lipolytic potential, following Theilgaard *et al*. (2006). Both body condition and energy mobilisation showed how the rabbits prioritise their energy reserves. Immediately after delivery, milk production is low and feed intake is sufficient to cover the nutritional needs for both maintenance and lactation (Feugier and Fortun-Lamothe, 2006), so the females tend to increase their body reserves between delivery and early lactation (Theilgaard *et al.*, 2009). Non-survivor females showed poorer body condition, higher energy mobilisation and less lipolytic potential than survivor females when the doe needs to manage its energy reserves, i.e. at delivery. The reduction in body condition is associated with diseases (Bareille *et al*., 2003), as the immune system in sick animals has greater nutrient requirements (Johnson, 1998).

Delivery and lactation are stressful stages for female mammals (Gellrich *et al.,* 2015). Several studies have reported that stress negatively affects the immune system, and hence disease susceptibility (see review by Webster-Marketon & Glaser, 2008). Stress also has a negative effect on resource allocation and body condition (Broom, 2008). Because of this, body condition has been proposed as an indicator for animal health and welfare (Blache *et al.,* 2011). Our results show that selection for litter size homogeneity in survivor females led in the low line to higher deposition of fat reserves at delivery and 10 days after delivery than in the high line. After injection of the adrenergic agent, lipolytic potential (∆NEFA) was higher in the homogeneous line at delivery. Survivor females from the homogeneous line presented greater perirenal fat thickness and ΔNEFA but, interestingly, they presented lower NEFA at second delivery. In addition, the energy challenge was higher in the low line than in the high line, since low line reared one kit more. This situation would suggest that this line has a greater amount of body reserves which can be used if required; however, they did not use these extra-reserves at delivery. Savietto *et al.* (2013) argued that females following this strategy safeguard body reserves to cope with future reproduction and longevity. In this sense, results from the 8^{th} to the 10^{th} generation show 12% lower involuntary elimination rate in the low line than in the high line (Argente *et al.*, 2018).

When body reserves, energy mobilisation and lipolytic potential were measured in non-survivor females, both lines showed similar body weight, perirenal fat thickness and NEFA throughout the second reproductive cycle. However, lipolytic potential was different between lines, showing the low line higher ∆NEFA than the high line at delivery. New research should be carried out to determine the different causes of the differences found in lipolytic potential. We conclude that a correlated response in

female body condition and fat mobilisation was obtained when selecting for litter size variability. The does selected for litter size homogeneity would be able to better deal with situations of high-energy demand than does with higher litter size variability, which should lead to higher health and welfare levels.

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Declaration of interest

The authors declare that they have no competing interests.

Ethics statement

All experimental procedures involving animals were approved by the Miguel Hernández University of Elche Research Ethics Committee (Reference number DTA-MJA-001-11), in accordance with Council Directives 98/58/EC and 2010/63/EU.

Software and data repository resources

Data and software are available upon request to the corresponding author.

References

Amat JA, Aguilera E and Visser GH 2007. Energetic and developmental costs of mounting an immune response in greenfinches (Carduelis chloris). Ecological Research 22, 282-287.

300 Bareille N, Beaudeau F, Billon S, Robert A and Faverdin P 2003. Effects of health disorders
301 on feed intake and milk production in dairy cows. Livestock Production Science 83,
302 53-62.
303 Blache D, Terlouw C and Maloney SK 2011. Physiology. In Animal Welfare (ed. MC Appleby,
304 BO Hughes, A Joy and JA Mench), pp. 155-182. CAB International, Wallingford, UK.
305 Blasco A. 2017. Bayesian data analysis for animal scientists. Springer. New York.
306 Blasco A, Martínez-Álvaro M, Garcia ML, Ibáñez-Escriche N and Argente MJ 2017. Selection
307 for environmental variance of litter size in rabbits. Genetic Selection Evolution 49:48.
308 Blasco A, Martínez-Álvaro M, Garcia ML, Capcarova M, Zbynovska K, Petruska P, Ibáñez-
309 Escriche N and Argente MJ 2018. Selection for genetic environmental sensitivity of
310 litter size changes resilience in rabbits. 11th World Congress on Genetics Applied to
311 Livestock Production. 11-16 February 2018. Auckland, New Zealand.
312 Broom MD 2009. Consequences of biological engineering for resource allocation and
313 welfare. In Resource allocation theory applied to farm animal production (ed. WM
314 Rauw), pp. 261-274. CAB International. Wallingford (UK).
315 Carenzi C and Verga M 2009. Animal welfare: Review of the scientific concept and definition.
316 Italian Journal Animal Science 8, 21–30.
317 Chilliard Y 1993. Dietary fat and adipose tissue metabolism in ruminants, pigs and rodents: A
318 review. Journal Dairy Science 76, 3897-3931.
319 Feugier A, Fortun-Lamothe L 2006. Extensive reproductive rhythm and early weaning
320 improve body condition and fertility of rabbit does. Animal Research 55,459-470.
321 Fortun L, Prunier A, Etienne M and Lebas F 1994. Influence of nutritional deficit on foetal
322 survival and growth and blood metabolites in rabbit does. Reproduction, Nutrition,
323 Development 34, 201-211.
324 Fortun-Lamothe L 2006. Energy balance and reproductive performance in rabbits does.
325 Animal Reproduction Science 93, 1-15.

326 Friggens NC 2003. Body lipid reserves and reproductive cycle: towards a better
327 understanding. Livestock Production Science 83, 219-236.
328 García ML, Argente MJ, Muelas R, Birlanga V and Blasco A 2012. Effect of divergent
329 selection for residual variance of litter size on health status and welfare. Proceedings
330 of the 10th World Rabbit Congress, 3-6 September 2012, Sharm El-Sheikh (Egypt),
331 pp. 103-106.
332 Garnsworthy PC and Wiseman J 2006. Recent advances in animal nutrition. Nottingham
333 University Press, 61-86
334 Geyer CM 1992. Practical markow chain Monte Carlo (with discussion). Statistical Science 7,
335 467-511.
336 Gellrich K, Sigl T, Mayer HHD and Wiedemann S 2015. Cortisol levels in skimmed milk
337 during the first 22 weeks of lactation and response to short-term metabolic stress and
338 lameness in dairy cows. Journal of Animal Science and Biotechnology 6, 31-38.
339 Johnson RW 1998. Immune and endocrine regulation of food intake in sick animals.
340 Domestic Animal Endocrinology 15, 309-319.
341 Martinez-Paredes E, Ródenas L, Martínez-Vallespín B, Cervera C, Blas E, Brecchia G, Boiti
342 C and Pascual JJ 2012. Effects of feeding programme on the performance and
343 energy balance of nulliparous rabbit does. Animal 6, 1086-1095.
344 Pascual JJ, Castella F, Cervera C, Blas E and Fernández-Carmona J 2000. The use of
345 ultrasound measurement of perirenal fat thickness to estimate changes in body
346 condition of young female rabbits. Animal Science 70, 435-442.
347 Pascual JJ, Blanco J, Piquer O, Quevedo F and Cervera 2004. Ultrasound measurements of
348 perirenal fat thickness to estimate the body condition of reproducing rabbit does in
349 different physiological status. World Rabbit Science 12, 7-22.
350 Pilorz V, Jäckel M, Knudsen K and Trillmich F 2005. The cost of a specific immune response
351 in young guinea pigs. Physiology & Behavior 85, 205-211.
352 Prunier A, Heinonen M and Quesnel H 2010. High physiological demands in intensively raised
353 pigs: impact on health and welfare. Animal 4, 886–898.

354 Rauw WM 2009. Resource allocation theory applied to farm animal production. CAB
355 International, Wallingford (UK).
356 Roche JR, Friggens NC, Kay JK, Fisher MW, Stafford KJ and Berry DP 2009. Invited review:
357 Body condition score and its association with dairy cow productivity, health, and welfare.
358 Journal Dairy Science 92, 5769-5801.
359 Rosell JM and de la Fuente LF 2009. Culling and mortality in breeding rabbits. Preventive
360 Veterinary Medicine 88, 120-127.
361 Rosell JM and de la Fuente LF 2016. Causes of mortality in breeding rabbits. Preventive
362 Veterinary Medicine 127, 56-63.
363 Savietto D, Cervera C, Blas E, Baselga M, Larsen T, Friggens NC and Pascual JJ 2013.
364 Environmental sensitivity differs between rabbit lines selected for reproductive intensity
365 and longevity. Animal 7, 1969-1977.
366 Sorensen D and Gianola D 2002. Likelihood, bayesian, and MCMC methods. Quantitative
367 genetics. 1st Edition. Springer-Verlag, New York (USA).
368 Theilgaard P, Facila S, Blas E, Baselga M and Pascual JJ 2005. Time and dose response of
369 blood non-esterified fatty acids to adrenergic stimulation in rabbit does. World Rabbit
370 Science 13, 189-195.
371 Theilgaard P, Sánchez JP, Pascual JJ, Friggens NC and Baselga M 2006. Effect of body
372 fatness and selection for prolificacy on survival of rabbit does assessed using a
373 cryopreserved control population. Livestock Science 103, 65-73.
374 Theilgaard P, Baselga M, Blas E, Friggens NC, Cercera C and Pascual JJ 2009. Differences in
375 productive robustness in rabbits selected for reproductive longevity or litter size. Animal
376 3, 637-646.
377 Webster-Marketon JI and Glaser R 2008. Stress hormones and immune function. Cell
378 Immunology 252 (1-2),16-26.

Table 1 *Features of the marginal posterior distribution of the differences for litter size, body weight and perirenal fat thickness between females that survive at third delivery and non-survivor does.*

Trait	S	NS	D_{S-NS}	$HPD_{95\%}$	P	SD
Litter Size at 1st parity	7.47	6.97	0.50	-0.25, 1.18	0.91	2.53
Litter Size at 2nd parity	7.09	7.21	-0.11	-1.22,0.84	0.42	3.65
Body Weight (g)						
Mating	3632	3526	107	-77 , 228	0.80	378
Delivery	3491	3252	238	59 , 383	1.00	415
10 d after Delivery	3574	3300	276	129 , 485	1.00	458
Perirenal Fat Thickness (mm)						
Mating	9.40	9.51	-0.11	-0.42 , 0.29	0.70	0.85
Delivery	9.20	9.05	0.15	-0.19 , 0.65	0.75	0.96
10 d after Delivery	9.33	8.71	0.62	0.20 , 0.99	1.00	1.05

S = survivor females; NS = non-survivor females; D_{S-NS} = median of the difference between survivor and non-survivor does; $HPD_{95\%}$ = highest posterior density region at 95%; P = probability of the difference being >0 when $D_{S-NS} > 0$ and being < 0 when $D_{S-NS} < 0$; SD = standard deviation.

Table 2 *Features of the marginal posterior distribution of the differences for basal non-esterified fatty acids (NEFA) and lipolytic potential of fat reserves (∆NEFA) between survivor and non-survivor does at third delivery, measured at second mating, delivery and 10 d after delivery*

Trait	S	NS	D_{S-NS}	$HPD_{95\%}$	P	SD
NEFA (mmol/l)						
Mating	0.51	0.54	-0.03	-0.10 , 0.11	0.57	0.25
Delivery	0.61	0.79	-0.18	-0.32 , -0.05	1.00	0.31
10 d after Delivery	0.56	0.50	0.06	-0.06 , 0.15	0.78	0.21
∆NEFA (mmol/l)						
Mating	0.36	0.31	0.05	-0.05, 0.19	0.88	0.39
Delivery	0.39	0.31	0.08	-0.02 , 0.20	0.94	0.34
10 d after Delivery	0.28	0.23	0.05	-0.07 , 0.17	0.77	0.33

S = survivor females; NS = non-survivor females; D_{S-NS} = median of the difference between the survivor and non-survivor does; $HPD_{95\%}$ = highest posterior density region at 95%; P = probability of the difference being >0 when $D_{S-NS} > 0$ and probability of the difference being < 0 when $D_{S-NS} < 0$; SD = standard deviation.

Table 3 *Features of the marginal posterior distribution of the differences for litter size variability, litter size at 1st and 2nd parity, body weight and perirenal fat thickness between the high and the low litter size variability lines*

	Survivor Females					Non-survivor Females				
Trait	High line	Low line	D_{H-L}	$HPD_{95\%}$	P	High line	Low line	D_{H-L}	$HPD_{95\%}$	P
N	88	73				38	29			
Litter size variability	4.64	3.27	1.33	0.22,2.51	0.99	3.27	1.62	1.65	-0.02, 3.54	0.97
Litter size 1st parity	7.37	7.55	-0.18	-0.95,0.54	0.67	6.15	7.97	-1.79	-2.97,-0.59	1.00
Litter Size 2nd parity	6.63	7.53	-0.89	-2.00,0.22	0.94	6.26	8.26	-1.99	-3.66, -0.12	0.99
N	57	55				25	21			
Body Weight (g)										
Mating	3638	3629	7	-127, 152	0.54	3512	3541	-28	-302 , 245	0.57
Delivery	3473	3530	-57	-202, 100	0.81	3334	3241	91	-206 , 386	0.73
10 d after Delivery	3543	3607	-65	-229, 97	0.83	3330	3270	59	-266 , 361	0.65
Perirenal Fat Thickness (mm)										
Mating	9.41	9.40	0.01	-0.29 ,0.32	0.52	9.45	9.57	-0.12	-0.62 , 0.35	0.70
Delivery	9.08	9.31	-0.23	-0.60 ,0.12	0.90	8.94	9.18	-0.24	-0.91 , 0.47	0.75
10 d after Delivery	9.19	9.47	-0.28	-0.64 ,0.11	0.92	8.75	8.67	0.08	-0.63 , 0.80	0.59

D_{H-L} = median of the difference between the high and the low lines; $HPD_{95\%}$ = Highest posterior density region at 95%; P = probability of the difference being >0 when $D_{H-L} > 0$ and probability of the difference being < 0 when $D_{H-L} < 0$.

Table 4 *Features of the marginal posterior distribution of the differences for basal non-esterified fatty acids (NEFA) and lipolytic potential of fat reserves (∆NEFA) between the high and the low litter size variability lines*

	Survivor Females					Non-survivor Females				
Trait	High line (n=57)	Low line (n=55)	D_{H-L}	$HPD_{95\%}$	P	High line (n=25)	Low line (n=21)	D_{H-L}	$HPD_{95\%}$	P
NEFA (mmol/l)										
Mating	0.52	0.51	0.01	-0.10 , 0.13	0.58	0.51	0.58	-0.07	-0.24 , 0.12	0.80
Delivery	0.65	0.55	0.10	-0.04 , 0.24	0.93	0.73	0.84	-0.11	-0.33 , 0.10	0.85
10 d after Delivery	0.55	0.56	0.00	-0.09 , 0.10	0.50	0.55	0.44	0.11	-0.08 , 0.29	0.87
∆NEFA (mmol/l)										
Mating	0.39	0.32	0.07	-0.0.6 , 0.20	0.87	0.44	0.18	0.26	0.07 , 0.47	1.00
Delivery	0.35	0.44	-0.08	-0.20 , 0.03	0.92	0.25	0.38	-0.14	-0.32 , 0.05	0.92
10 d after Delivery	0.29	0.29	0.01	-0.10 , 0.12	0.58	0.32	0.18	0.14	-0.08 , 0.33	0.90

D_{H-L} = median of the difference between the high and the low lines; $HPD_{95\%}$ = Highest posterior density region at 95%; P = probability of the difference being >0 when $D_{H-L} > 0$ and probability of the difference being < 0 when $D_{H-L} < 0$.