Herd specific effects of the *ESR* gene on litter size and production traits in Czech Large White sows

E. Goliášová[1], J. Wolf[2]

[1]Department of Genetics, Mendel University of Agriculture and Forestry, Brno, Czech Republic
[2]Department of Genetics and Biometrics, Research Institute of Animal Production, Prague-Uhříněves, Czech Republic

ABSTRACT: The effect of *Pvu*II polymorphism of the oestrogen receptor gene on litter size and production traits in a Czech Large White population (882 sows, 2 455 litters) was evaluated. Data were analysed by four four-trait animal models. The traits analysed were lifetime average daily gain in the field test, lean meat percentage, number of piglets born alive in parity 1 and number of piglets born alive in parity 2 and subsequent parities. The animal models differed in considering the herd-year-season effect as random or fixed and in including or not including the dominance effect. For each model two variants were calculated: the *ESR* effects were calculated either across herds or within herds. On the genetic level, no significant overall effects of the *ESR* gene were detected. The effects of the *ESR* gene within herds were mostly insignificant as well. There was a certain tendency for allele *A* to increase litter size in parity 1. Nevertheless, on the basis of the present knowledge, the use of the *ESR* gene for selection for litter size in the Czech Large White breed seems to be of questionable benefit and cannot be recommended.

Keywords: pig; reproduction; litter size; candidate gene; *ESR*

For modern pork production it is essential to maintain high levels of reproductive performance (Rothschild *et al.*, 1996). Though reproductive traits such as litter size have a low heritability, the consequent use of animal models in combination with progress in computer technology makes a successful selection for this trait possible. Further acceleration of the genetic progress might be expected by incorporating marker-assisted selection methods into breeding programs. This will first require identification of candidate genes or anonymous genetic markers associated with the traits of interest.

Described associations between markers and reproductive traits have recently been overviewed by several authors (Bidanel and Rothschild, 2002; Rohrer, 2004). It has been reported that *Pvu*II polymorphism of the oestrogen receptor (*ESR*) gene is associated with litter size in several populations. In U.S. and European Meishan synthetics and in Large White populations, the *ESR* gene was shown to contribute by a significant proportion to litter size variation (Rothschild *et al.*, 1995; Southwood *et al.*, 1995; Short *et al.*, 1997). Estimates of allelic effects ranged in these papers from 1.43 piglets/litter in Meishan synthetics to 0.42 piglets/litter in Large White lines, both per copy of the *B* allele. Although many studies indicate that *B* allele is the favourable allele for litter size, other studies show no effect or even indicate that the allele of favour is allele *A* (Van der Lende *et al.*, 2002).

The effect of *Pvu*II polymorphism of the oestrogen receptor gene on litter size and production traits in a Czech Large White population (1 250 sows, 3 600 litters) was evaluated recently (Goliášová and Wolf, 2004). The *ESR* gene showed on average mostly a significant effect ($P < 0.05$) on litter size traits in favour of allele *A*.

As there are different indications on the favourable allele of the *ESR* gene in the literature, the aim of the present study was to investigate to what extent the effect of the *ESR* gene is herd specific.

Supported by the National Agency of Agricultural Research, Ministry of Agriculture of the Czech Republic (Project QD 1 039).

MATERIAL AND METHODS

The investigation was based on a data set on 882 Czech Large White sows from 10 herds. In all herds, the minimal number of genotyped sows was 40 and each of the three possible *ESR* genotypes was presented by 6 animals at least. The following traits were analysed:

ADG – lifetime average daily gain in field (in g/day) calculated as weight at the end of test divided by age at the end of test. The weight at the end of test ranged from 70 to 110 kg.

LM – lean meat percentage in the field test (in %) calculated from ultrasonic measurements without any live weight pre-adjustment.

NBA1 – number of piglets born alive in parity 1.

NBA2+ – number of piglets born alive in parity 2 and subsequent parities.

All animals were genotyped for the porcine oestrogen receptor gene (*ESR*). *Pvu*II polymorphism was analysed by the PCR-RFLP procedure as described in Short *et al.* (1997). In the presence of *B* allele an amplified 120 bp long PCR product was cut into 55 bp and 65 bp long fragments while in the presence of allele *A* the product remained intact.

Basic statistical characteristics of the data set are summarized in Table 1 for all traits and all *ESR* genotypes within traits. In the first analysis, simple phenotypic means and standard errors were calculated for the individual *ESR* genotypes in all herds. The approximate *t*-test according to Welch (1947) was used for the statistical evaluation of differences between means for *ESR* genotypes within herds.

The analysis of phenotypic means was complemented by more sophisticated procedures. Four four-trait animal models were calculated. The factors included in the models are summarized in Table 2. The four models differed by considering the herd-year-season effect as random (Models 1 and 3) or fixed (Models 2 and 4) and by including (Models 3 and 4) or not including (Models 1 and 2) the dominance effect of *ESR*. Two variants of each model were calculated. In the first variant, the additive and the dominance effect of the *ESR* gene were calculated as effects averaged across herds. In the second variant, these effects were estimated within herds. The additive effect was defined as *B-A*, where *B* and *A* were the effects of the appropriate alleles. The difference between *BB* and *AA* sows is therefore twice the additive effect. The dominance effect was

Table 1. Number of observations, means and their standard errors for individual genotypes within traits

Trait	Genotype	*n*	Mean	Std. error
Lean meat percentage (%)	AA	218	61.34^{c}	0.123
	AB	434	61.47^{a}	0.093
	BB	230	61.81^{b,d}	0.119
		882	61.53	0.063
Average daily gain from birth to test end in the field test (g/d)	AA	218	630.6	3.76
	AB	434	632.0	2.81
	BB	230	634.8	3.96
		882	632.4	1.96
Number of piglets born alive in parity 1	AA	181	12.33	0.163
	AB	367	12.01	0.114
	BB	164	12.05	0.158
		712	12.10	0.081
Number of piglets born alive in parity 2 and subsequent parities	AA	547	12.64	0.098
	AB	870	12.69	0.079
	BB	326	12.62	0.132
		1 743	12.66	0.056

Significance of differences: $^{a,b}P < 0.05$, $^{c,d}P < 0.01$

Table 2. Used four-trait animal models (when there are differences between models, the numbers of models the specified option is applied to are given in parentheses)

Effect	Type of effect for trait			
	ADG	LM	NBA1	NBA2+
Additive effect of ESR (averaged across herds or within herd)	C	C	C	C
Dominance effect of ESR (averaged across herds or within herd)	–(1,2) C (3,4)	–(1,2) C (3,4)	–(1,2) C (3,4)	–(1,2) C (3,4)
Year-season for production traits	F	F	–	–
Type of feeding (ad libitum or restricted) in test	F	F	–	–
Herd	R	R	–	–
Litter	R	R	–	–
Live weight at the end of test	–	C	–	–
Age at 1st farrowing linear and squared	–	–	C	–
Farrowing interval linear and squared	–	–	–	C
Mating type (AI or natural mating)	–	–	F	F
Boar breed	–	–	F	F
Parity	–	–	–	F
Herd-year-season for reproduction	–	–	R (1,3) F (2,4)	R (1,3) F (2,4)
Permanent effect of sow	–	–	–	R
Animal	A	A	A	A

Traits:
ADG – average daily gain from birth to test end, LM – lean meat percentage, NBA1 – number of piglets born alive in parity 1, NBA2+ – number of piglets born alive in parity 2 and subsequent parities
Types of effects:
C – covariable, F – fixed effect, R – random effect, A – random effect with relationship matrix

defined as *AB*–(*AA* + *BB*)/2, where *AA*, *AB* and *BB* were the appropriate genotypes of the *ESR* gene. The main reason for using different models was to increase the validity of the results by excluding the chance that a certain result was caused just by a concrete model.

The models were identical to a great extent with the model used for these traits in routine genetic evaluation of maternal pig breeds in the Czech Republic (Peškovičová *et al.*, 2002). All calculations were carried out by the PEST program (Groeneveld *et al.*, 1992). The genetic parameters used were the same as in routine genetic evaluation of the Czech Large White breed (Wolf *et al.*, 1999). The SMP solver was used to get fixed effect results with exact standard errors.

RESULTS

First the phenotypic means averaged across herds will be considered (Table 1). The *BB* sows outperformed the *AA* sows by nearly 0.5% lean meat. The *AB* sows showed a similar performance like the *AA* sows. The difference between *BB* sows and *AA* sows in average daily gain was as small as 4 g/day. In the number of piglets born alive in parity 1, there was a certain tendency in favour of the *AA* sows, whereas in the number of piglets born alive in parity 2 and subsequent parities, the performance of all three *ESR* genotypes was nearly equal.

When considering the phenotypic means of the production traits for the *ESR* genotypes in individual herds (Table 3), a tendency of better performance

Table 3. Number of observations, means and standard errors of means for production traits of the individual genotypes within herds

Herd	*n*			Mean ± std. error for *ESR* genotypes		
	AA	*AB*	*BB*	*AA*	*AB*	*BB*
Lean meat percentage (%)						
1	9	29	10	61.11 ± 0.61	60.81 ± 0.41	60.42 ± 0.46
2	31	54	17	61.98 ± 0.28	62.51 ± 0.27	62.71 ± 0.52
3	27	43	33	**61.25 ± 0.33^{a,c}**	**62.18 ± 0.29^{b}**	**62.72 ± 0.26^{d}**
4	25	42	10	61.43 ± 0.26	60.96 ± 0.19	60.58 ± 0.64
5	11	41	38	60.95 ± 0.61	**60.96 ± 0.29^{a}**	**62.00 ± 0.29^{b}**
6	12	25	7	59.85 ± 0.75	61.02 ± 0.31	60.96 ± 0.70
7	12	43	36	61.84 ± 0.55	62.37 ± 0.33	62.09 ± 0.27
8	19	39	23	61.35 ± 0.38	60.68 ± 0.26	61.29 ± 0.32
9	930	32	9	61.26 ± 0.28	61.28 ± 0.24	61.32 ± 0.39
10	42	86	47	61.34 ± 0.33	61.31 ± 0.21	61.50 ± 0.27
Average daily gain (g/day)						
1	9	29	10	628.9 ± 17.63	627.6 ± 10.45	627.1 ± 15.02
2	31	54	17	**609.2 ± 10.43^{a}**	**636.4 ± 8.17^{b}**	616.8 ± 11.19
3	27	43	33	666.1 ± 8.36	658.1 ± 6.41	660.2 ± 7.80
4	25	42	10	648.0 ± 7.07	665.3 ± 7.23	664.4 ± 16.52
5	11	41	38	660.0 ± 9.72	657.9 ± 9.06	655.9 ± 8.63
6	12	25	7	596.8 ± 12.81	587.0 ± 9.73	612.4 ± 17.04
7	12	43	36	**609.8 ± 4.53^{a}**	**590.1 ± 8.52^{b}**	**580.2 ± 10.78^{b}**
8	19	39	23	**569.0 ± 13.71^{a}**	591.7 ± 10.37	**620.3 ± 15.05^{b}**
9	30	32	9	631.4 ± 10.88	615.6 ± 7.74	610.2 ± 12.58
10	42	86	47	649.0 ± 7.49	647.3 ± 4.89	658.6 ± 6.51

Significance of differences within rows: $^{a,b}P < 0.05$, $^{c,d}P < 0.01$

connected with allele *B* was evident for lean meat percentage. In seven out of ten herds the *BB* genotype showed a higher value than the *AA* genotype, but the difference was significant only in one herd. In average daily gain the situation was more balanced, exactly in one half of the herds *AA* sows showed slightly higher values than *BB* sows. The difference was significant in two herds, once in favour of the *AA* sows and once in favour of the *BB* sows.

The phenotypic means for the *AB* sows for lean meat percentage were intermediary in six herds, higher than the values of both homozygotes in two herds and lower than the values for homozygotes in two herds too. In average daily gain, these situations occurred five, two and three times, respectively. Therefore, no clear conclusions can be drawn.

The average positive effect of *AA* genotype on the number of piglets born alive in parity 1 was confirmed in six herds, in the remaining four herds the value for the *BB* sows was better (Table 4). Only in one herd was a significant difference in favour of the *AA* sows observed. The situation for the second reproductive trait, the number of piglets born alive in parity 2 and subsequent parities, was balanced. Better values for *AA* sows or *BB* sows occurred each in five herds, whereby the differences were significant twice in favour of the *AA* sows and once in favour of the *BB* sows.

Table 4. Number of observations, means and standard errors of means for reproductive traits of individual genotypes within herds

Herd	*n*			Mean ± std. error for *ESR* genotypes		
	AA	*AB*	*BB*	*AA*	*AB*	*BB*
Number of piglets born alive in parity 1						
1	8	26	10	14.38 ± 0.73	12.81 ± 0.66	13.30 ± 0.60
2	28	43	14	12.64 ± 0.25	12.84 ± 0.24	12.50 ± 0.51
3	24	38	23	13.04 ± 0.39	13.21 ± 0.29	13.61 ± 0.37
4	24	38	8	11.58 ± 0.63	11.82 ± 0.45	11.25 ± 0.82
5	8	27	20	12.00 ± 0.50	11.85 ± 0.27	12.10 ± 0.24
6	9	23	6	10.33 ± 0.71	11.43 ± 0.42	8.83 ± 1.22
7	7	28	25	11.29 ± 0.61	10.82 ± 0.42	11.36 ± 0.35
8	13	31	14	13.08 ± 0.58	11.68 ± 0.35	12.50 ± 0.39
9	22	30	8	11.59 ± 0.31	11.30 ± 0.23	12.13 ± 0.35
10	38	83	36	**12.61 ± 0.37**^{a}	11.88 ± 0.23	**11.50 ± 0.33**^{b}
Number of piglets born alive in parity 2 and subsequent parities						
1	21	66	23	13.48 ± 0.58	13.24 ± 0.35	12.52 ± 0.53
2	49	61	12	13.16 ± 0.22	13.16 ± 0.17	13.50 ± 0.45
3	86	65	40	**13.63 ± 0.19**^{c,}	**13.86 ± 0.22**^{a}	**14.68 ± 0.21**^{d,b}
4	113	146	21	12.07 ± 0.27	12.08 ± 0.21	12.05 ± 0.57
5	7	44	23	**11.86 ± 0.34**^{a}	**13.05 ± 0.28**^{b}	**12.22 ± 0.27**^{a}
6	42	66	19	**12.64 ± 0.39**^{c}	**12.15 ± 0.38**^{a}	**10.53 ± 0.81**^{d,b}
7	15	74	60	**15.20 ± 0.76**^{a,c}	**13.14 ± 0.29**^{b,c}	**12.32 ± 0.29**^{d}
8	44	123	20	**12.27 ± 0.35**^{a}	**13.13 ± 0.19**^{b}	12.45 ± 0.39
9	89	81	24	11.61 ± 0.12	11.32 ± 0.14	11.13 ± 0.26
10	81	144	84	12.75 ± 0.24	12.63 ± 0.19	12.94 ± 0.27

Significance of differences within rows: $^{a,b}P < 0.05$, $^{c,d}P < 0.01$

Table 5 shows the estimates of additive and dominance effects of the *ESR* gene on all four traits investigated as averaged across all herds. There was a good agreement between the results for all four models, only in the number of piglets born alive in parity 2 and subsequent parities there were larger differences in the estimates of the additive effect. No significant effect of the *ESR* gene on the population level could be detected when assuming a significance level of 5% or lower. In the number of piglets born alive in parity 1, the additive effect indicating better performance for the *A* allele was significant on the 10% level for Models 1 and 3 and was also very close to the 10% significance level for Models 2 and 4. The observed phenotypic effect of *ESR* on lean meat percentage could not be confirmed on the genetic level.

In the results referring to individual herds, there was a good agreement in the estimates of the additive effect between Models 1 and 2 and between Models 3 and 4 in production traits. In reproductive traits, the best agreement was between Models 1 and 3 and Models 2 and 4. Therefore, the results are only presented for Models 1 and 3 in production traits and for Models 3 and 4 for reproductive traits.

The additive and dominance effects for production traits (Table 6) did not differ significantly from zero with one exception (additive effect for average daily gain in herd 8) and it seems that they were more or less equally distributed around zero, which

Table 5. Estimates of additive and dominance effects of the *ESR* locus for production and reproductive traits averaged across herds

	Additive effect ± std. error	Dominance effect ± std. error
Lean meat percentage		
Model 1	–0.001 ± 0.1168	
Model 2	–0.002 ± 0.1168	
Model 3	–0.001 ± 0.1168	0.061 ± 0.1394
Model 4	–0.002 ± 0.1168	0.058 ± 0.1394
Average daily gain		
Model 1	3.10 ± 2.971	
Model 2	3.08 ± 2.971	
Model 3	3.10 ± 2.971	1.32 ± 3.767
Model 4	3.08 ± 2.971	1.30 ± 3.767
Number of piglets born alive in parity 1		
Model 1	–0.173 ± 0.1024	–
Model 2	–0.185 ± 0.1173	–
Model 3	–0.169 ± 0.1025	–0.131 ± 0.1330
Model 4	–0.180 ± 0.1174	–0.179 ± 0.1480
Number of piglets born alive in parity 2 and subsequent parities		
Model 1	–0.072 ± 0.0827	–
Model 2	–0.027 ± 0.0875	–
Model 3	–0.079 ± 0.0834	0.065 ± 0.1068
Model 4	–0.038 ± 0.0884	0.090 ± 0.1103

[1] See Table 2 for the definition of Models 1 to 4

means there was no clear preference for positive or negative values.

The herd-specific effects of the *ESR* gene on reproductive traits are listed in Table 7. Only a limited number of significant effects was detected. There was a tendency to negative values in the additive effects indicating that allele *A* could be more favourable for litter size than allele *B*. But this tendency was not unambiguous. In each model, one (parity 1) or two (parity 2) significant negative additive effects were estimated; for the number of piglets born alive in parity 2 there was also one significant positive additive effect.

The estimates of dominance effects were distributed around zero, being positive in about 50% of all cases. In the number of piglets born alive in parity 1, the dominance effect differed significantly from zero only in one herd; in the same trait for parity 2 and subsequent parities, significant effects were calculated for two herds.

In some herds, relatively marked differences between the results for Model 3 and Model 4 were observed. These differences might be caused by low numbers of observations in the given herds like in herd 1 and 6, where in the number of piglets born alive in parity 1 considerable differences in the estimates of the additive effects were observed. Another reason might be an unfavourable distribution of the *ESR* genotypes within the given herd. This was the case for example in herds 5 and 7 for the number of piglets born alive in parity 2 and subsequent parities; in these two herds the number of litters from *AA* sows was only about one third of the number of litters from *BB* sows.

DISCUSSION

In the literature, there are no studies on the effect of the *ESR* gene on lean meat percentage, but some

Table 6. Estimates of additive and dominance effects of the *ESR* locus for production traits for Models 1 and 3

Herd	Model 1	Model 3	
	Additive effect ± std. error	Additive effect ± std. error	Dominance effect ± std. error
Lean meat percentage (%)			
1	–0.06 ± 0.46	–0.03 ± 0.46	–0.23 ± 0.55
2	–0.12 ± 0.33	–0.19 ± 0.35	0.33 ± 0.41
3	0.13 ± 0.30	0.13 ± 0.30	–0.09 ± 0.41
4	–0.30 ± 0.40	–0.33 ± 0.42	0.11 ± 0.49
5	0.09 ± 0.34	0.10 ± 0.34	–0.07 ± 0.46
6	0.30 ± 0.51	0.26 ± 0.52	0.20 ± 0.68
7	–0.07 ± 0.34	–0.01 ± 0.35	0.30 ± 0.44
8	–0.05 ± 0.35	–0.03 ± 0.35	0.00 ± 0.46
9	0.04 ± 0.38	–0.01 ± 0.41	0.20 ± 0.53
10	0.03 ± 0.23	0.03 ± 0.23	0.05 ± 0.30
Average daily gain (g/day)			
1	0.06 ± 12.03	0.35 ± 12.07	–0.25 ± 14.97
2	9.89 ± 8.64	7.42 ± 8.91	12.23 ± 11.04
3	1.05 ± 7.50	1.24 ± 7.51	1.53 ± 11.22
4	6.25 ± 10.48	0.77 ± 11.00	18.58 ± 12.87
5	–1.38 ± 8.54	–1.66 ± 8.72	–1.58 ± 12.18
6	0.60 ± 13.29	4.92 ± 13.60	–24.41 ± 17.72
7	–11.53 ± 8.78	–12.92 ± 9.16	–7.89 ± 12.10
8	**20.27 ± 8.96**	**19.86 ± 9.03**	1.96 ± 12.21
9	–9.92 ± 10.00	–9.75 ± 10.65	–0.74 ± 14.08
10	6.76 ± 6.04	6.74 ± 6.04	–3.72 ± 7.98

[1]See Table 2 for the definition of Models 1 to 3

Bold letters: Effect differs significantly from zero on the level $P = 0.05$ at least

authors showed significant allele effects on backfat thickness which is a trait very closely related to lean meat percentage. Short *et al.* (1997) estimated a small, but significant negative additive effect of allele *B* on backfat thickness, which is in agreement with the tendency to better performance of allele *B* from phenotypic means across herds in the current study. On the other hand, Rothschild *et al.* (1995) and Leeds *et al.* (2002) showed allele *A* as allele of favour. As in the present paper no genetic effect of the *ESR* polymorphism on lean meat percentage was confirmed, the observed phenotypic differences might be caused by environmental effects.

In accordance with our results across herds, Rothschild *et al.* (1995), Short *et al.* (1997) and Leeds *et al.* (2002) reported no significant association between the *ESR* gene and average daily gain. In contrast to the current study all these authors considered average daily gain during the test period and not lifetime daily gain. Though there was no overall effect of the *ESR* gene, a significant additive effect in favour of allele *B* was confirmed by all animal models in one herd. It cannot be fully ruled out that this result could originate by random. On the other hand, average daily gain is a very rough description of the sophisticated growth process and does not say anything about the course of the growth curve. This is one reason that a certain gene can have an effect in a given concrete situation and be without effect in a situation that looks identical as a whole but differs in details which are not visible.

There is an opinion that the favourable allele for litter size may be the unfavourable allele for pre-weaning piglet growth (van Rens and van der Lende, 2002). Furthermore, the *ESR* polymorphism could influence the individual birth weight of piglets (van Rens and van der Lende, 2002; Leeds *et al.*, 2002). But both of these effects will occur at the beginning of the growth curve and can be compen-

Table 7. Estimates of additive and dominance effects of the *ESR* locus for litter size traits for Models 3 and 4

Herd	Model 3		Model 4	
	Additive effect ± std. error	Dominance effect ± std. error	Additive effect ± std. error	Dominance effect ± std. error
Number of piglets born alive in parity 1				
1	0.39 ± 0.31	–0.26 ± 0.46	–0.99 ± 0.58	–0.87 ± 0.63
2	–0.13 ± 0.27	0.21 ± 0.37	–0.17 ± 0.33	0.15 ± 0.41
3	0.28 ± 0.22	0.17 ± 0.35	0.16 ± 0.30	0.08 ± 0.43
4	–0.53 ± 0.34	0.15 ± 0.44	–0.49 ± 0.44	0.75 ± 0.54
5	–0.05 ± 0.25	–0.36 ± 0.42	0.10 ± 0.40	–0.18 ± 0.53
6	**–1.64 ± 0.40**	0.90 ± 0.57	–0.40 ± 0.80	1.18 ± 0.92
7	–0.43 ± 0.23	**–0.89 ± 0.40**	–0.31 ± 0.53	–1.24 ± 0.65
8	0.10 ± 0.27	–0.50 ± 0.42	–0.21 ± 0.39	–0.95 ± 0.55
9	–0.09 ± 0.33	–0.79 ± 0.46	0.13 ± 0.40	–0.68 ± 0.51
10	–0.33 ± 0.18	0.02 ± 0.26	**–0.55 ± 0.24**	–-0.11 ± 0.29
Number of piglets born alive in parity 2 and subsequent parities				
1	–0.06 ± 0.25	0.16 ± 0.36	–0.22 ± 0.40	0.17 ± 0.44
2	0.15 ± 0.31	–0.12 ± 0.41	0.05 ± 0.36	–0.09 ± 0.44
3	**0.54 ± 0.20**	–0.07 ± 0.32	0.43 ± 0.23	–0.41 ± 0.35
4	–0.28 ± 0.25	–0.08 ± 0.31	0.32 ± 0.29	–0.02 ± 0.32
5	–0.22 ± 0.24	0.44 ± 0.41	0.29 ± 0.45	0.47 ± 0.58
6	**–1.03 ± 0.27**	**0.89 ± 0.40**	**–1.15 ± 0.34**	**0.88 ± 0.44**
7	–0.20 ± 0.18	0.42 ± 0.31	**–1.36 ± 0.34**	–0.80 ± 0.42
8	–0.12 ± 0.26	**0.72 ± 0.33**	–0.12 ± 0.31	**0.92 ± 0.37**
9	**–0.50 ± 0.24**	–0.39 ± 0.33	–0.09 ± 0.27	–0.04 ± 0.35
10	0.11 ± 0.15	–0.28 ± 0.22	0.11 ± 0.18	–0.22 ± 0.24

[1] See Table 2 for the definition of Models 3 to 4

Bold letters: Effect differs significantly from zero on the level $P = 0.05$ at least

sated in a later phase resulting in no overall visual effect of the *ESR* gene.

Summarizing studies with Large White lines, it can be judged that the influence of the *ESR* gene on litter size differs between populations in size and also in allele of favour. Rothschild *et al.* (1995) and Short *at al.* (1997) detected a statistically significant favourable additive effect of the *B* allele on litter size ranging from 0.3 to 0.5 piglets born and piglets born alive. In other studies no clear association between the *ESR* polymorphism and litter size traits was shown. Additive effects in these studies were insignificant, but with a tendency to better (Rothschild *et al.*, 1996; Legault *et al.*, 1996) or worse (Southwood *et al.*, 1995) prolificacy of *BB* sows compared to *AA* sows. In a population of Czech Large White sows, a negative additive effect of the *B* allele on litter size traits across parities as well as in the first and in the second and subsequent parities was found (Goliášová and Wolf, 2004). For the number of piglets born alive across herds, the additive effect in the first parity was twice as high as the additive effect in the second and subsequent parities in this paper. When considering individual herds as done in the current investigation, the additive effect for the number of piglets born alive in parity 1 was statistically significant only in one herd, but for the number of piglets born alive in parity 2 and subsequent parities in two or three herds, depending on the model used. These outcomes could probably be influenced by the smaller number of observations in particular herds for litter size in parity 1 and/or by a different allele of favour in particular herds for litter size in parity 2 and subsequent parities.

For sows involved into the current study, no deviation from the Hardy-Weinberg equilibrium was detected, although the population was under selection for litter size. Generally in literature, differences between *ESR* allele frequencies are not significant within breeds but mainly between breeds. In accordance with our study, no deviation from the Hardy-Weinberg equilibrium was observed in INRA hyperprolific and control lines (Legault *et al.*, 1996). Furthermore, no significant influence of selection on the *ESR* genotype frequencies was found (Linville *et al.*, 2001). In contrast to Large White lines, allele *B* disappeared in Landrace based populations (Drogemuller *et al.*, 1999, 2001) or its frequency is very low (Noguera *et al.*, 2003).

No clear general conclusions can be drawn about the impact of the *ESR* gene on litter size traits. The *ESR* effect can differ in its magnitude and direction not only across but also within populations. It is not obvious whether the estimated allelic effects are directly caused by *ESR* polymorphism or by a major gene for litter size, which is in linkage with the *ESR* gene. So far it is not clear if *Pvu*II polymorphism is connected with a functional change of the gene expression as this mutation is located in an intron (Gibson *et al.*, 2002). Questionable is also the way the *ESR* could influence litter size. Probably, *ESR* polymorphism does not affect the number of *corpora lutea* nor hormone profiles (van Rens *et al.*, 2000; Gibson *et al.*, 2002), but acts on foetal survival through an effect on the placental size (van Rens, 2001).

It is expected that complex traits, such as litter size, are usually determined by a number of genes with various interactions of its products on various levels of biological organization. In our opinion it is very difficult to estimate effects of individual genes without knowing the genetic background. Therefore, in the given stage of knowledge, we cannot recommend the use of the *ESR* gene for selection for litter size in the Czech Large White population. It does not mean that the gene has no effect on litter size. The main problem is that the effective use of this effect (in case there is any) will demand more profound knowledge of the mechanism of gene action.

Ackonwledgements

Thanks are due to the Laboratory of Applied Genetics at the Mendel University of Agriculture and Forestry in Brno and the Association of Pig Breeders in the Czech Republic for making the data available.

REFERENCES

Bidanel J.P., Rothschild M.F. (2002): Current status of quantitative trait loci mapping in pigs. Pig News Info., *23*, N39–N54.

Drogemuller C., Hamann H., Thieven U., Krieter J., Distl O., Harlizius B. (1999): Influence of the genome region surrounding the estrogen receptor (*ESR*) gene on litter size in a German Landrace population. Arch. Tierz., *42*, 175–177.

Drögemüller C., Hamann H., Distl O. (2001): Candidate gene markers for litter size in different German pig lines. J. Anim. Sci., *79*, 2565–2570.

Gibson J.P., Jiang Z.H., Robinson J.A.B., Archibald A.L., Haley C.S. (2002): No detectable association of the *ESR* *Pvu*II mutation with sow productivity in a Meishan × Large White F_2 population. Anim. Genet., *33*, 448–450.

Goliášová E., Wolf J. (2004): Impact of the *ESR* gene on litter size and production traits in Czech Large White pigs. Anim. Genet., *35*, 293–297.

Groeneveld E., Kovac M., Wang T.L., Fernando R.L. (1992): Computing algorithms in a general purpose BLUP package for multivariate prediction and estimation. Arch. Tierz., *35*, 399–412.

Leeds T.D., Irvin K.M., Moeller S.J. (2002): The association between the estrogen receptor locus and growth, carcass and developmental traits in pigs. In: Proc. 7th World Cong. Genet. Appl. Livest. Prod. (CD-ROM), Montpellier, Communication 03–26.

Legault C., Gruand J., Lebost J., Garreau H., Ollivier L., Messer L.A., Rothschild M.F. (1996): Fréquence et effet sur la prolificité du gène *ESR* dans deux lignées Large White en France. Journées Rech. Porcine en France, *28*, 9–14.

Linville R.C., Pomp D., Johnson R.K., Rothschild M.F. (2001): Candidate gene analysis for loci affecting litter size and ovulation rate in swine. J. Anim. Sci., *79*, 60–67.

Noguera J.L., Varona L., Gómez-Raya L., Sánchez A., Babot D., Estany J., Messer L.A., Rothschild M., Pérez-Enciso M. (2003): Estrogen receptor polymorphism in Landrace pigs and its association with litter size performance. Livest. Prod. Sci., *82*, 53–59.

Peškovičová D., Wolf J., Groeneveld E., Wolfová M. (2002): Simultaneous estimation of the covariance structure of traits from field test, station test and litter recording in pigs. Livest. Prod. Sci., *77*, 155–166.

Rohrer G.A. (2004): An overview of genomics research and its impact on livestock reproduction. Reprod. Fert. Develop., *16*, 47–54.

Rothschild M.F., Vaske D.A., Tuggle C.K., Messer L.A., McLaren D.G., Short T.H., Eckardt G.R., Mileham A.J., Plastow G.S., Southwood O.I., van der Steen H.A.M. (1995): Estrogen receptor locus is a major gene for litter size in the pig. In: 46th Annual Meeting of the EAAP, Prague, paper G5.2.

Rothschild M., Jacobson C., Vaske D., Tuggle C., Wang L., Short T., Eckardt G., Sasaki S., Vincent A., McLaren D., Southwood O., van der Steen H., Mileham A., Plastow G. (1996): The estrogen receptor locus is associated with a major gene influencing litter size in pigs. Proc. Nat. Acad. Sci. USA, *93*, 201–205.

Short T.H., Rothschild M.F., Southwood O.I., McLaren D.G., de Vries A., van der Steen A., Eckardt G.R., Tuggle C.K., Helm J., Vaske D.A., Mileham A.J., Plastow G.S. (1997): Effect of the estrogen receptor locus in reproduction and production traits in four commercial pig lines. J. Anim. Sci., *75*, 3138–3142.

Southwood O.I., van der Steen H.A.M., Mileham A.J., Cuthbert-Heavens D. (1995): Evaluation of the estrogen receptor (*ESR*) gene in Meishan synthetic and Large White pigs. In: 46th Annual Meeting of the EAAP, Prague, paper G5.3.

Van der Lende T., Knol E.F., van Rens B.T.T.M. (2002): New developments in genetic selection for litter size and piglet survival. Thai J. Vet. Med., *32*, 33–46.

Van Rens B.T.T.M. (2001): Physiological aspects of two candidate genes for litter size in pigs. [Ph.D. Thesis.] University of Wageningen, 210 pp.

Van Rens B.T.T.M., van der Lende T. (2002): Piglet and placental traits at term in relation to the estrogen receptor genotype in gilts. Theriogenology, *57*, 1651–1667.

Van Rens B.T.T.M., Hazeleger W., van der Lende T. (2000): Periovulatory hormone profiles and components of litter size in gilts with different estrogen receptor (*ESR*) genotypes. Theriogenology, *53*, 1375–1387.

Welch B.L. (1947): The generalization of Student's problem when several different population variances are involved. Biometrika, *34*, 28–35.

Wolf J., Groeneveld E., Peškovičová D., Wolfová M., Jelínková V., Pražák Č., Fiedler J. (1999): Odhad plemenné hodnoty u prasat v České republice – aktuální stav a perspektivy dalšího vývoje. In: Wolf J., Groeneveld E., Peškovičová D. (eds.): Odhad plemenné hodnoty u prasat v Česku a na Slovensku – aktuální stav a perspektivy dalšího vývoje. Research Institute of Animal Production, Prague-Uhříněves. 15–39.

Received: 04–02–24
Accepted after corrections: 04–08–05

ABSTRAKT

Efekty genu *ESR* na velikost vrhu a produkční znaky u prasnic plemena bílé ušlechtilé v rámci stáda

Efekt *Pvu*II polymorfismu genu estrogenového receptoru (*ESR*) na velikost vrhu a produkční znaky byl vyhodnocován v deseti chovech plemene bílé ušlechtilé (882 plemenic, 2 455 vrhů) v České republice. K analýze vlastností průměrný denní přírůstek od narození, podíl libového masa, počet živě narozených selat na prvních vrzích a počet živě narozených selat na druhých a dalších vrzích byly využity čtyři čtyřznakové animal modely. Jednotlivé animal modely se lišily typem efektu stádo-rok-období (pevný nebo náhodný) a zahrnutím nebo nezahrnutím dominantního efektu. Efekty *ESR* byly hodnoceny jak v rámci jednotlivých chovů, tak i přes chovy. Při vyhodnocování přes chovy nebyly na genetické úrovni zjištěny průkazné efekty *ESR* na žádnou z hodnocených vlastností, efekty *ESR* v rámci chovů byly také převážně neprůkazné. Na prvních vrzích byla patrná tendence k vyšší užitkovosti spojené s alelou *A*. Nicméně při současné úrovni znalostí nelze využití *ESR* pro selekci na velikost vrhu jednoznačně doporučit, protože dosud existuje příliš mnoho otázek, které musí být před její aplikací zodpovězeny.

Klíčová slova: prase; reprodukce; velikost vrhu; kandidátní gen; ESR

Corresponding Author

Dr. Jochen Wolf, Výzkumný ústav živočišné výroby, P.O.Box 1, 10 401 Praha-Uhříněves, Česká republika
Tel. +420 267 009 573, fax +420 267 710 779, e-mail: wolf.jochen@vuzv.cz