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Evidence for a new major gene influencing meat quality in pigs

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Summary

The present investigation primarily deals with the inheritance of a pigmeat quality trait, the Napole technological yield (RTN), a measure of cooked weight to fresh weight. This trait as well as lean percentage at 100 kg liveweight and fattening length from 20 to 100 kg liveweight were recorded on 3459 offspring from 67 sires and 433 dams, and 3052 offspring from 64 sires and 405 dams in Penshire (P66) and Pen Ar Lan (P77) composite lines respectively. The hypothesis of a major 2-allele locus contributing to RTN was tested by use of a segregation analysis method. Highly significant likelihood ratios (mixed *vs.* polygenic transmission models) lead us to conclude that a major gene RN^- exerting an unfavourable effect on RTN is segregating in both lines. Maximum likelihood estimates of the parameters under the hypothesis of mixed (monogenic + polygenic) inheritance show that the difference between the means of the 2 homozygotes amounts to about 3 phenotypic standard deviations of the trait, whereas the complete dominance of RN^- cannot be rejected. The frequency of RN^- is about 0·6 in both lines. These results are discussed in connection with the previously reported 'Hampshire effect' on pigmeat quality, as the Hampshire breed is a common component of the foundation stock of the 2 composite lines under study.

1. Introduction

Over the past 25 years, a large number of research has been conducted on the genetic variability of pig meat quality. The usual criteria (colour, pH, water holding capacity) are submitted to a moderate additive genetic variation ($h^2 = 0{\cdot}10$–$0{\cdot}35$), and, in general, show no heterosis (Sellier, 1988), even if the 'Hampshire effect' on meat quality (Monin & Sellier, 1985) could be inherited as a dominant trait in Hampshire crosses (Sellier, 1987). On the other hand, it is well known that the occurrence of the Pale-Soft-Exudative (PSE) meat condition is linked to the genotype at the halothane sensitivity (*Hal*) locus (e.g. Webb *et al.* 1985). More recently, a study dealing with a new meat quality measurement, the so-called 'Napole' technological yield (RTN) (Naveau *et al.* 1985), suggests the segregation of a major gene other than the halothane sensitivity gene (Hal^n) acting on meat quality (Naveau, 1986). Indeed, a bimodal frequency distribution of the trait in both composite lines studied (P66 Penshire and P77 Pen Ar Lan), as well as a positive relationship between the sire family means and the within-sire family variances support the hypothesis of a major locus, with 2 alleles: a recessive normal allele 'rn^+' and a dominant allele 'RN^-' which lowers RTN.

This preliminary study was pursued in the present investigation on the basis of a larger amount of data, using a segregation analysis method (Elston & Stewart, 1971).

2. Material and methods

2.1. *Animals and traits*

Animals of our sample were born on the Pen Ar Lan farm at Maxent (Ille-et-Vilaine, France) and belong to 2 composite sire lines P66 (Penshire) and P77 (Pen Ar Lan) established by this breeding company. The Pen Ar Lan line, selected since 1973, was founded on with the three breeds Hampshire, Pietrain and Large White in equal proportions. The Penshire line, selected since 1977, was founded on with Hampshire (50%), Large White (15%) and Duroc (35%). In both lines,

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selection was based on the same performance-test index including days on test from 20 to 100 kg and predicted lean percentage at 100 kg liveweight. At the end of the test period, all males and females not kept for breeding were slaughtered in the same commercial abattoir. A sample of semi-membranosus (SM) muscle was then taken on the slaughterline and the 'Napole' technique was applied, as described by Naveau *et al.* (1985). The sample was kept in a plastic bag for 24 h in a refrigerator. It was then dressed with a knife and 1 cm^3 cubes were cut out. Fresh muscle weighing 100 g was put in a beaker with 20 g of pickle (136 g per litre nitrited salted water). The muscle cubes were totally immersed, the beaker was closed and kept at 4 °C for 24 h, then put on a circular holder and plunged in boiling water for 10 min. The homogeneity of cooking was obtained through the holder rotation. Finally, the beaker was opened and left on a draining rack for 2·5 h. The RTN is defined as the ratio of cooked weight to fresh weight.

The analysis deals with 3459 P66 animals (from 67 sires and 433 dams), and 3052 P77 animals (from 64 sires and 405 dams), born between July 1983 and March 1986. The sample is restricted to the sire families comprising at least 10 measured offspring. The analysed traits are:

Lean. Lean percentage at 100 kg liveweight, defined as:

$$\text{Lean} = 54{\cdot}42 + 0{\cdot}13W - 0{\cdot}94BT,$$

where BT is average backfat thickness (in mm), measured by ultrasonics on the live animals, and W is weight (in kg) on the day of ultrasonic recording (Naveau & Flého, 1980).

Days. Days on test from 20 to 100 kg, defined as:

$$\text{Days} = 80{\cdot}0975 - 1{\cdot}1464W + \text{A} + 2{\cdot}784\text{w} - 0{\cdot}0265\text{w}^2 - 1{\cdot}3746\text{a} + 0{\cdot}0032\text{a}^2$$

where A and W are age (in days) and liveweight (in kg) at the ultrasonic recording, a and w are age and liveweight at the beginning of test period (Naveau & Flého, 1980).

RTN. Napole technological yield (%).

pH_u. Ultimate pH of SM sample at 24 h after slaughter. This variable has been measured on a subsample of 1219 and 1252 animals for the P66 and P77 lines, respectively.

2.2. *Methods*

Data collected in each of the two lines were analysed separately using the same statistical methods.

2.2.1. *Experimental design*

With the mating plans used in the lines, the sows had 1–6 litters (Table 1) and the same sow was never mated twice to the same boar. Neglecting the maternal half-sib covariances (a sow mated to 2 boars was considered as 2 different dams), this family structure was modelled as a hierarchical plan: each sample was assumed to be a set of n sire families ($i = 1, \ldots, n$) with m_i mates for sire i ($j = 1, \ldots, m_i$) and l_{ij} measured offspring for dam ij ($k = 1, \ldots, l_{ij}$). Sires and dams were assumed to be unrelated.

Table 1. *Distribution of number of litters per dam*

No. of litters	No. of dams	
	P66	P77
1	189	165
2	123	125
3	67	62
4	39	32
5	11	17
6	4	4
Total	433	405

2.2.2. *Estimation of genetic parameters*

The following linear model was applied for each of the 4 traits:

$$Y_{abcd*ijk} = \mu + a_a + b_b + s_c + D^*_{abd} + U_i + V_{ij} + E_{abcd*ijk}, \quad (1)$$

where

μ is the mean of the dependent variable $Y_{abcd*ijk}$ (Lean, Days, RTN, pH_u),

a_a is the fixed effect of year of birth with 3 levels (July 83–June 84, July 84–June 85 and July 85–March 86),

b_b is the fixed effect of slaughter season. After a preliminary analysis, the 12 months have been grouped in 2 levels (June–October and November–May), the month effect within group being not significant,

s_c is the effect of sex (female and male),

D^*_{abd} is the random effect of within year-season slaughter date. This effect, known to influence pork quality traits (Charpentier *et al.* 1971), was taken into account only for RTN and pH_u,

U_i is the effect of the ith sire, a random variable distributed as a normal with mean 0 and variance σ^2_u,

V_{ij} is the effect of the jth dam mated to the ith sire, a random variable distributed as a normal with mean 0 and variance σ^2_v,

$E_{abcd*ijk}$ is the error, a random variable distributed as a normal with a mean 0 and variance σ^2_e.

The heritabilities h^2 were derived from the variance components according to:

$$\hat{h}^2 = \frac{4\hat{\sigma}^2_u}{\hat{\sigma}^2_u + \hat{\sigma}^2_v + \hat{\sigma}^2_e}.$$

As models of analysis are not the same for all variables, the phenotypic and genetic correlations

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between the 4 studied variables were estimated in two steps. First, data were adjusted for environmental effects using the following model:

$$Y_{abcd*e} = \mu + a_a + b_b + s_c + D^*_{abd} + E_{abcd*e}. \quad (2)$$

Then the adjusted values were analysed using the nested model:

$$E_{ijk} = U_i + V_{ij} + E'_{ijk}.$$

The phenotypic correlation between two traits was estimated by the correlation between adjusted values E_{ijk}, and the genetic correlation by the correlation between sire effects U_i.

2.2.3. *Segregation analysis of RTN*

The hypothesis of a major locus contributing to RTN, as postulated earlier by Naveau (1986), was tested using a segregation analysis method based on comparison of likelihoods under different transmission models (Elston & Stewart, 1971).

2.2.3.1. *Models*

The RTN values were assumed to follow one of the following two models

General hypothesis H_1 ('mixed transmission model for RTN'). In this model, a major locus effect is added to the 'classical' polygenic variation of RTN. In the case where 2 alleles RN^- and rn^+ are segregating, 3 genotypes can be encountered: RN^-RN^-, RN^-rn^+ and rn^+rn^+, coded 1, 2 and 3 respectively. Sires have the genotype s ($s = 1, 2, 3$) with a probability p_s, and dams have the genotype t ($t = 1, 2, 3$) with a probability p_t. The RTN value of offspring with the genotype r, Y^r_{abcijk}, follows the model:

$$Y^r_{abcijk} = \mu_r + a_a + b_b + s_c + U_i + V_{ij} + E_{abcijk},$$

where μ_r is the mean of offspring with genotype r, and a_a, b_b, s_c, U_i, V_{ij} and E_{abcijk} are defined as in (1) and assumed to be independent of r. This model depends on 12 parameters: $\mu_1, \mu_2, \mu_3, \sigma_e, \sigma_u, \sigma_v, p_1, p_2$ ($p_3 = 1 - p_1 - p_2$), a_1, a_2 ($a_3 = -a_1 - a_2$), b_1 ($b_2 = -b_1$) and s_1 ($s_2 = -s_1$).

Null hypothesis H_0 ('polygenic transmission model for RTN'). This subhypothesis of the general hypothesis is given by: $\mu_1 = \mu_2 = \mu_3 = \mu_0$. The Y_{abcijk} data then follow the model (1) depending on 8 parameters: μ_0, σ_e, σ_u, σ_v, a_1, a_2, b_1 and s_1.

2.2.3.2. *Notation*

Genotypes. S_i is the genotype of the ith sire, and s_i its realization, T_{ij} is the genotype of the jth mate of the sire i, and t_{ij} its realization, and R_{ijk} is the genotype of the kth progeny of the dam ij, and r_{ijk} its realization.

Probabilities. $P(R_{ijk} = r_{ijk} | s_i, t_{ij})$ is the probability of r_{ijk} given the genotypes s_i and t_{ij} of the sire i and the dam ij.

Densities. f is the distribution of the sire effect U_i

$$f(u_i) = \frac{1}{\sqrt{2\pi}\sigma_u} \exp\left(-\frac{1}{2}\frac{u_i^2}{\sigma_u^2}\right)$$

g is the distribution of the dam effect v_{ij}

$$g(v_{ij}) = \frac{1}{\sqrt{2\pi}\sigma_v} \exp\left(-\frac{1}{2}\frac{v_{ij}^2}{\sigma_v^2}\right)$$

h is the distribution of the dependent variable, given u_i and v_{ij}

$$h_{r_{ijk}}(y_{abcijk} | u_i, v_{ij}) = \frac{1}{\sqrt{2\pi}\sigma_e} \times \exp\left(-\frac{1}{2}\left(\frac{y_{abcijk} - \mu_{r_{ijk}} - u_i - v_{ij} - a_a - b_b - s_c}{\sigma_e}\right)^2\right).$$

2.2.3.3. *Formulation of the likelihood ratio*

The test statistic is the ratio of the sample maximum likelihoods with the $H_0(M_0)$ and $H_1(M_1)$ hypotheses. This ratio is

$$l = -2 \ln \frac{M_0}{M_1}.$$

With our notation, and considering the preceding hypotheses

$$M_1 = \prod_{i=1}^{n} \sum_{s_i=1}^{3} p_{s_i} \int_{u_i} f(u_i) \prod_{j=1}^{m_i} \sum_{t_{ij}=1}^{3} p_{t_{ij}} \int_{v_{ij}} g(v_{ij}) \prod_{k=1}^{l_{ij}} \sum_{r_{ijk}=1}^{3} P(R_{ijk} = r_{ijk} | s_i, t_{ij}) \times h_{r_{ijk}}(y_{abcijk} | u_i, v_{ij})$$

and

$$M_0 = \prod_{i=1}^{n} \int_{u_i} f(u_i) \prod_{j=1}^{m_i} \int_{v_{ij}} g(v_{ij}) \prod_{k=1}^{l_{ij}} h_0(y_{abcijk} | u_i, v_{ij}).$$

Asymptotically, the likelihood ratio l under H_0 is distributed according to a central χ^2 with $2d$ degrees of freedom, d being the number of parameters with fixed values under H_0 (Wolfe, 1971). Here, we have 4 degrees of freedom. Thus if l is larger than the threshold of a χ^2_4 distribution at the α level, we can reject H_0 and decide that a major gene is segregating, with a probability α of error.

2.2.3.4. *Analysis of adjusted data*

For numerical reasons, estimates of the slaughter date effects could not be obtained jointly with the other parameters. Two different approaches were used in order to evaluate this effect: the segregation analysis was first made on the 'raw' data as described just above (i.e. unadjusted for the effect of slaughter date) and then on the data previously adjusted using model (2). In the latter case, estimation of the effects a, b and s is not needed, and the number of parameters to be estimated is 4 and 8 under H_0 and H_1, respectively.

Moreover, in view of the results of the first analysis,

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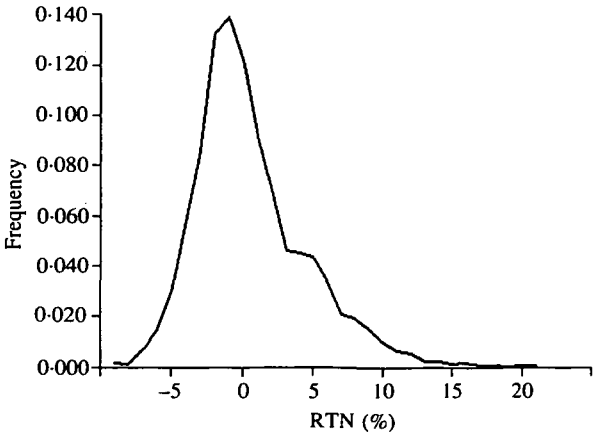

Fig. 1. Frequency distribution of RTN in the P66 line (adjusted data).

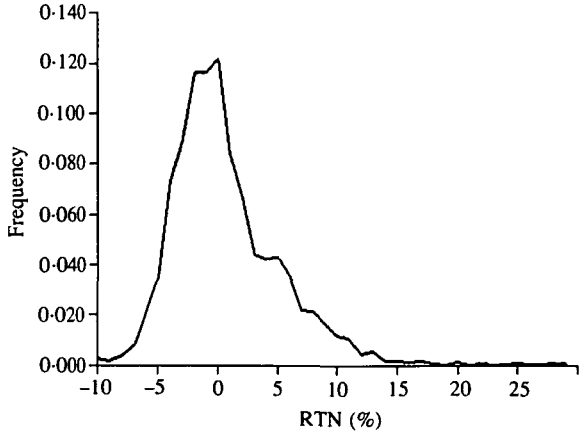

Fig. 2. Frequency distribution of RTN in the P77 line (adjusted data).

the mixed model assuming different means for the 3 genotypes was compared to a complete dominance model (H_2 hypothesis) where μ_1 and μ_2 are equal (7 parameters). Under this model, the 'Hardy–Weinberg frequencies hypothesis' (H_3 hypothesis), with $p_1 = q^2$ and $p_2 = 2q(1-q)$, q being the RN^- frequency, has also been tested (6 parameters).

2.2.3.5. *Transformation to remove skewness*

MacLean *et al.* (1975) and Demenais *et al.* (1986) showed that skewness may lead to the false inference of a major gene under the mixed model. On the other hand, skewness is expected when a major gene is segregating. In order to resolve between genuine skewness of the trait and segregation at a major locus, it has been suggested to transform the data using, for instance, the Box–Cox power transform $y = (c_1/c_2)[((x/c_1)+1)^{c_2}-1]$ where c_1 is a scale parameter and c_2 is a power parameter (MacLean *et al.* 1976).

As our distributions appeared to be strongly skewed (Fig. 1, 2) (the skewness coefficient g_1 being 0·876 and 1·059 for the adjusted data of P66 and P77 lines, respectively), we applied segregation analysis with a simultaneous Box–Cox transform on those adjusted data: two more parameters (c_1 and c_2) were estimated in this analysis, under both H_0 and H_1 hypotheses.

2.2.4. *Algorithms*

Estimation of genetic parameters was made using the procedures of linear model analyses of the SAS Library. The heritabilities were estimated by a MIVQUE0 method, and the phenotypic and genetic correlations by an Henderson I method. Calculations of the segregation analysis were made using the Gauss–Hermite quadrature (D01BAF) and optimization (E04JBF) subroutines of the NAG Fortran Library. A quasi Newton algorithm in which the derivatives were estimated by finite differences, was used for maximizing the likelihoods of the sample.

3. Results

3.1. *Estimation of genetic parameters*

Results of the analysis of variance for model (2) are given in Table 2. The P77 line, as compared to the P66 line, appears, on average, to be leaner (+2·0%), to show a faster growth (−1·2 days) and to give a slightly lower meat quality (−0·4% for RTN). Nevertheless, all the least-squares means for the 3 fixed effects point out the consistency of the results obtained separately for the two lines.

For growth and carcass performance, the gain in lean percentage during the 3-year period considered is 0·9% for P66 and 1.6% for P77, whereas there is no notable time trend in length of fattening. Animals slaughtered between June and October exhibit lower lean percentage and a longer fattening period as compared to those slaughtered between November and May. The sex effect is highly significant for these 2 traits, the females being fatter (−1·7 and −1·1% in lean content, for P66 and P77, respectively) and with a slower growth (+6·9 and +6·8 days on test).

For the two meat quality traits, RTN is continuously increasing during the 3-year period of study (+3·3 and +3·0%), whereas there is no clear trend in pH_u. Slaughtering between June and October induces a decrease in RTN (−1·1 and −1·1%) and pH_u (−0·04 and −0·17). Females show a higher RTN than males (+1·2 and +1·0%), but no sex effect is found in pH_u.

The analysis of variance using the model (1) shows that slaughter date has a real effect on RTN, explaining 8·9 and 10·1% of the total variance of the trait for the two lines P66 and P77, respectively. The effect of slaughter date on pH_u is still much larger, since 54·0% for P66 and 63·4% for P77 of the total variance are explained by this effect.

Estimates of genetic parameters are given in Table 3. As a general rule, estimates are not significantly different from one line to another, even if the heritabilities are always slightly higher for the P66 line. Genetic correlations, estimated with a low accuracy, are generally of low magnitude. Never-

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Table 2. *Results of the analysis of variance using model (2): least-squares means for the fixed effects*[a]

	Traits ...	Lean (%)		Days (d)		RTN (%)		pH_u	
	Line ...	P66	P77	P66	P77	P66	P77	P66	P77
Effect	Phenotypic S.D.	2·0	1·9	7·4	8·1	4·0	4·4	0·16	0·15
Year of birth	1983–84	55·4a	56·7a	99·7a	99·2a	88·1a	87·7a	5·64a	5·80a
	1984–85	55·3a	57·8b	100·5b	98·0b	89·9b	90·0b	6·03b	6·11b
	1985–86	56·3b	58·4c	99·7a	99·3a	91·5c	90·7c	5·62a	5·59c
Slaughtering season	June–October	55·5a	57·5a	101·0a	100·2a	89·3a	88·9a	5·74a	5·75a
	November–May	55·8b	57·7b	99·0b	97·4b	90·4b	90·0b	5·78a	5·92b
Sex	Female	54·8a	57·1a	103·5a	102·2a	90·4a	90·0a	5·76a	5·83a
	Male	56·5b	58·2b	96·5b	95·4b	89·2b	89·0b	5·76a	5·84a

[a] The standard errors of these estimates vary between 0·1 and 0·3 for Lean, Days and RTN, between 0·01 and 0·03 for pH_u. a, b, c: means with the same letter, for a given effect and a given line, are not significantly different at the 5% level.

Table 3. *Estimates* (±S.E.) *of heritabilities (on diagonal), phenotypic (above diagonal) and genetic (below diagonal) correlations*

	Lean		Days		RTN		pH_u	
	P66	P77	P66	P77	P66	P77	P66	P77
Lean	0·62 ±0·10	0·58 ±0·10	0·05 ±0·02	−0·05 ±0·02	−0·04 ±0·02	−0·11 ±0·02	−0·06 ±0·03	−0·06 ±0·03
Days	0·12 ±0·16	0·19 ±0·20	0·39 ±0·08	0·15 ±0·05	−0·03 ±0·02	−0·06 ±0·02	0·03 ±0·03	0·01 ±0·03
RTN	0·12 ±0·15	−0·41 ±0·16	0·33 ±0·15	−0·23 ±0·22	0·46 ±0·07	0·26 ±0·06	0·70 ±0·01	0·70 ±0·01
pH_u	−0·32 ±0·22	−0·13 ±0·30	−0·13 ±0·23	−0·14 ±0·48	0·81 ±0·09	0·73 ±0·17	0·24 ±0·08	0·09 ±0·08

theless, in the P77 line, lean percentage is negatively correlated with RTN. Phenotypic as well as genetic correlations between RTN and pH_u are in the range 0·70 to 0·80.

3.2. *Segregation analysis of RTN data*

In order to verify that no bias comes from our approximations on the family structure of the data sets a segregation analysis was first made using data collected on pigs from first-parity litters. The results being similar to those obtained on the whole sample, only the latter analysis will be given.

The likelihood ratios $-2\ln(M_0/M_1)$ given by the segregation analyses comparing mixed and polygenic models, widely exceed 13·28, the 1% threshold of the χ^2_4. It is concluded in each case that H_0 must be rejected, showing that a major gene acting on RTN was segregating.

The segregation analysis with Box–Cox transformation of the adjusted data leads to likelihood ratios of 113·4 and 115·8 for the P66 and P77 lines respectively. These figures are still highly significant even if they are much lower than the nontransformed ones.

For both lines, the estimated likelihood ratio $-2\ln(M_2/M_1)$ (0·6 for P66 line and 1·0 for P77 line) shows that a model of complete dominance fits the data adequately. However, the 'Hardy–Weinberg frequencies hypothesis' is not strictly valid when the 2 complete dominance mixed models with free or Hardy–Weinberg frequencies are compared, the likelihood ratio $-2\ln(M_3/M_2)$ being 7 and 13·6 (1 D.F.) for P66 and P77 lines respectively.

The maximum-likelihood estimates of the parameters for all these models are given in Table 4 for the 'raw' data, in Table 5 for the adjusted data and in Table 6 for the transformed data. In each case, the difference between the mean values of the 2 homozygotes is about 3 phenotypic standard deviations of the trait.

The frequency of the RN^- allele is about 0·6 in both lines. The estimated heritabilities are lowered from H_0 to H_1, for the 'raw' data from 0·57 to 0·17 (P66 line) and from 0·27 to 0·13 (P77 line), and for the adjusted data from 0·38 to 0·19 (0·15 for H_2) (P66 line) and from 0·20 to 0·17 (0·16 for H_2) (P77 line).

Under H_3 hypothesis, the additive genetic variance due to the segregation at the major locus ($RN^- rn^+$) is given by

$$\sigma^2 = 2q(1-q)^3(\mu_1-\mu_3)^2,$$

q being the RN^- frequency.

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Table 4. *Results of segregation analysis on 'raw' data: ML estimates of the parameters*[a]

Hypothesis ...	H_0 (polygenic model)		H_1 (mixed model)	
Line ...	P66	P77	P66	P77
μ_0	89·86	89·48	—	—
μ_1 (RN^-RN^-)	—	—	88·22	87·73
μ_2 (RN^-rn^+)	—	—	88·90	88·35
μ_3 (rn^+rn^+)	—	—	96·53	96·75
σ_e (residual)	3·44	3·77	2·50	2·54
σ_u (sire)	1·62	1·21	0·62	0·58
σ_v (dam)	1·96	2·38	1·49	1·87
p_1 (RN^-RN^-)	—	—	0·30	0·32
p_2 (RN^-rn^+)	—	—	0·57	0·62
a_1 (83–84)	−1·46	−1·06	−1·31	−1·15
a_2 (84–85)	−0·12	−0·28	−0·19	−0·17
b_1 (June–Oct)	−0·16	−0·39	−0·20	−0·37
s_1 (female)	0·65	0·49	0·53	0·46

[a] Likelihood ratio: l_{P66} = 313·0*** and l_{P77} = 355·1***.

Table 5. *Results of segregation analysis on adjusted data: ML estimates of the parameters*[a]

Hypothesis ...	H_0^*		H_1^{**}		H_2^{***}		H_3^{****}	
Line ...	P66	P77	P66	P77	P66	P77	P66	P77
μ_0	−0·10	0·00	—	—	—	—	—	—
μ_1 (RN^-RN^-)	—	—	−1·54	−1·68	−1·31	−1·41	−1·33	−1·48
μ_2 (RN^-rn^+)	—	—	−1·25	−1·27	−1·31	−1·41	−1·33	−1·48
μ_3 (rn^+rn^+)	—	—	6·24	6·91	6·21	6·87	6·22	6·84
σ_e (residual)	3·43	3·67	2·43	2·50	2·45	2·50	2·46	2·53
σ_u (sire)	1·18	0.95	0·59	0·61	0·52	0·59	0·54	0·59
σ_v (dam)	1·14	1·89	0·94	1·42	0·92	1·45	0·90	1·37
p_1 (RN^-RN^-)	—	—	0·31	0·33	0·30	0·33	—	—
p_2 (RN^-rn^+)	—	—	0·60	0·62	0·61	0·63	—	—
q (RN^-)	—	—	—	—	—	—	0·61	0·64

* H_0, polygenic model; ** H_1, mixed model; *** H_2, mixed model with complete dominance at the major locus; **** H_3, mixed model with complete dominance and Hardy-Weinberg frequencies at the major locus.
[a] Likelihood ratios: H_0 *vs.* H_1, l_{P66} = 390·0*** and l_{P77} = 401·8***. H_2 *vs.* H_1, l_{P66} = 0·6 and l_{P77} = 1·0. H_3 *vs.* H_2, l_{P66} = 7·0** and l_{P77} = 13·6***.

Table 6. *Results of segregation analysis on transformed data: ML estimates of the parameters*[a]

Hypothesis ...	H_0 (polygenic model)		H_1 (mixed model)	
Line ...	P66	P77	P66	P77
μ_0	−0·49	−0·73	—	—
μ_1 (RN^-RN^-)	—	—	−1·48	−1·67
μ_2 (RN^-rn^+)	—	—	−1·86	−2·27
μ_3 (rn^+rn^+)	—	—	4·53	4·35
σ_e (residual)	3·34	3·52	2·37	2·48
σ_u (sire)	1·09	1·01	0·54	0·67
σ_v (dam)	1·43	1·73	0·92	1·25
p_1 (RN^-RN^-)	—	—	0·23	0·25
p_2 (RN^-rn^+)	—	—	0·60	0·57
c_1 (scale)	11·7	23·3	1582·1	1586·4
c_2 (power)	−0·01	−0·85	−69·1	−80·7

[a] Likelihood ratio: l_{P66} = 113·4*** and l_{P77} = 115·8***.

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The major locus accounts for 79 and 75% of the total additive genetic variance ($\sigma^2+4\sigma_u^2$) for the P66 and P77 lines respectively.

The estimates of the parameters under H_1 hypothesis when data are submitted to a power transformation are similar to the estimates found on untransformed data, though the gene frequencies are closer to each other and the mean value of the heterozygote is below that of the 'unfavourable' homozygote (the inverse transformation gives for the 3 means μ_1, μ_2 and μ_3: $-1{\cdot}43$, $-1{\cdot}69$ and $5{\cdot}05$ in P66 line and $-1{\cdot}60$, $-2{\cdot}15$ and $4{\cdot}93$ in P77 line).

Estimates of fixed effects derived from the segregation analysis of the 'raw' data (under the mixed model) are similar to those given in Table 2: RTN significantly increases during the considered 3-year period ($+3{\cdot}0$% for P66 and $+2{\cdot}6$% for P77), RTN is lower in animals slaughtered from June to October ($-0{\cdot}4$ and $-0{\cdot}7$%) and females exhibit a higher RTN than males ($+1{\cdot}1$ and $+0{\cdot}9$%).

4. Discussion and conclusions

When averaged over the two lines, estimates of the genetic parameters are in general agreement with those reported by Naveau (1986) who analysed pooled data of the two lines. The observed line differences could be explained by a greater genetic variability of the P66 line which was created more recently. However, the heritability of ultimate pH of meat, found to be close to 0 by Naveau (1986), reaches here a more classical value (Sellier, 1988), the heritability of RTN being still higher in the P66 line.

On the other hand, the negative genetic correlation between RTN and Lean, previously reported by Naveau (1986), seems to be specific to the P77 line where the Hal^n gene is segregating. As proposed by Sellier (1988), the significant Hal^n allele frequency in this line (7·2% of the animals are halothane-positive) could explain this genetic antagonism by its favourable effect on lean percentage and its unfavourable effect on technological quality of meat.

Our segregation analyses confirm that a major gene acting on RTN is segregating in P66 and P77 lines and its contribution to RTN is quite similar in both lines. As outlined by Naveau (1986), the values of the parameters relative to this gene tend to prevent any confusion with the Hal^n gene. Indeed, the RTN unfavourable allele (RN^-) is completely dominant over the normal one (rn^+), a situation opposite to that of the 2 alleles Hal^n and Hal^N (e.g. Webb *et al.* 1985). Moreover, the estimate of the RN^- allele frequency is about 0·6 in both lines, while the P66 line is practically free from Hal^n (0·4% of halothane-positive animals).

The values of the likelihood ratios l_{P66} and l_{P77} were extremely high in the analyses. As shown by Le Roy & Elsen (1989), for a simpler but similar design, the conditions for using Wolfe's approximation (1971) are probably not fulfilled in our sample. Nevertheless, the results of this study show the good robustness of the segregation analysis method when the family size varies. Even if the high likelihood ratio obtained may be partly explained by the increase of the test statistic variance compared to a χ_4^2 variance, it cannot invalidate the conclusion drawn from these tests.

The means and frequencies of the 3 genotypes RN^-RN^-, RN^-rn^+ and rn^+rn^+ are close to those found by Naveau (1986) working on phenotype distribution. In view of the subtantial part of the total genetic variance that is due to this major locus, it would be very efficient to take into account this mixed inheritance for improving RTN.

Concerning the residual standard deviation σ_e, it should be pointed out that we tried to analyse a model where σ_e depended on the major locus genotype. Unfortunately, numerical difficulties occurred during the maximization process, and the optimal solution consisted of a classification of the population into 2 groups: one group with an intermediate mean and a variance equal to the population variance, and a second group with an extreme value and a null variance made of only one individual. This solution, a classical situation when estimating a mixture of distributions (MacLachlan & Basford, 1988), was obtained with different initial values.

As both lines contain Hampshire in their foundation stock, the relationship between the unfavourable effect of the RN^- allele on RTN and the 'Hampshire effect' on meat quality (Monin & Sellier, 1985) was suggested by Naveau (1986). The effect of the RN^- allele could be a large increase in muscle glycolytic potential (GP) inducing a great extent of post-mortem pH fall, a low ultimate pH of meat, and thus a lowered technological quality. Monin *et al.* (1987) recently found that the P66 line indeed exhibits high GP levels, as compared to the Large White, Pietrain and Belgian Landrace breeds, which are essentially similar in this respect (Sellier *et al.* 1988).

All these remarks should be considered with caution, and need to be confirmed by experiment.

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