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RESEARCH ARTICLE

REML estimates of genetic parameters of sexual dimorphism for wing and thorax length in *Drosophila melanogaster*

SANDRINE MIGNON-GRASTEAU[1*], JEAN DAVID[2], PATRICIA GIBERT[3], HÉLÈNE LEGOUT[2], GEORGES PÉTAVY[2], BRIGITTE MORETEAU[2] and CATHERINE BEAUMONT[1]

[1] *Station de Recherches Avicoles, Institut National de la Recherche Agronomique, 37380 Nouzilly, France*
[2] *CNRS, Laboratoire Populations, Génétique et Evolution, 91198 Gif-sur-Yvette Cedex, France*
[3] *CNRS, Laboratoire Biométrie, Biologie Evolutive, Université de Lyon I, 69622 Villeurbanne Cedex, France*

Abstract

Restricted maximum likelihood was used to estimate genetic parameters of male and female wing and thorax length in isofemale lines of *Drosophila melanogaster,* and results compared to estimates obtained earlier with the classical analysis of variance approach. As parents within an isofemale line were unknown, a total of 500 parental pedigrees were simulated and mean estimates computed. Full and half sibs were distinguished, in contrast to usual isofemale studies in which animals were all treated as half sibs and hence heritability was overestimated. Heritability was thus estimated at 0.33, 0.38, 0.30 and 0.33 for male and female wing length and male and female thorax length, respectively, whereas corresponding estimates obtained using analysis of variance were 0.46, 0.54, 0.35 and 0.38. Genetic correlations between male and female traits were 0.85 and 0.62 for wing and thorax length, respectively. Sexual dimorphism and the ratio of female to male traits were moderately heritable (0.30 and 0.23 for wing length, 0.38 and 0.23 for thorax length). Both were moderately and positively correlated with female traits, and weakly and negatively correlated with male traits. Such heritabilities confirmed that sexual dimorphism might be a fast evolving trait in *Drosophila*.

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Introduction

Sexual dimorphism, the difference between male and female traits, is present in most animal species. In *Drosophila melanogaster,* females have a larger body size and longer wings and thorax. Other differences, for example the pigmentation of the abdomen, make it possible to distinguish flies from the two sexes. This sexual dimorphism is believed to evolve under the pressure of natural and sexual selection, which implies that genes controlling body size differ at least partially between males and females (Cowley *et al.* 1986). However, there are very few estimates of the genetic parameters of sexual dimorphism for body size, even in this widely studied species (Bird and Schaffer 1972; Cowley *et al.* 1986; David *et al.* 2003). This is probably due to the fact that dimorphism cannot be scored on individuals.

Usually, analyses of variance are performed on mean fa milial performance and the individual variations are ignored (David *et al.* 1994). Moreover, male and female traits are treated as the same trait, thus assuming that the genetic correlation between male and female traits is equal to one, and that variances of both traits are equal. Finally, the parental structure is not taken into account in isofemale line studies, and this necessarily has an impact on estimates of genetic parameters. The REML (Restricted maximum likelihood) method developed by Patterson and Thompson (1971) is now widely used for variance component estimation, has been shown to be the best method to estimate genetic parameters (Gianola *et al*. 1986), especially when selection is occurring in the population (Gianola and Fernando 1986), and is now widely used in livestock selection programs (Colleau 1996). Using it could make it possible to treat male and female traits as distinct and to simultaneously estimate genetic parameters of traits in both sexes, the genetic correlations between male and female traits, and the genetic parameters of sexual dimorphism (Mignon-Grasteau *et al.* 1998). Moreover, REML allows us to take greater account of the genealogic information than usual in isofemale stud-

*For correspondence. E-mail: grasteau@tours.inra.fr.

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ies, as we can distinguish between full and half sibs, whereas all animals are treated as half sibs in classical isofemale studies. Thus, the aim of this study was to estimate genetic parameters of sexual dimorphism in isofemale lines using an REML approach and to compare the results with those obtained from analysis of variance.

Materials and methods

Animal material

Three samples of ten isofemale lines were studied. Female flies were collected in vineyards at Grande Ferrade near Bordeaux, France, at the end of the autumns of 1992, 1997 and 1999. Ten males and ten females were randomly sampled from each wild fly progeny to become parents of the next generation (figure 1). When adult, the 20 parents of the same line were placed in the same vial to produce the next generation. Progeny of the parental generation thus originated from full sib matings. A short egg-laying duration (4 h at 20-21°C) was used to limit larval density. Animals were reared at 25°C. Ten adult males and ten adult females per line were recorded for wing length (WL) from the thoracic articulation to the wing tip on the left side view and thorax length (TL) from the neck to the tip of the scutellum.

As measured animals within a line were reared together, the grandmother was known. However, although their parents were full sibs, the sire and dam of each animal remained unknown. We, therefore, simulated pedigree files by creating the missing parental generation in order to link measured animals to their grandmothers. As we did not have any prior information on the true structure of the families, an equal probability was given to each possible structure. This was achieved by independently sampling one of the ten possible sires and one of the ten possible dams for each individual in two independent uniform distributions, i.e. giving an equal probability to each possible sire and dam. However, no constraint was put on the number of offspring per sire or dam in order to allow unbalanced pedigrees with unequal contributions of parents. Finally, to alleviate bias due to simulation, 500 pedigrees were simulated and analysed. The distribution of the mean number of offspring per parent in the 500 repetitions is depicted in figure 2. The mode was two offspring per parent, as expected, as the population consisted of 600 recorded animals and 600 parents (i.e., 300 sires and 300 dams). Elementary statistics on recorded traits can be found in table 1.

Model of analysis

In order to estimate the genetic parameters of sexual dimorphism, male and female traits were treated as different, thus leading to a four-trait analysis including male wing length (WL_m), female wing length (WL_f), male thorax length (TL_m) and female thorax length (TL_f). Male traits were missing for females and female traits were missing for males. The model of analysis was thus:

$$\mathbf{y} = \mathbf{x\beta} + \mathbf{Zu} + \mathbf{e} \qquad (1)$$

with $\mathbf{E}\begin{bmatrix}\mathbf{u}\\ \mathbf{e}\end{bmatrix} = \mathbf{0}$ and $\mathbf{V}\begin{bmatrix}\mathbf{u}\\ \mathbf{e}\end{bmatrix} = \begin{bmatrix}\mathbf{A}\otimes\mathbf{G} & \mathbf{0}\\ \mathbf{0} & \mathbf{R}\end{bmatrix}$

where $\mathbf{y}$ was the vector of performances (i.e. WL_m, WL_f, TL_m, TL_f), $\boldsymbol{\beta}$ the vector of fixed effect for the year of capture of

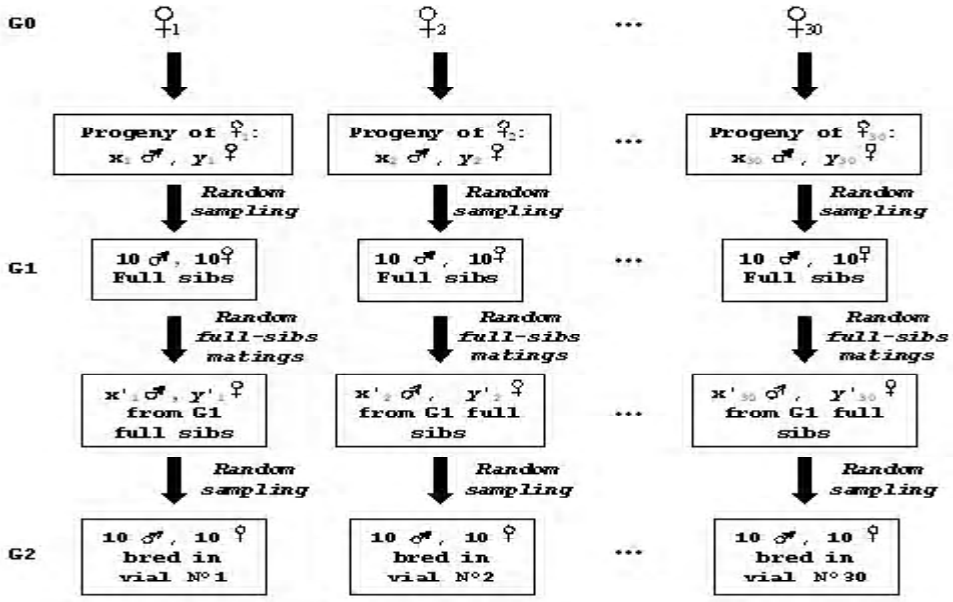

Figure 1. Structure of population in isofemale studies.

Table 1. Elementary statistics on the data file.

Trait*	*N*	Mean ± SD	Skewness	Kurtosis
WL_m (mm×10^2)	300	233.37±6.46	-0.20	-0.31
WL_f (mm×10^2)	300	270.83±7.23	-0.11	-0.40
TL_m (mm×10^2)	300	94.89±2.43	-0.08	0.28
TL_f (mm×10^2)	300	109.03±2.61	-0.04	-0.23
WL_f-WL_m (mm×10^2)**	30	37.45±3.07	0.03	-0.86
TL_f-TL_m (mm×10^2)**	30	14.13±1.36	0.69	0.71
WL_f/WL_m**	30	1.161±0.014	0.30	-0.21
TL_f/TL_m**	30	1.149±0.015	0.82	0.81

$WL_{m(f)}$: wing length of males (females); $TL_{m(f)}$: thorax length of males (females).
The difference between female and male traits and ratio of female to male traits were calculated using family means.

the grandmother (3 levels), **u** the vector of direct genetic effects of each animal, including parents and grandparents (1230 levels), and **e** the vector of residuals. **X** and **Z** were the incidences matrices corresponding to **β** and **u**, respectively. **G** was the matrix of the genetic (co)variances between traits, **A** the numerator relationship matrix calculated with classical Henderson's rules (1976), **V** the matrix of (co)variances, **R** the matrix of residual (co)variances between traits, and ⊗ the symbol for direct matrix product.

Estimation of genetic parameters

Due to the structure of our data file, which did not allow us to distinguish between vial and genetic effects, we had to estimate isofemale heritabilities (h^2_i) instead of classical heritabilities. The VCE.4 software (Neumaier and Groeneveld 1997) provided residual and additive (co)variances of male and female traits. These estimates were used to calculate isofemale heritabilities as in Hoffmann and Parsons (1988), e.g. for wing length in males:

$$h_i^2(WL_m) = \frac{\sigma_{ia}^2(WL_m)}{\sigma_{ip}^2(WL_m)} \qquad (2)$$

with $\sigma_{ia}^2(WL_m) = 2F\sigma_a^2(WL_m)$

and $\sigma_{ip}^2(WL_m) = \sigma_p^2(WL_m) + F\sigma_a^2(WL_m)$

where $\sigma^2_a(WL_m)$ and $\sigma^2_p(WL_m)$ were respectively the additive and phenotypic variances of WL_m, $\sigma^2_{ia}(WL_m)$ and $\sigma^2_{ip}(WL_m)$ the "isofemale" additive and "isofemale" phenotypic variances of WL_m, and F the inbreeding coefficient among recorded offspring (i.e. 0.25 as they originated from full sibs matings).

Additive and phenotypic variances of the difference between female and male traits were computed using classical formulae of the variance of the difference between two traits, e.g. for wing length:

$$\sigma_a^2(WL_f - WL_m) = \sigma_a^2(WL_m) + \sigma_a^2(WL_f) - 2\sigma_a^2(WL_m, WL_f) \qquad (3)$$

$$\sigma_p^2(WL_f - WL_m) = \sigma_p^2(WL_m) + \sigma_p^2(WL_f) - 2\sigma_p^2(WL_m, WL_f) \qquad (4)$$

where $\sigma^2_a(WL_m, WL_f)$ and $\sigma^2_p(WL_m, WL_f)$ were the additive and phenotypic covariances between WL_m and WL_f. Isofemale heritability of the difference was then calculated using equation 2. If this heritability is different from zero, selection on sexual dimorphism should be possible.

Isofemale heritability of the ratio of female to male traits were computed as in Pearson (1897) and Sutherland (1965), e.g. for wing length:

$$h_i^2\left({}^{WL_f}\!/_{WL_m}\right) = \frac{C_{ig}^2(WL_m) + C_{ig}^2(WL_f) - 2r_g(WL_m, WL_f)C_{ig}(WL_m)C_{ig}(WL_f)}{C_i^2(WL_m) + C_i^2(WL_f) - 2r_{ip}(WL_m, WL_f)C_i(WL_m)C_i(WL_f)} \qquad (5)$$

with $C_i(WL_m) = \frac{\sigma_{ip}(WL_m)}{\mu(WL_m)} = \frac{\sigma_p(WL_m) + F\sigma_a^2(WL_m)}{\mu(WL_m)}$ (6)

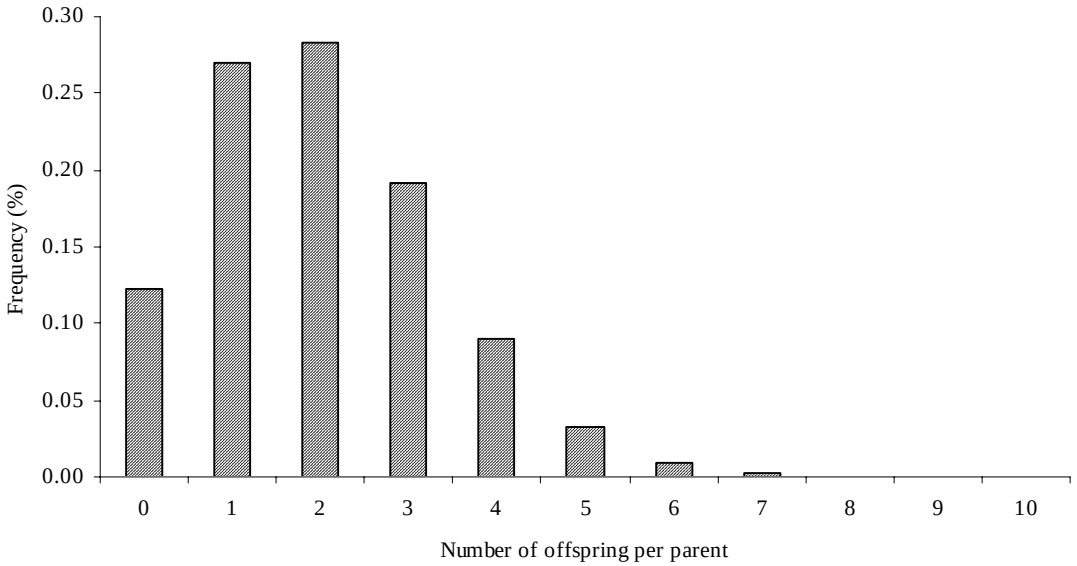

Figure 2. Distribution of the mean number of offspring per parent across the 500 simulated pedigrees..

and $$C_{ig}(WL_m) = C_i(WL_m)h_i(WL_m) \quad (7)$$

and $$r_{ip}(WL_m, WL_f) = \frac{(1+F)\sigma_a(WL_m, WL_f)}{\sigma_{ip}(WL_m)\sigma_{ip}(WL_f)} \quad (8)$$

where $C_i(WL_{m(f)})$ was the phenotypic coefficient of variation of male (female) wing length, $C_{ig}(WL_{m(f)})$ the genetic coefficient of variation of male (female) wing length, $r_{ig(p)}(WL_m, WL_f)$ the genetic (phenotypic) correlation between male and female wing length and $\mu(WL_{m(f)})$ the mean of wing length in males (females). If heritability of the ratio is not equal to zero, then sexual dimorphism cannot be reduced only to a scale effect. Finally, genetic correlations between the difference or the ratio and male or female traits were computed as (e.g., for WL_m)*:*

$$r_g(WL_m, WL_f - WL_m) = \frac{\sigma_a(WL_m, WL_f) - \sigma_a^2(WL_f)}{\sigma_a(WL_f - WL_m) - \sigma_a(WL_f)} \quad (9)$$

$$r_g\left(WL_m, {}^{WL_f}\!/_{WL_m}\right) =$$

$$\frac{r_g(WL_m, WL_f)C_{ig}(WL_f) - C_{ig}(WL_m)}{\sqrt{C_{ig}^2(WL_m) + C_{ig}^2(WL_f) - 2r_g(WL_m, WL_f)C_{ig}(WL_m)C_{ig}(WL_f)}} \quad (10)$$

Means and standard deviations of estimates of genetic pa-

Table 2. Estimates of genetic parameters of wing and thorax length in males and females, of difference between female and male traits, and of the ratio of female to male traits for wing and thorax length. Isofemale heritabilities are on the diagonal (bold type), genetic correlations above, phenotypic correlations below. Estimates are presented with their standard errors.

	WL_m	WL_f	TL_m	TL_f	WL_f-WL_m	TL_f-TL_m	WL_f/WL_m	TL_f/TL_m
WL_m	**.33±.02**	.85±.05	.74±.03	.54±.06	.03±.14	-.14±.08	-.05±.13	-.18±.08
WL_f	.75±.05	**.38±.01**	.64±.06	.75±.02	.55±.06	.23±.07	.48±.07	.18±.07
TL_m	.58±.06	.53±.05	**.30±.03**	.62±.07	.03±.14	-.31±.09	-.03±.14	-.37±.09
TL_f	.44±.05	.66±.05	.48±.06	**.33±.03**	.56±.09	.55±.06	.52±.10	.49±.07
WL_f-WL_m					**.30±.06**	.65±.10	.99±.01	.63±.10
TL_f-TL_m						**.38±.06**	.66±.10	.99±.01
WL_f/WL_m							**.23±.06**	.64±.10
TL_f/TL_m								**.23±.01**

rameters across the 500 simulations were obtained using the procedure MEANS of SAS (1989). Distributions of isofemale heritabilities of wing and thorax length, of sexual dimorphism, of the ratio and of the genetic correlation between male and female traits were also plotted.

Results and discussion

Genetic parameters of wing and thorax length

Using REML allowed us to take into account information from the pedigree, in contrast to analysis of variance. We thus estimated heritability (table 2) and not the intra-class correlation *t* as in David *et al.* (2003). Indeed, the latter overestimates heritability as it includes non-additive variance as dominance and epistasis in addition to additive variance. It is logical as no difference is made between full and half sibs, whereas it is possible with a REML. Not surprisingly, our heritability estimates were slightly lower than intra-class correlations obtained by David *et al.* (2003) on the same data set. For example, our heritabilities were moderate for wing and thorax length (0.33-0.38 and 0.30-0.33, respectively; table 2, figure 3) whereas estimates of intra-class correlation by David *et al.* (2003) were higher (0.46-0.54 for wing length and 0.35-0.38 for thorax length). However, our estimates were in agreement with the literature for wing length (0.32 in the review of Roff and Mousseau 1987) and for thorax length (ranging from 0.12 to 0.66 in Prout and Barker 1989; Capy *et al.* 1994; Reeve and Fairbairn 1996; Norry *et al.* 1997). Genetic correlations between wing and thorax length within a sex were also slightly higher in our study (figure 4) than in David *et al.* (2003).

While estimated heritabilities slightly differed between this study using REML and the study of David *et al.* (2003) using analysis of variance, the difference between male and female heritabilities were comparable in both studies (14.1% for WL and 9.6% for TL in our study, and 16.0% for WL and 8.2% for TL in David *et al.* 2003). This difference between males and females, which had also been noted earlier for wing and thorax length in *Drosophila* (Cowley *et al.* 1986, Cowley and Atchley 1988; Ruiz *et al.* 1991; David *et al.* 1994; Reeve and Fairbairn 1996) is not likely to be due to the method of analysis. It could be explained by the importance of sex-linked effects in *Drosophila* as this species has a low number of chromosomes and the X chromosome represents 20% of the genetic material (Bird and Schaffer 1972). Nevertheless, even with REML and a better treatment of available information, genetic and environmental contributions to variance between vials are still confounded, as all animals of the same family are reared in the same vial. However, Gibert *et al.* (1998) estimated in a similar population that the environmental effect due to vial was low, hence the error on genetic parameters may be expected to be small.

Sexual dimorphism

As the REML made it possible to deal with missing data, we were able to distinguish between male and female traits. Thus, sexual dimorphism was here studied on 600 individual performances, as contrasted with the 30 mean familial performances in the analysis of variance of David *et al.* (2003). However, the genetic correlations between male and female traits (figure 4) were very close to those estimated by David *et al.* (2003). Our expectations were equal to 0.84 and 0.62 for WL and TL, respectively, as compared to 0.83 and 0.68 in David *et al.* (2003). Both sets of genetic correlations were

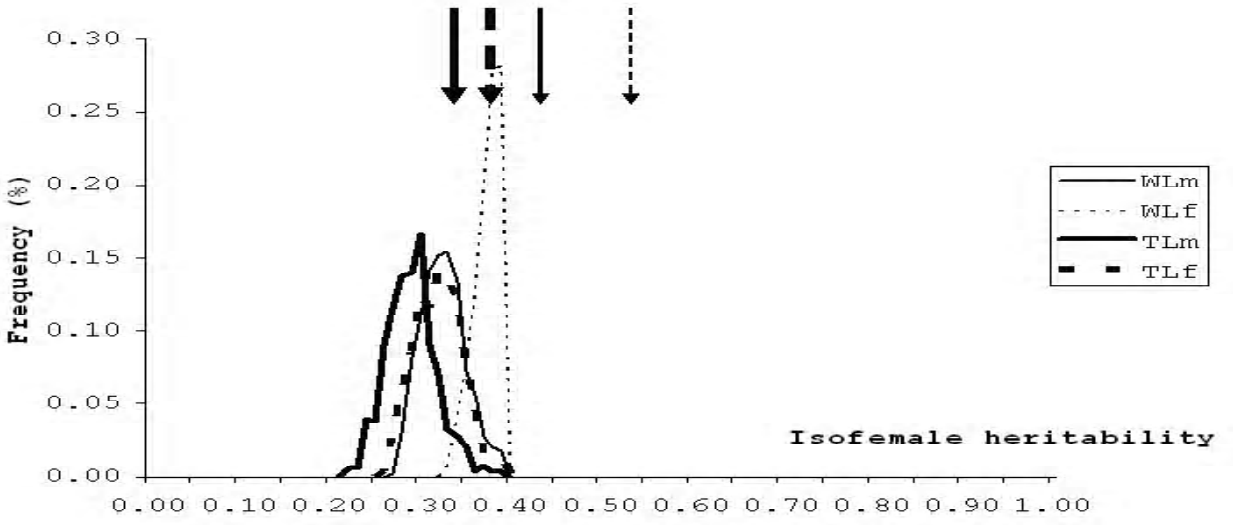

Figure 3. Distribution of isofemale heritability estimates for male and female traits over the 500 simulations. Arrows indicate estimates obtained on the same population with an analysis of variance by David *et al.* (2003).

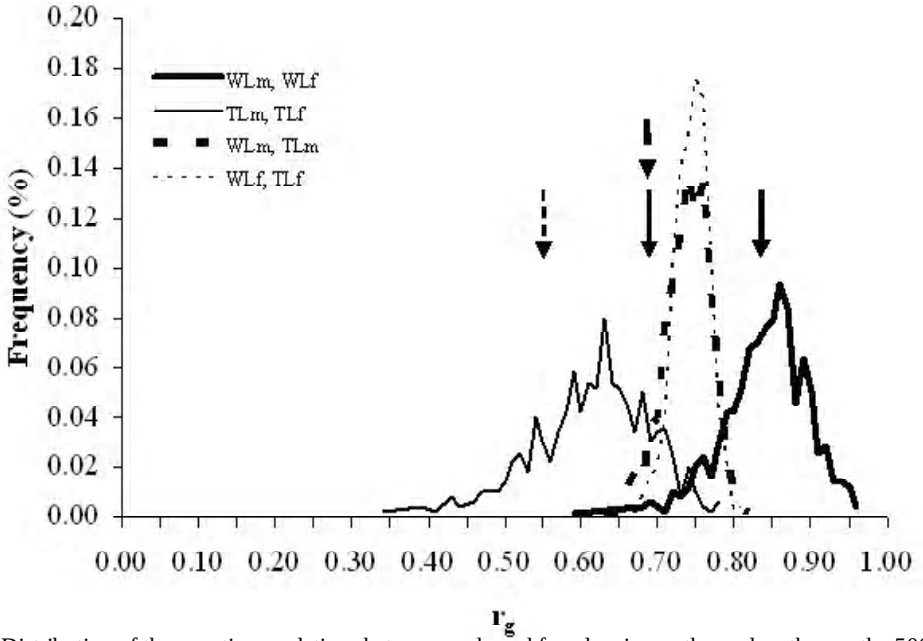

Figure 4. Distribution of the genetic correlations between male and female wing or thorax length over the 500 simulations. Arrows indicate estimates obtained on the same population with an analysis of variance by David *et al.* (2003).

significantly lower than one ($p < 0.01$), thus indicating that genes controlling both traits partially differ between sexes. On the contrary, heritability estimates of sexual dimorphism of both traits were much higher in our study (0.30 for WL, 0.38 for TL) than in David *et al.* (2003) (0.18 for WL and 0.23 for TL), mostly as a result of differences in heritability estimates for male and female traits. On the other hand, heritabilities of the ratio of male and female trait values were comparable with both methods, which is to relate to the stability of the difference between male and female heritabilities over both methods. These estimates of heritability of sexual dimorphism indicate that sexual dimorphism is alterable by artificial or natural selection. As both the ratio and the difference were positively correlated with female traits, while correlations with male traits were zero for WL to negative for TL, evolution of sexual dimorphism would likely affect female traits more than male traits.

Distribution of genetic parameters

As the exact genealogy was unknown, we simulated 500 different possible pedigrees, in which full sibs could be distinguished from half sibs, whereas in the study of David *et al.* (2003) all animals were treated as half sibs. We were thus able to draw the distributions of the estimated genetic parameters (figures 3, 4, 5). For example, depending on the pedigree structure considered, estimated heritability of TL in males varied from 0.22 to 0.40. The distribution of estimated heritabilities were to some extent not normal, especially when the classical heritability ($\sigma^2_a/\sigma^2_a+\sigma^2_e$) was close to one, as it is the case for WL_f, as REML estimates lay within the parameter space.

Finally, we can also observe the importance of the amount of information taken into account by observing distributions of estimates. It is to note that all individual information has been used with the REML, whereas two separate data sets are analysed for males and females with analysis of the variance, thus ignoring half of the available information.

For example, the figure 4 showed that estimations were more precise for genetic correlations between traits measured in the same sex than for traits measured in opposite sexes. For the latter, information used to estimate the correlation came only from related animals, as no animal was measured for both traits. The importance of direct versus indirect information could also be deduced by comparing distributions of heritabilities of WL or TL and those of sexual dimorphism for the same traits, the latter being much flatter than the former.

Conclusion

Our REML study globally confirmed results obtained earlier with analysis of variance. However, heritabilities of WL and TL were lower as distinguishing between full and half sibs allowed to better disentangle between additive genetic effects and other effects. Moreover, when considering sexual dimorphism, this technique gave the possibility of dealing with individual performances instead of familial means. This led to consistently higher heritabilities of the difference between males and females traits in REML than in analysis of variance. As 500 different parental structures were investigated, this study also showed how estimated genetic parameters were dependent on the pedigree.

However, the possibility of confusion between genetics and environment still remains due to the experimental de-

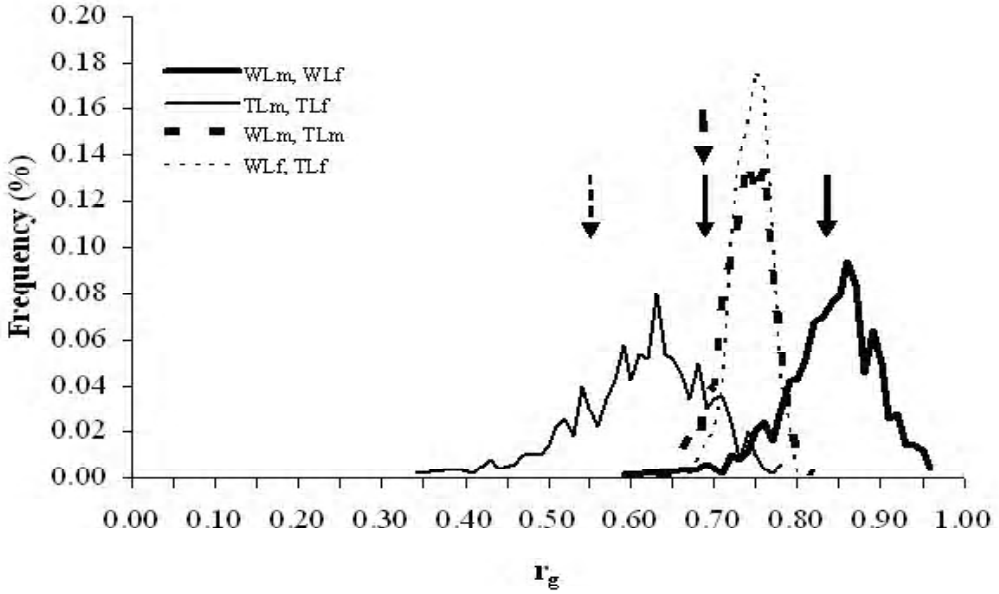

Figure 5. Distribution of the heritability estimates for the differences between female and male traits and for the ratios of female to male traits over the 500 simulations. Arrows indicate estimates obtained on the same population with an analysis of variance by David *et al.* (2003).

vice specific to isofemale studies. Further analyses are now needed to define to what extent these traits are submitted to genotype environment interactions, as suggested by David *et al.* (1994).

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