TWELVE REMARKS ON CANALISATION IN LIVESTOCK PRODUCTION

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INTRODUCTION

Several works published in recent years concerned the possible genetic component of phenotypic variability in livestock. Although all of them dealt with the genetic of variability, some were connected with plasticity, which is the ability to modulate the mean level of performance according to the environment. Other studies dealt with performance stability over different environments or with canalising selection (canalisation) which strives to obtain an homogenous population around an optimal average. These studies assumed different genetic hypotheses to explain the genetic control of variability and particularly the natural stability : a) Heterozygous individuals are best suited to fit variation of environments (Gillepsie and Turelli, 1989) ; b) Some genes which control the mean of a trait can also act on fitness, with overdominance effect (Robertson 1956) ; c) Due to pleiotropic effects, alleles of some genes controlling the mean can lead to different phenotypic expressions in different environments (Via and Lande, 1985) ; d) Mean and variability of a trait are under the control of different genes (Scheiner and Lyman, 1991). Experimental results confirm the existence of plasticity genes (Gibson and Hogness, 1986 ; Reilly *et al*., 1991 ; Lukens and Doebley, 1999).

Based on the last hypothesis, a statistical model has been proposed for populations under selection (SanCristobal-Gaudy *et al*. 1998) to allow the statistical treatment of canalising selection [1]. This model assumes that the mean production level depends on classical fixed and random effects and that the residual variance can be decomposed into a part under genetic control and the usual unexplained variability. Let Y_{ij} be the vector of n_i performance of an individual i. $Y_{ij} \mid u,v \sim N(x_i \beta + z_i u , \exp(p_i \delta + q_i v))$ $j=1,...n_i$ and $i=1,...I$

where n_i is the number of observations for an individual i, x_i, z_i, p_i, q_i are incidence vectors; β and δ are fixed effects; u and v are random genetic effects.

$$(\mathbf{u},\mathbf{v}) \mid \sigma_u^2, \sigma_v^2, r \sim N\left(\mathbf{0}, \begin{pmatrix} \sigma_u^2 & r\sigma_u\sigma_v \\ r\sigma_u\sigma_v & \sigma_v^2 \end{pmatrix} \otimes \mathbf{A}\right) \qquad [1]$$

where σ_u^2 and σ_v^2 are additive genetic variances, r is the correlation between u and v, **A** represents the relationship matrix and $\otimes$ denotes the Kronecker product. Implementation of models of genetic effect on environmental variability in livestock selection schemes is relatively recent and raises problems. Some of them are discussed below.

1. Is it possible to increase the phenotypic stability by way of selection ?
Several canalising or stabilising selection experiments have been undertaken and are reported in literature. They clearly show that this type of selection does work, *i.e.* performance can be totally stable across environments after some generations of selection (SanCristobal-Gaudy *et al.* 1998, for a review). This clearly demonstrates the existence of some genetic mechanisms controlling phenotypic variability. Although these experiments concern mainly laboratory animals, we can assume that canalising selection would also work in livestock. Experiments are being carried out in different species for further evidence.

2. How to deal with variance heterogeneity due to the presence of a major gene ?
If we consider a sire design where performance is measured on the progeny, the difference in within-sire variability can be due to a genetic effect on variance. It also may reflect the segregation in some families of a major gene which acts on the mean level of performance. In contrast, the difference of variability observed between individuals measured repeatedly in a large variety of environments can only reveal the existence of genes that neutralize environmental effects. The statistical confounding described above is higher when there are fewer repetitions of the within individual measurements and can be total for traits which cannot be measured several times on the same individual (e.g. birth weight). Nevertheless the within-sire variance may be used when the within-individual variability cannot be measured, only after checking the absence of mixed inheritance (polygenic and a major gene).

3. Why not simply select on the phenotypic variance or coefficient of variation ?
Considering unadjusted phenotypic standard deviation (Damgaard *et al*., 2001) or coefficient of variation (CV) denies the existence of fixed factors and genetic components explaining the classical heterogeneity of the mean production level. This may lead to miss-interpret this variability due to a mixture of several distributions (represented by different levels of a significant factor affecting the trait) and to assign it to genetic effects on the variance. Thus when environmental factors modify the performance, a joint modelling of mean and residual variance is essential although more complex. However, the CV approach can be useful and efficient when a large number of balanced observations per individual neutralize the environmental effect on the mean (diameter of 1000 fibres within a wool sample Allain *et al*., 1998). It is possible to firstly adjust the data for the different significant effects and in a second step analyse the residual variance. This approach can lead to biased estimates when the sample size is small (it was the case with the Henderson II method before introduction of BLUP).

4. Do we want plasticity or stability ?
Some authors are interested in plasticity or adaptation of production level according to resource availability. They consider large variation of clearly identified environmental factors and look for animals able to fit their production to these variations (Kolmodin *et al*., 2001). Other authors are interested in stability or canalisation of production traits obtained in relatively stable environments. Canalisation is then expected to reduce phenotypic variability around a mean level of production already observed in the concerned population. In this context, the lack of plasticity is not damaging since large environment variations are not expected.

5. Why not using existing models of GxE interactions ?
Such models assume that the genes controlling the mean production level interact with the environment (Strandberg *et al*., 2000). In this approach, environments are clearly defined and

identified. In livestock production, very few factors are known to interact with the genotype (Robert *et al*., 1995ab) and most of the small genotype-environment interactions have unknown and/or uncontrollable causes. The model presented in [1] takes into account these uncontrolled GxE interactions: it includes a genetic control of the variability by modelling the residual variance.

6. Do we have to select in one environment or several environments ?

It is different to select animals less sensitive to variations of a known factor (flock, season, age…) and to select for reducing the environmental variance (canalisation). In the first case, it is necessary to observe the expression of genotypes in each level of this factor. That imposes to measure all animals in all environments and not to consider, in the mean and in the variance models, the incidence of this particular factor as a fixed effect. When such a design is not feasible, confounding may arise between effects of genes which control variability and incidence of this particular factor on the mean. In contrast, canalising selection merely strives to reduce the environmental variance beyond the effects of identified factors. It is then necessary to adjust observations for the significant fixed effects, to reduce their influence or even better to select in only one environment.

7. What is the optimal selection strategy ?

The objective of canalising selection is twofold : to get performance close to the optimum and to reduce variability around it. To reach this objective at a given time horizon, different strategies can be envisioned. For instance, decrease the environmental variance and then reach the optimal mean or vice versa. Dynamic selection must be considered to optimise cumulative profit according to the number of generations, the number of measurements and animals involved, and all economic factors. Note that reducing first the environmental variance does not impair necessarily later genetic progress on the mean.

8. Does canalisation reduce genetic diversity ?

If genetic control of performance homogeneity is due to the homozygosity level, then selection for homogeneisation will unfortunately reduce genetic diversity in a drastic way. If a specific core of gene exists which controls the sensitivity to the environmental variability, as usual, canalising selection will reduce its diversity. It should not affect the diversity of genes controlling the mean. Literature shows a decrease or maintenance of the genetic variance (Holland *et al*., 2000). Heritability estimations during and at the end of several canalising selection experiments often showed that the selected trait genetic variance decreased, this statement not being general.

9. How to propose experimental canalising selection designs ?

The implementation of experimental protocols or selection programs for canalisation requires large number of measurements on each animal as well as large number of animals because variances rather than means have to be estimated. These numbers are even larger for designs aimed at estimating genetic parameters of canalisation ability (variance of variance estimations). Accuracy of these various estimations is currently studied by Ros *et al*. (2002).

10. What about the mean-variance relationship ?

For some traits of interest, mean and variances are mathematically linked (for example frequencies). In this situation, it seems difficult to decrease the variability without decreasing

the mean and vice versa. To break this mean-variance relationship, a specific statistical model is needed with relevant data transformations. In the case of polytomous data, which are usually described with an underlying Gaussian distribution, it is possible to apply the model proposed by SanCristobal-Gaudy *et al.* (2001). Stability genes controlling the environmental variability of this underlying Gaussian trait can be efficiently selected to reduce the underlying variability, although with limited impact on the observed scale.

11. Beyond canalisation, should a genetic effect be included in heteroscedastics models ?

Improvement of genetic models allows greater accuracy of breeding value estimations and an increase in the genetic progress of traits under selection. For instance, heteroscedastic models have been recently developed (Foulley and Quaas, 1995; Meuwissen *et al.* 1996; Robert-Granié *et al*. 1999). These models have increased the accuracy of breeding value estimations. But they did not assume a genetic determinism of the heteroscedasticity. If genetic factors are associated to this heterogeneity of variance, they should also be included in the model to fit better the reality. In this way, the model presented in formula 1 is a natural extension of the heteroscedastic infinitesimal model.

12. Model validation

The key point of the majority of the previous remarks is the genetic model underlying performance homogeneity. The biological reality is complex, several statistical models can be proposed. What is the impact of these models if the biological process is completely different ? Computer simulations may be performed to give clues to this question, but an experimental process should be a better way to approach the underlying genetical process.

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