Theor Appl Genet (1983) 65:317–322

Estimation of variance components and heritability in populations affected by disease: lymphoid leukosis in chickens*

J. S. Gavora[1], J. Chesnais[1,**] and J. L. Spencer[2]

[1] Animal Research Centre, Agriculture Canada, Ottawa, Ontario K1A OC6, Canada

[2] Animal Diseases Research Institute, Agriculture Canada, P.O. Box 11300, Station "H", Ontario K2H 8P9, Canada

Received January 6, 1983; Accepted February 14, 1983
Communicated by H. Aplanalp

Summary. The effects of disease, particularly when congenitally transmitted, on variance components and heritability were studied. Observations on lymphoid leukosis, a congenitally transmitted, viral disease of chickens, were used as the basis of the considerations but the results are deemed applicable to other situations where a population is similarly affected by a disease or another factor resulting in alteration of performance.

The numbers of pullets tested for lymphoid leukosis virus (LLV) shedding into eggs were 1785 in 1976 and 1699 in 1977. A comparison of the distribution of LLV shedders (approximately 8% of the birds tested) among sire and dam families with its binomial expectations supported earlier reports that only dams play a role in congenital transmission of LLV.

The effects of LLV infection on variance components and heritability were assessed in the 1976 data by comparing estimates from both LLV-shedders and non-shedders (population A) with estimates from non-shedders only (population B). Sire variances for age at first egg, number of eggs per hen housed, egg production rate, and egg weight were 3 to 18% greater in population A compared to population B. The corresponding differences in dam variances were generally larger (5 to 48%) while relative differences in individual variances were small (1 to 10%). Total phenotypic variances for the traits were 2 to 13% larger in population A than B. Corresponding changes in percent sire heritability ranged from −1 to 6%, and in dam heritability from −2 to 12%. The significance of these effects was not established with certainty due to standard errors of the estimates (9 to 13%). The study pointed out the need to consider possible effects of agents such as LLV on designing breeding plans, experiments and in data analyses.

Key words: Disease – Lymphoid leukosis – Chickens – Variance – Heritability

Introduction

Diseases disrupt or change body functions and may impair productive capacity of animals to various degrees and, in extreme cases, cause the death of the host. The lymphoid leukosis virus (LLV) is an example of an infectious agent that can reduce the productive capacity of chickens and yet only occasionally causes death. In a study by Gavora et al. (1980), subclinical LLV infections in Leghorn chickens were shown to be associated with delayed sexual maturity, lower egg production and egg weight, thinner egg shells, increased mortality from causes other than LL, as well as lower fertility and hatchability. Similar negative effects on production traits in egg stocks were reported by Garwod et al. (1981), Fadly and Okazaki (1982) and Maas et al. (1982). The negative effects of LL virus infection on egg production and mortality were confirmed in meat-type birds and a 5% lower broiler weight of LLV-infected chickens was reported (Gavora et al. 1982).

Ample evidence exists that LLV is vertically transmitted through the egg from the dam to some of her progeny. The sire does not seem to play a role in the congenital transmission of the disease (Spencer et al. 1980). The disease can also be transmitted horizontally from chicken to chicken and the frequency of such transmission seems to depend on rearing environment,

* Animal Research Centre Contribution No. 1011
** Present address: Animal Production Division, Agriculture Canada, Ottawa, Ontario K1A OC5, Canada

presence of maternal antibody, as well as other factors (Witter et al. 1966; Weyl and Dougherty 1977; Harris 1979; and Gavora et al. 1980).

It was shown by Gavora et al. (1980), that in the process of selection for high egg production the hens that shed LLV into their eggs tend to be inadvertently eliminated. Under these circumstances, part of the difference between the performance of a selected and control population is due to reduction in the frequency of the less productive LLV-shedder birds, rather than due to "true genetic gain", i.e., a genetic change in the selected population.

The objective of this study was to examine the effects of LLV infection on the estimation of variance components and heritability. Although the subject was considered in terms of LL, the phenomena described may occur in other situations where a part of a population is affected by a disease or another factor resulting in an effect on production. A secondary objective of the study was to compare the observed distribution of LLV infected birds among families with its expectation under the assumption of vertical and horizontal transmission of the virus.

Materials and methods

The strains of Leghorns used in this study were from a long-term selection study (Gowe and Fairfull 1980), and the data analysed, described earlier by Gavora et al. (1980), were from flocks of hens some of which were detected to shed LLV into their eggs. From the pullets hatched in 1976, 1785 individuals that were daughters of 236 sires and 882 dams were tested for shedding LLV into eggs. In 1977, the numbers were 1699 pullets from 341 sires and 999 dams. In the second year, the numbers of dams per sire and progeny per dam were very low. Therefore, only the data from the first year (1976) that had a more suitable structure for variance components estimation were used for the analysis of variance in this study. Data from both years were used to examine the distribution of LLV positive individuals among families.

Briefly, the birds were reared in group cages, and at 20 weeks of age transfered into individual cages in two laying houses. They were fed mash rations ad libitum throughout the experiment. In 1976, one half of the birds in the laying house received a diet containing 0.4% and the other 0.35% phosphorus. In 1977, one half of the birds were given a free choice of oyster shell spread on top of the layer ration containing 3.1% calcium. Eggs for tests (one egg per hen) on LLV were collected in 1976 from 1935 hens from 2 unselected control strains and 3 strains selected for high egg production and related economic traits. The egg collection period was between 255 and 287 days of age of the hens. In 1977, the 1848 eggs were similarly tested from hens of 3 control and 6 selected strains between 242 and 250 days of age. An LLV infected or shedder bird was defined as an individual that had LLV infection in the oviduct that was detected by tests on egg albumen.

Distribution of LLV positive individuals among families

In a population produced by random mating of sires each to several dams, and under the absence of vertical transmission of the virus from parents to progeny, the distribution of the LLV positive individuals among sire families is expected to follow the binomial distribution, and the expected relative frequency of families of size s, that will contain r individuals affected by LLV is given by

$$P = \binom{r}{s} p^r (1-p)^{s-r},$$

where p is the proportion of LL shedders in the population studied.

Frequencies of sire and dam families, observed in the study by Gavora et al. (1980), were expressed by strain according to the total number of individuals and the number of LLV shedders among the family members. Using the actual proportion of LLV shedders in each strain, the expected frequencies of families in the various categories were calculated, and the expected and observed frequencies of families of the various sizes and with the different number of LLV-shedder members were then summarized for all strains within the year.

The Chi-square test was used to compare the expected and observed frequencies of families in the various categories. For the test, categories with small expectations were combined within a given family size to reach expectation of at least 1. Thus in Table 1, within families of three pullets, the expected frequency of families with 3 LLV shedder birds was less than 1. Therefore, the categories of families of three pullets with 2 and 3 positive birds were combined giving 3.1 expected and 7 observed families. In some instances, such as in the case of family size 6 in Table 1, it was impossible to obtain two classes of families with expectations greater than 1 and the entire family size in the particular year was eliminated from the Chi-square comparison.

Effects of LLV infection on variance components and heritability

Individual data on the production traits of the 1976 hens were subjected to analyses of variance using the model:

$$Y_{ijklm} = u + a_i + s_{j:i} + d_{k:j:i} + n_l + an_{il} + w_{ijklm},$$

where

y_{ijklm} = individual observation,
u_i = population mean,
a_i = fixed effect of strain,
$s_{j:i}$ = random effect of sire within strain,
$d_{k:j:i}$ = random effect of dam, within sire, within strain,
n_l = fixed effect of nutrition treatment,
an_{il} = strain by nutrition treatment interaction,
w_{ijklm} = random individual effects.

Sire (σ^2_S), dam (σ^2_D), and individual (σ^2_W) variance components were estimated by equating the appropriate mean squares to their expectations and solving the resulting equations. Phenotypic variance was calculated as the sum of the above variance components and the components were used to obtain heritability estimates as follows:

sire heritability

$$h^2_S = \frac{4\sigma^2_S}{\sigma^2_S + \sigma^2_D + \sigma^2_W}$$

dam heritability

$$h^2_D = \frac{4\sigma^2_D}{\sigma^2_S + \sigma^2_D + \sigma^2_W}.$$

The analyses of variance and the corresponding estimates of variance components and heritability were first performed on all birds tested (population A). The LLV shedder birds were then excluded and the remaining data from the LLV negative birds (population B) were again analysed the same way. The effects of LLV infection on variance components and on heritability were estimated as the difference between the corresponding parameters estimated in population A and population B.

Results

Distribution of LL virus shedders within dam families

An example of the expected and observed frequencies of families of various sizes and with different numbers of LL virus shedders is in Table 1 which shows the data for dam families of pullets hatched in 1976, the first year of the study. Similar tables were prepared for the same pullets grouped into sire families, as well as for sire and dam families of pullets hatched in 1977.

In both 1976 and 1977 hatch years, the observed frequencies of sire families did not significantly differ from the frequencies expected on the basis of the binomial distribution. The respective numbers of degrees of freedom and Chi-squares were 45 and 33.48 in 1976 and 30 and 35.73 in 1977, indicating that no "clustering" of shedder daughters occurred in specific sire families and thus supporting previous findings that sire does not affect the frequency of LL virus shedders in his progeny.

In contrast, the observed frequencies of the various categories of dam families were significantly different ($P < 0.01$) from those expected under the assumption of no effect of dam on the frequency of LLV shedders. The respective numbers of degrees of freedom and Chi-square values were 14 and 53.06 in 1976 and 8 and 37.01 in 1977. As stated above, the transmission of LL through the egg is expected to cause an excess of families with extreme (high and low) frequencies of LL positive individuals and a shortage of families with intermediate frequencies. The direction of the deviations of observed from expected frequencies is illustrated in Table 1. It was in general agreement with the anticipated deviations thus providing supporting evidence for the existence of vertical transmission of the LL virus from dam to progeny through the egg. Similar general patterns were also found in the 1977 dam family data.

Effects of LLV infection on variance components and heritability

The estimates of variance components and heritability from the 1976 population containing only LLV negative birds (population B), as well as the effects of LLV infection calculated as the difference between the estimates from population A, and from population B are in Table 2.

For all four traits analysed, the size of the sire variance in population A was somewhat larger than in population B. The differences, relative to variance components in population B, were 3, 8, 13 and 18% respectively for egg weight, hen-housed egg number, egg production rate, and age at first egg. The increases of dam variance due to LLV infection were generally larger and, relative to the population B estimates, amounted to 5% for egg production rate, 20% for egg weight, 45% for hen-housed egg number and 48% for age at first egg. The changes in individual variance components were low with respective relative increase of 10 and 9% in the individual variance of egg production rate and in hen-housed egg production. The relative increases in the individual variances of age at first egg and egg weight amounted to less than 1%. The

Table 1. Expected and observed frequencies of dam families of various sizes and with different numbers of shedders of lymphoid leukosis virus (LLV). Pullets hatched in 1976; all strains combined

No. of LLV shedders in family	No. of pullets in dam family						
	1	2	3	4	5	6	7
	Expected/observed no. of families						
0	280.1/272	214.1/227	137.7/147	70.0/72	33.2/38	4.1/4	3.6/5
1	46.9/55	37.6/17	25.2/12	12.7/9	6.9/2	0.8/0	1.2/0
2		3.3/11	2.9/3	1.2/1	0.8/0	0.1/1	0.2/0
3			0.2/4	0.1/0	0.1/1	0/0	0/0
4				0/2	0/0	0/0	0/0
5					0/0	0/0	0/0
6						0/0	0/0
7							0/0
Total	327	255	166	84	41	5	5

Table 2. Effects of LLV infection of variance components and heritability of egg production and related traits in chickens

Variance or heritability[b]	Variance component or heritability estimates (%) from population A/differences between estimates from population A and B[a]			
	Age at first egg (days²)	Egg production rate (%²)	Hen-housed egg production (eggs²)	Egg wt. (g²)
σ_S^2	13.7/2.4	7.5/1.0	120/9	1.82/0.05
σ_D^2	5.2/2.5	13.2/0.6	200/89	0.66/0.13
σ_W^2	67.3/0.7	89.2/9.3	1581/147	9.00/0.02
σ_P^2	86.2/5.6	109.9/10.9	1901/245	11.49/0.20
h_S^2	64/6	27/1	25/−1	64/1
SE[c]	± 12	± 9	± 9	± 12
h_D^2	24/10	48/−2	42/12	23/4
SE[c]	± 13	± 13	± 12	± 13

[a] Population A contained data from both LLV shedder and non-shedder birds. Data on LLV shedders were eliminated from population B

[b] σ_S^2 = sire variance component, σ_D^2 = dam variance component, σ_W^2 = individual variance component, σ_P^2 = phenotypic variance ($\sigma_S^2 + \sigma_D^2 + \sigma_W^2$), h_S^2 = sire heritability, h_D^2 = dam heritability

[c] Standared error of the estimates from population B

size of the individual variance components was generally larger than the size of sire and dam variance components combined. Therefore, the changes in the phenotypic variance, calculated as the sum of the sire, dam and individual components, attributable to the presence of LLV-infected birds in the population were relatively small. Relative to the size of δ_P^2 population B, they amounted to increases of 2, 7, 10 and 13% for egg weight, age at first egg, egg production rate and number of eggs per hen housed respectively.

The influence of the above increases on the size of the variance components is reflected in changes of the corresponding estimates of heritability (Table 2). In absolute terms, the sire component heritabilities in LLV-free birds were higher for egg weight and for the two egg production traits. Dam heritabilities from the same population were smaller than sire heritability for age at first egg and egg weight but were almost double the size of the sire heritabilities for the two egg production traits. The relative effects of LLV infection were found to be generally smaller on sire than on dam heritability estimates. They amounted to an average increase of 3% over all four estimates of sire heritability shown in Table 2 while the corresponding average increase in the dam heritabilities was 21%, relative to the heritability estimates in population B.

Discussion

The absence of significant deviations in the distribution of LLV positive birds among sire families from the binomial distribution expected, provided further support for the generally accepted hypothesis that sire does not play a role in the congenital transmission of the disease. The detection of significant deviations of this distribution from its expectations in dam families was in agreement with the known role of the dam in vertical transmission of the disease.

The effects of LLV infection on variance and its components would be best assessed by comparing the size of these statistics in a LVV-free population and in a population of LLV-positive birds. Unfortunately, there were only 150 LLV-shedder hens in the 1976 population used in this study. This number was considered insufficient for a separate analysis and, therefore, as described above, LLV effects were estimated as differences in the size of variance components from a mixture of LLV-positive and -negative birds, and from negative birds only. As will be discussed below, the size of LLV effects on σ_P^2 depends on the proportion of the population affected and, therefore, it should be noted that only about 8% of the birds used in this study were LLV-positive.

The fact that the increases in variance due to sires were relatively small, along with the evidence of no effect of sires on the distribution of LLV positives among families discussed above, suggest that changes in the sire variance components resulting from LLV infection in a population are of little importance and those observed in this study may be a result of sampling, since populations A and B were not identical (population B being a sub-set of population A). If real, the increases may have resulted from differential abili-

ties of the progenies of various sires to cope with LLV infection.

Dam variance components in the population containing both LLV-positive and -negative birds were on the average about 30% higher that those from the LLV negative birds only. This increase can be mostly attributed to the dam's role in the congenital transmission of the disease but may also be reflecting differential responses of individual full sib families to LLV infection. The small relative increase in the individual variance in LLV negative birds, resulted from the large size of these components (Table 2). For the two egg production traits, the absolute increase in the individual variance was larger than the corresponding increases in sire and dam variances combined. The total increase in the phenotypic variance was of a magnitude that should be of concern to breeders and researchers.

The increases in the phenotypic variances (Δ_V) expected to result from the effects of lymphoid leukosis were also estimated using the formula developed in the Appendix. The estimates were based on the frequency of LLV-positive birds (0.08) and overall size of the effects d (3 days delayed sexual maturity, 4.2% lower egg production rate, 30 eggs less per hen housed and 1.5 g lower egg weight in LLV positive birds) reported earlier (Gavora et al. 1980). The resulting Δ_V were much lower than the increases in the phenotypic variances shown in Table 2. The exception was egg weight where Δ_V from the above formula had a value of 0.1, compared to 0.2 in Table 2. This indicates that, at least for sexual maturity and egg production traits, sizes of d must vary greatly and that the product pVd (see the Appendix), not considered in these calculations of Δ_V, is likely of a significant magnitude. This is in agreement with the earlier observation of relatively large strain differences in the size of d mainly for egg production traits (Gavora et al. 1980), and with the suggestion by Harris (1979), that congenitally infected birds are more severely infected by LLV than horizontally infected birds.

The changes in the size of sire and dam heritabilities observed in this study are a result of the effects of LLV infection on variance components discussed. The effects of LLV infection on sire heritabilities were relatively small and should be considered negligible. However, at least for hen-housed egg production and age at first egg the increases in dam heritabilities are substantial. The size of LLV effects on sire heritability was generally smaller than the errors of the estimates from population B (Table 2). The effects of LLV infection on the heritability estimated from the dam variance components were larger but still mostly fell within the standard errors of the estimates and larger population sizes would be required to estimate their size more accurately.

The results point to the possible effects of agents such as LLV on variance components and heritability. They are generally ignored by geneticists, breeders and researchers but their further investigation and consideration in the design of breeding programs, experiments and in data analyses would appear desirable and potentially beneficial.

Acknowledgements. The birds used in this study and the data on all traits except the tests for LLV were part of Dr. R. S. Gowe's long-term selection study and the use of the data for the analyses is gratefully acknowledged. The authors wish to thank Drs. Nagai, Harris and Abplanalp for critical reviews of the paper and valuable suggestions for its improvement

Appendix

Estimation of the increase in phenotypic variance as a consequence of disease effects, based on the frequency of affected individuals and size of the effect

The expected size of the increase in the phenotypic variance can be derived under certain assumptions. For instance consider that all individuals that are affected by a disease will have their performance changed by a fixed amount d, and a proportion p of a population of size n is affected by the disease. The number of affected individuals in the population is:

$m = pn.$

We further define the estimate of variance in a disease-free population as:

$$V_1 = \frac{\sum_1^n (x_i - \bar{x})^2}{n-1}, \quad \text{where} \quad \bar{x} = \frac{\sum_1^n x_1}{n}.$$

Analogously in a population of the same size, affected by the disease, the variance is estimated as

$$V_2 = \frac{\sum_1^n (y_i - \bar{y})^2}{n-1}.$$

The mean of the latter population can be expressed as

$$\bar{y} = \frac{\sum_1^m (x_i + d) + \sum_{m+1}^n x_i}{n} = \bar{x} + pd.$$

Assuming that

$$E(\bar{y}) = E(y) \quad \text{and} \quad E\sum_1^m (x_i - \bar{x}) = E\sum_{m+1}^n (x_i - \bar{x}) = 0,$$

the variance in the disease affected population can be estimated as

$$\hat{V}_2 = \frac{1}{n-1}\left[\sum_1^m (x_i + d - \bar{y})^2 + \sum_{m+1}^n (x_i - \bar{y})^2\right]$$

$$= E(\hat{V}_1) + \frac{1}{n-1} d^2 p(1-p).$$

Thus the expected difference between the estimates of variance in a disease affected and a disease free population will be

$$E(\hat{\Delta}_V) = E(\hat{V}_2 - \hat{V}_1) = \frac{n}{n-1} d^2 p(1-p),$$

and the expectation of the difference between population variances

$$E(\Delta_V) = E(V_1 - V_2) = d^2 p(1-p).$$

The above expectations of Δ_V assume a constant d, i.e., it is assumed that the effect of the disease will be the same in all affected individuals. However, it is likely that the size of d will vary. With a mean d and variance V_d, and no correlation between the basic, disease free performance of the individual and the size of the disease effect ($cov(d, x) = 0$), the expectation of the difference between population variances becomes

$$E(\Delta_V) = \bar{d}^2 p(1-p) + pV_d.$$

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