SOME ASPECTS OF AGEING FOR EGG PRODUCTION OF LEGHORNS

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Summary

White Leghorn strains were crossed reciprocally in a complete factorial mating producing 6 pure strains and 30 strain crosses which were kept for two laying cycles: 133-496 days of age (d) and 547-909 d (364 and 363 d, respectively). Hens were individually housed for lay in four two-tiered batteries of cages. Strain additive effects (Ai), strain sex-linked effects (Zi), strain-cross heterotic effects (hi) and residual effects were calculated using regression. Phenotypic performance for egg production decreased with age. The magnitude of genetic effects increased with age as did their variation. Environmental variation also increased with age. Heterosis had a generally positive influence on late performance, which was greater than that of other genetic effects. Overall, these results suggest that genetic expression does not become fixed and that the use of performance data from late in the first laying cycle or from the second cycle would allow the utilization of genetic variation not available earlier.

I. INTRODUCTION

Many people portray the life cycle of organisms as an extremely complex set of events. In reality, life cycles are very simple when you eliminate unnecessary detail: only two events are especially important, birth and death. Everything between them comprises a process called ageing. For example, are sexual maturity and reproduction important? Yes, they are, after all, ageing processes. Ageing is the sum of many genetic, physiological, developmental and environmental (internal and external) processes. It is not a single isolated event and does not result from a single mechanism. Most physiological systems are affected by ageing, for instance the neuro-endocrine, the immune, and the reproductive systems. Ageing is characterized mainly by declining vigor, reduced viability and increasing environmental sensitivity.

On an individual basis is ageing important? Possibly not: there is overhead cost in producing an independent adult and on an individual basis reducing the reproductive value of each adult in a retrogressive process does not seem worthwhile. On a population basis, however, replacement units must be continuously available and the ability of a population to react to crisis or adapt would be reduced without ageing to help get rid of those obsolete models. In evolutionary terms, it allows for genetic flexibility. In a population sense, it allows for growth and change.

Theories of ageing revolve around two central themes: i) ageing results from an accumulation of random events; and ii) ageing is a programmed, time-dependent process.

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Some theories of ageing (see Esser and Martin, 1995) are: 1) the somatic mutation theory - an accumulation of random, deleterious somatic mutations; 2) the error catastrophe theory - an accumulation of random, deleterious mutations resulting in transcription and translation errors; 3) mutation accumulation theory - an accumulation of random, deleterious mutations expressed only late in life, modifier genes delay expression; 4) the antagonistic pleiotropic theory - alleles favorable to early fitness and deleterious to late fitness become fixed; and 5) the disposable soma theory - maximum fitness balances somatic maintenance and reproduction. In evolutionary biology, a circular theory has emerged. Specifically, assuming genetic variation in life patterns, if the intensity of natural selection for age specific fitness declines with age, then senescence developed as a by-product of evolution. There are several problems with evolutionary explanations: the theory requires genes with high early fitness with late low fitness trapped by natural selection. However, natural selection has little effect late in life and there is no satisfactory mechanism for random events.

Information on ageing processes does not always aid understanding of genetic mechanisms of ageing in a straightforward manner. The rate of protein synthesis declines with age, but there are no qualitative differences with age (Arking and Dundas, 1989). The ability of the cells to communicate with each other and receive information from the environment (signal transduction) alters with age (Roth, 1990). This does not seem to be related to the concentration of receptors as some decrease with age, other remain unchanged, while still others increase. However, the ability of steroid hormone receptors to bind to DNA acceptor sites declines with age. The expression of structural genes varies with age (Thaker et al., 1993): some have high early expression then decline; others have low early expression that increases to a maximum then decline; still others steadily increase expression to a high level late in life. In humans the most probable causes of ageing are genetic damage (Strehler, 1989) and genetic instability (Slagboom and Vijg, 1989). Also, there are several examples of specific genes directly causing ageing in several species, for example Werners syndrome in humans. Individual variation in regulation exists (Cinader, 1989a,b): activation of genes later in life and delayed manifestation of activated genes. In mice, inactivated X-chromosome genes are activated later in life (Cattanach, 1974; Wareham et al., 1987). What conclusions can be drawn from this information? First, ageing and its effects are very complex. Second, these observations support several theories of ageing. Third, no single theory of ageing is adequate.

Heterosis is the deviation between the cross mean and the mean of the parents. Genetically, it is due to interactions: among alleles (dominance) or among different genes (epistasis). Heterosis seems to provide buffering (homeostasis). In laying hens, it is larger under adverse conditions and in traits associated with fitness (Fairfull et al., 1987; Fairfull, 1990).

II. MATERIALS AND METHODS

The information reported here derives mainly from a study conducted with six SC White Leghorn strains from three genetic base populations developed at the Animal Research Centre in Ottawa. All the six strains were selected primarily for egg production from housing to 273 days of age, and for egg weight, eggshell strength, viability, fertility, hatchability and egg quality traits. The strains were crossed reciprocally in a factorial design with 56 males and 112 females used to produce each of six pure strains and 30 strain crosses.

All chicks were reared in four batteries of a 3-tier cage system in a windowless house to 132 days of age. At 133 days of age, the pullets were housed one per cage (20.3 cm wide) in four batteries of a 2-tier cage system in a windowless house. For each cross and pure
strain, 57 pullets were housed. All mash diets were fed *ad libitum* throughout the study. Birds were vaccinated for Marek's disease, avian encephalomyelitis, bronchitis and Newcastle disease. Artificial light was provided for 24 h after hatch and then reduced to 6 h daily until 132 days of age. At housing, light was augmented to 8 h daily and increased by 30 minutes per week to a maximum of 16 h daily. At the end of the first egg production cycle (496 days of age), all birds were induced to molt (Fairfull, 1982). The second egg production cycle started at 546 days of age when 16 h light daily was resumed.

Egg production was recorded 5 d per week from 133 to 496 days in the first cycle and from 547 to 909 days of age in the second cycle. For analysis, the egg record of each hen was divided into four week periods that started with the week the first egg was laid, so that each cycle had 11 periods. Mortality was recorded daily and all dead hens were necropsied. Hens that died as the result of an accident were removed from consideration. Strain additive effects (*A*)\(_i\), strain sex-linked effects (*Z*)\(_i\) and strain-cross heterotic effects (*h*)\(_{ij}\) were estimated by regression (Robison *et al.*, 1981) using the model:

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Y_{ijk} = \mu_p + \frac{1}{2}A_i + \frac{1}{2}A_j + Z_i + h_{ij} + \epsilon_{ijk},
\]

where \(Y_{ijk}\) = the observation on the \(k^{th}\) individual of a mating between a strain \(i\) sire and a strain \(j\) dam; \(\mu_p\) = mean of the \(p\) pure strains, \(i = 0\) when \(i = j\) or \(i = 1\) when \(i \neq j\), and \(\epsilon_{ijk}\) = a random residual effect.

### III. RESULTS

(a) **Performance**

Egg production decreased within cycles and across cycles for both crosses and pure lines (Liljedahl *et al.*, 1984, 1998). After an initial small increase from the first to the second period in the first cycle, egg production declined steadily. In the second cycle, egg production recovered somewhat, then decreased to the end of the cycle (Figure 1). In the second cycle, the rate of decline was greater than that of the first cycle.

![Figure 1. Mean egg production by period.](image)
(b) **Additive genetic effects**

Additive genetic variation for egg production increased with age within each cycle and across cycles (Figure 2; Liljedahl *et al.*, 1984, 1998). Also, the magnitude of strain somatic ($A_s$) and sex-linked ($Z_s$) additive genetic effects for egg production increased with age within each cycle and across cycles. Patterns of age changes in total additive genetic effects ($A_s, Z_s$) varied widely from strain to strain (Figure 3) and differences among strains increased notably with age.

(c ) **Heterosis**

Generally, strain cross heterotic effects ($h_y$) increased with age in each of the two laying cycles and from cycle 1 to cycle 2 (Figure 4). Heterosis was consistently large and positive although three crosses exhibited negative heterosis in some periods. In addition, the large differences among strain crosses for heterotic effects increased clearly across ages and the variance of heterotic effects increased with age (Liljedahl *et al.*, 1998). Thus, genetic interactions seem to be more important to late as opposed to early reproduction.

![Graph showing additive genetic variance by period.](image)

**Figure 2.** Additive genetic variance by period.
Environmental variation for egg production increased with age within each cycle and the increase in the second cycle was greater than that of the first cycle (Figure 5; Liljedahl et al., 1984, 1998). The environmental variance increased in an almost linear fashion across the first laying cycle. In the second cycle, the increases became more pronounced in the earlier periods, but rose to a maximum by about the 20th period after which it declined slightly (Figure 5).
Figure 5. Environmental variance by period.

IV. DISCUSSION

(a) Strategies of ageing

The great variation among genotypes across ages in genetic effects implies that genotypes apply different genetic strategies to reduce the negative impact of ageing. Both additive and non-additive genetic effects are important in ageing; however, heterotic effects are clearly larger than other genetic effects and exert more influence over the effects of ageing. Genetic interactions increase in importance with respect to reproduction as chickens increase in age. Basal metabolic rate is lower and reproductive performance higher in crosses. Heterosis is greater in fitness traits which are influenced by both dominance and epistasis (Fairfull et al., 1987). Also, heterosis is greater under adverse environmental conditions. The increasing heterosis with age may reflect in part a deteriorating internal environment. Heterosis seems to provide a capacity to buffer (reduce) the deleterious effects of ageing. This might be expected as heterozygosity increases flexibility: 2n versus n pathways. Conclusion: gene interactions are an important genetic mechanism in ageing and extended performance.

These results and other recent findings (see Gowe and Fairfull, 1985, 1986) cast doubt on the validity of using only early records in selection. The variation of genetic effects for egg production and other fitness traits rises markedly with age. This suggests that genetic expression does not become fixed and that consideration of reproductive traits at later ages may allow the utilization of genetic variation not available earlier.

(b) Influence of the Environment

The environment has a great influence on egg production at all ages. Also, increases in environmental variation with age are proportionately greater than those of genetic effects. In examining causes of mortality, only renal failure showed a significant age trend: it was higher at the end of each cycle. This suggests that diet may be an environmental factor that is
important in extended performance. Perhaps, tailoring layer diets to the physiological needs of hens at various stages of life could reduce negative environmental influences and improve extended performance.

V. CONCLUSIONS

First, gene interactions are very important in buffering the deleterious effects of ageing. Second, different genotypes use different strategies to diminish the effects of ageing.

REFERENCES