MAGNIFICATION THROUGH COMPETITION OF GENETIC DIFFERENCES IN YIELD CAPACITY IN CARP*

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Received 7.xi.73

SUMMARY

Ten experiments were carried out during the period 1961-69. In each experiment a pair of genetically different groups of carp was tested simultaneously for differences in growth rate in mixed ponds as well as in separate ponds. The aim of the experiments was to estimate the genetic correlation of relative growth rate under inter-group competition in mixed ponds and in the absence of inter-group competition in separate ponds. The estimate of the genetic correlation coefficient was found to be 1.0, and that of the regression coefficient of growth rate in mixed ponds on growth rate in separate ponds was 2.0. The inter-group genetic variance in mixed ponds was four times higher than that of the variance in separate ponds. The higher variance in mixed ponds was partitioned into a component dependent on growth rate in separate ponds and a second independent component. The high genetic correlation means that the dependent component could account for all the amplification of the variance in the mixed ponds and that, apparently, in carp there are no specific genetic factors affecting growth rate under competition. The practical implications of this result for carp breeding is that selection for the improvement of yield capacity can be performed more effectively in mixed ponds than in separate ponds. A theoretical model for analysis of competition is presented and discussed.

1. INTRODUCTION

In selection programmes of fish aimed at improving yield capacity the units under selection (individuals or groups of fish) may be grown in separate ponds (or other containers), *i.e.* in the absence of competition between the units of selection. The major advantage of this method is that yield capacity is measured directly. Its disadvantages are the need for a large number of ponds (containers) to overcome the confounding of genetic variation with environmental differences between containers. Because of these drawbacks, selection is usually performed on individuals or groups grown within the same pond (mixed ponds). Here the fish are subjected to mutual competitive interactions, therefore *yield capacity* cannot be measured directly and the measured and selected *character is relative growth rate in mixed stocking*. Since the breeders' interest is in yield capacity, evaluation of results obtained from mixed ponds requires an estimate of the genetic correlation of growth rate in the presence and absence of competition. The critical importance of this association has been pointed out (Schaeperclaus, 1958; Kirpichnikov, 1966), but no attempt has been made to estimate it in the past.

If competitive ability is unrelated or negatively correlated with yield capacity, growth rate under competition may be a poor measure of yield capacity. But if competitive ability is positively correlated with yield

* This paper is part based on the Ph.D. thesis of S. W. Wohlfarth submitted to the Hebrew University, Jerusalem.

capacity, genetic differences in yield capacity will be *magnified* in mixed stocking. In the latter case relative growth rate in mixed stocking may be a better measure of yield capacity than growth rate in separate ponds.

The relationship of competitive ability to selection for higher yields in cultivated plants, particularly cereals, has been a subject of numerous investigations (Sakai, 1955; Donald, 1963; McGilchrist and Trenbath, 1971; Norrington-Davies, 1972; and others). Sammeta and Levins (1970) reviewed an extensive list of reports on competition in plants and animals and concluded: "... The relation between competitive ability and other fitness components may be different in different organisms and competitive situations. So it is important not to assume *a priori* that productivity and competitive ability are positively correlated."

In order to estimate the correlation between growth rate in separate and in mixed ponds in carp a series of 10 experiments was carried out during the period 1961-69. In each experiment two genetically different groups of carp were tested simultaneously in mixed and separate ponds. A high genetic correlation and a strong magnification of inter-group differences in mixed ponds were found. These experiments, their analysis and interpretation are described in the present paper.

2. THE GENETIC STOCKS

The common carp (*Cyprinus carpio* L.) has been cultivated in ponds in China since at least the fifth century B.C. In the Middle Ages it was introduced to Europe for use as a cultivated fish (Hickling, 1968). Carp cultivation in ponds in Israel was initiated in 1939 through a number of introductions, mainly from Europe (Tal and Shelubsky, 1952; Yashouv, 1955;

TABLE 1
*The types of carp tested**

Experiment No.	1	2	3	4	5	6	7	8	9	10
Group A	FS	FS	FS	J-In	J-In	Out	Out	Out	In	In
Group B	In	Out	In	L-in	L-In	In	In	In	Out	In

*FS = Full sibs of an unrelated pair of parents.

In = Inbred for one to several generations.

Out = Outbreds

J = Jumpers, the largest individuals selected from a spawn.

L = Laggards, the smallest individuals selected from a spawn.

In all cases other than FS, several females and several males participated in the spawn.

Moav *et al.*, 1964). The carp population of Israel, including all the groups tested in this series of experiments, is thus mostly of European descent.

The intra-group relations of the tested groups, *i.e.* *full-sibs*, *inbreds* and *outbreds*, are listed in table 1. The two groups of carp tested in eight of the ten experiments were chosen from the 15 to 25 groups of carp tested annually in our research programme (Wohlfarth *et al.*, 1965; Moav and Wohlfarth, 1966). A deliberate non-random choice of dissimilar groups ensured the presence of genetic differences between the two groups tested in each experiment. Experiments 4 and 5 differed from the other eight experiments since here the two tested groups consisted of the largest and smallest individuals selected from the same spawn. Thus, in these two experiments, the

expected difference between the two groups was not only of genetic origin but also of environmental origin of the so-called developmental type.

3. EXPERIMENTAL METHODS

All the tests were carried out in earthen ponds. In experiments 1 to 5, both *separate* and *mixed* ponds were small experimental ponds of 400 or 1000 m² at the Fish Aquaculture Research Station, Dor. The same ponds also served as the separate ponds in experiments 6-10, but the *mixed* ponds in these experiments were much larger (5000 to 30,000 m²) and were located at the fish farms of the Carp Breeders Union (Wohlfarth *et al.*, 1965). Since in *mixed* ponds there is no confounding of pond differences with genetic differences between the groups, each *mixed* pond could be located at a different farm. The use of larger ponds enabled larger samples of fish to be tested. In addition to the pairs of groups relevant to the present study, the large *mixed* ponds in experiments 6 to 10 were stocked with other groups of fish. These are considered in the context of the present study as a part of the external environment. Details of the *mixed* ponds of the experiment carried out in 1964 are given by Wohlfarth *et al.* (1965).

Spawning was carried out in late April, each group in a different pond. As a rule, the fish spawned within 12 hours of being introduced into the spawning pond, and the eggs hatched within three to four days. A few weeks after spawning, fry were transferred from the spawning ponds to the nursery ponds, where they grew to a mean weight of 20 to 50 g. The nursed fingerlings were marked by branding (Moav *et al.*, 1960) and stocked into the experimental ponds in June to August. Experiments 1 and 2 differed from the remaining experiments in that they were initiated in February and March, respectively, and were stocked with fish spawned during the previous year. These fish differed from those of other experiments in their age, sexual maturity and higher initial weights. The tests lasted from 70 to 140 days. At the termination of each test, the ponds were drained and all the fish removed, sorted, counted and weighed.

4. CORRECTIONS FOR ABSOLUTE MEAN WEIGHTS, INITIAL WEIGHTS AND FINAL POND DENSITY

(i) *Absolute mean weight*

The final mean weights of the tested groups varied considerably between experiments and even between different mixed ponds of the same experiment. This variation was largely due to environmental factors such as number of fish per unit area, pond fertility, management, etc. Since the standard-deviation of weight in carp is approximately proportional to the mean weight, an adjustment to a computed mean weight of 500 g served to reduce the variation between standard-deviations of the different experiments. The factor 500 g was chosen for the correction because it is a round number close to the mean of all the ten experiments and to the mean of the market weight of carp in Israel. The correction was made by multiplying the difference in weight gains between the two tested groups, (d), by the factor ($500/W$). In *separate ponds* W was the mean weight of the two tested groups over *all* the separate ponds of a given experiment. The large weight differences between different *mixed* ponds even of the same experiment (especially in experiments

6-10) required an individual adjustment for each *mixed* pond. Thus for a given mixed pond, W was the mean weight of the two tested progenies in that pond.

(ii) *Initial weight*

Considerable variation in mean initial weight occurred between the two groups tested in each experiment. This was a result of lack of complete control over environmental conditions in the nursery ponds. Since weight gain in fish is positively correlated with initial weight, groups stocked at higher initial weights (due to chance favourable conditions in nursery ponds) tend to grow faster than those stocked at lower initial weights. In order to remove this bias the observed weight gains were corrected as follows (Wohlfarth and Moav, 1972): $Y = Y' - b(X - X.)$, where

Y = corrected weight gain (it is uncorrelated with initial weight and taken as a measure of growth capacity),

Y' = observed weight gain,

X = initial weight,

$X.$ = mean initial weight of the two tested groups, and

b = coefficient of linear regression of weight gain on initial weight.

b is a decreasing function of initial weight and an increasing function of weight gain. Therefore it requires empirical estimation for different experiments (Wohlfarth, 1971; Wohlfarth and Moav, 1971). In the *mixed* ponds of the present experiments b varied between 2 to 5. In separate ponds the value of b is considerably *lower* than in mixed ponds, and therefore no correction was applied. The higher value of b in *mixed* ponds is due to competitive interactions between the mixed groups which magnify differences, resulting from variation in initial weights (Wohlfarth, 1971).

(iii) *Final fish density*

The separate ponds within each experiment were of equal size, stocked with equal number of fish and treated similarly. Nevertheless, during the test period unequal mortality created some differences in fish number. Since weight gain is a decreasing function of density (number of fish per unit area) (Hepher, 1967), the average weight gain per fish tended to be higher in ponds with higher mortality. This distortion was corrected by multiplying the average weight gain of each pond by the factor $(n/n.)$, when n is the final number of fish in a given pond and $n.$ is the average of all the ponds of a given experiment. This adjustment makes the assumption that yield is independent of fish density, *i.e.* a complete compensation of mean growth per fish for differences in density. This seems a reasonable assumption when relative mortality is low and varies little between ponds.

5. MODELS

We shall treat the tested groups of fish as a sample drawn from a population of *average genotypes* grown under two sets of environmental circumstances, *separate ponds* and *mixed ponds*, defined respectively *Environment one* (E_1) and *Environment two* (E_2). The genetic value of growth rate in E_1 is designated g_1 (measured as a deviation from the population mean), and its variance $\sigma_{g_1}^2$. The variable g_1 may be considered as a measure of the relative efficiency of

exploiting external resources in the absence of competition with other groups of carp and is referred to as *yield capacity*. The genetic value of growth rate in E_2 designated (g_2), and again measured as a deviation from the population mean in E_2 , may be presented as a linear function of three components: g_1 , competitive ability which is dependent on g_1 (αg_1), and competitive ability which is independent of g_1 (I),

$$g_2 = g_1 + \alpha g_1 + I = (1 + \alpha)g_1 + I. \quad (1)$$

α is the *amplification factor* measuring the average proportion of genetic changes in E_2 due to competition which is fully predictable by growth capacity in E_1 . On the other hand, I is unrelated to efficiency of exploiting external resources. For distinction between the two genetic components of competition αg_1 and I , the latter will be referred to as the genetic component of *independent aggressiveness*.

The genetic parameters of interest, derived from the model defined by equation 1, may now be presented.

The genetic variance in Environment 2,

$$\sigma_{g_2}^2 = (1 + \alpha)^2 \sigma_{g_1}^2 + \sigma_I^2. \quad (2)$$

The difference in genetic variances of E_1 and E_2

$$(\sigma_{g_2}^2 - \sigma_{g_1}^2) = \alpha(2 + \alpha)\sigma_{g_1}^2 + \sigma_I^2. \quad (3)$$

The covariance of g_1 and g_2 ,

$$\sigma_{12} = (1 + \alpha)\sigma_{g_1}^2. \quad (4)$$

The coefficient of genetic regression of g_2 on g_1 ,

$$\beta_g = \frac{\sigma_{12}}{\sigma_{g_1}^2} = (1 + \alpha). \quad (5)$$

The coefficient of genetic correlation of g_2 and g_1 ,

$$\rho_g = \frac{(1 + \alpha)\sigma_{g_1}^2}{\sigma_{g_1}((1 + \alpha)^2 \sigma_{g_1}^2 + \sigma_I^2)^{\frac{1}{2}}} = \left(\frac{(1 + \alpha)^2 \sigma_{g_1}^2}{(1 + \alpha)^2 \sigma_{g_1}^2 + \sigma_I^2} \right)^{\frac{1}{2}} \quad (6)$$

Note that when $\sigma_I^2 = 0$ then $\beta_g = 1$.

The fundamental biological difference between the two genetic parameters I and α (or αg_1) is shown by their different contributions to the above regression and correlation coefficients. Both α and I increase the genetic variability in E_2 over that in E_1 , and both result from inter-genotype competition. However, α does not change the rankings of the different genotypes (groups) while I , which may be controlled by a different set of genes than g_1 , reduces the correlation between growth rates of the two environments.

Conventionally the present problem is handled by a different model than that defined in equation 1. The conventional linear model partitions the j th genotype of the i th environment into the following three components: the environmental effect (a_i), the average genetic effect (g_i) and the genotype-environment interactions (ga_{ij}),

$$g_{ij} = a_i + g_j + ga_{ij} \quad (7)$$

(note that a_i is absent from equation 2 because g_2 was defined as a deviation from the mean of environment 2 rather than from the general mean).

$i = 1$ and $i = 2$ represent, respectively, *separate* and *mixed* stockings and $j = 1, 2, \dots, \mathcal{J}$ the tested groups (genotypes). The interaction ga_{ij} can be divided into two components, one a linear function of the average genotype (γg_j) and the second independent of g_j , (I'). That is:

$$ga_{ij} = \gamma_i g_j + I'_{ij}, \quad (8)$$

and by substitution into equation 7,

$$g_{ij} = a_i + g_j + (\gamma_i g_j + I'_{ij}) = a_i + (1 + \gamma_i)g_j + I'_{ij}. \quad (9)$$

This equation has the advantage that it can be applied to more than two stocking conditions (environments), and when only two stocking conditions are involved, as in the present case, it can be simplified as follows:

$$\gamma = \gamma_2 = -\gamma_1 \quad \text{and} \quad I' = I'_2 = -I'_1$$

so that

$$g_{1j} = a_1 + g_j - \gamma g_j - I'_j = a_1 + (1 - \gamma)g_j - I'_j$$

while

$$g_{2j} = a_2 + g_j + \gamma g_j + I'_j = a_2 + (1 + \gamma)g_j + I'_j$$

or, generally,

$$g_{ij} = a_i + [1 + (-1)^i \gamma]g_j + (-1)^i I'_j. \quad (10)$$

When $i = 2$ the coefficient in front of g_j is $(1 + \gamma)$ and it equals the regression coefficient of mixed stocking ($i = 2$) on the average genotype (over mixed and separate stocking). Similarly, $(1 - \gamma)$ is the regression coefficient of separate stocking on the average genotype. The coefficient $(-1)^i$ obviates the need of an absolute value which was used in similar circumstances (*e.g.* Bucio Alanis, 1966).

γ and I' of equations 9 and 10 are equivalent, respectively, to α and I of equation 2, and their functional relationships are given by the following two equations,

$$\gamma = \frac{\alpha}{2 + \alpha} \quad \text{and} \quad I' = \frac{I}{2 + \alpha}. \quad (11)$$

Bucio Alanis (1966) partitioned the genotype-environment interaction term into a linear function of the environmental deviation ($\beta_j a_i$) and a residual independent component (δ_{ij}). This concept was further elaborated in a series of reports (*e.g.* Perkins and Jinks, 1968; Freeman and Perkins, 1971). Their model, corresponding to equation 9, can be presented (in the present notations) as

$$g_{ij} = (1 + \beta_j)a_i + g_j + \delta_{ij}. \quad (12)$$

In all these reports the writers were primarily interested in expressing genotype-environment interactions as functions of the environment (a_i). This, in contrast with the present study where the interaction is expressed as a function of the genotype (g_j). The difference is due to the fact that here interest is centred exclusively on the correlation of performance in the absence and the presence of inter-genotype competition. The environmental deviation a_i represents different states of intra-specific competition rather than variation in physical attributes. Furthermore, for the present problem there is no need to estimate a_i , and it even may be assumed to be zero (for this reason it has been omitted from equation 1). The interaction term

measures the specific effects of competition on the individual genotype and therefore it is best presented as a function of the genotype (g_j) rather than the environment (a_i).

The regression coefficient β_j of equation 12 may be due to two causes, differing widely in their biological significance. The first cause is a *scale effect* produced by the well-known phenomenon of positive correlation between the intra-environment variation and the environment's mean. This factor cannot be considered a *true* genetic interaction in the sense of specific

FIG. 1.—Graphic presentation of a genotype as a function of the environment. (Linear components are those of equation 14. An arbitrary genotype $\bar{j}$ was partitioned at an arbitrary environment I . Wavy curve corresponds to the "true" functional relationship of the genotype $\bar{j}$ to the environment).

adaptation (sensitivity) to varying environments. The second cause is sensitivity or responsiveness to an underlying environmental variable which cannot be expressed as a scale function. The distinction between these two causes was described by Robertson (1959) and further elaborated with examples and implications to animal breeding by Dickerson (1962). In order to separate the effects of the above two causes the regression term $\beta_j a_i$ of equation 12 may be broken as follows:

$$\beta_j a_i = sg_j a_i + \beta'_j a_i.$$

When s is a *scale* coefficient and $\beta'_j = (\beta_j - sg_j)$ is a function of the environment after elimination of the *scale* effects. Substitution into equation 12 yields

$$g_{ij} = (1 + \beta'_j) a_i + g_j + sg_j a_i + \delta_{ij}. \quad (13)$$

Now the Bucio Alanis' expression of the interaction as a function of the environment (equation 12 and 13) and the present expression of the interaction as a function of the genotype (g_j) may be combined by partitioning the residual interaction term δ_{ij} into a function of the genotype plus an independent residual term ($\delta_{ij} = \alpha_i g_j + I'_{ij}$) and substituting into equation 13:

$$g_{ij} = (1 + \beta'_j) a_i + (1 + \gamma_i) g_j + sg_j a_i + I'_{ij}. \quad (14)$$

The last equation represents a complete partitioning of the genotype-environment interaction where each one of the four components ($\gamma_i g_j$, $\beta'_j a_i$, $sg_j a_i$ and I'_{ij}) has a distinct biological significance, each may be (at least partially) under different genetic control, and each has different implications to selection and breeding.

Freeman and Perkins (1971) showed that when the environment is measured independently of the data used for estimating the regression coefficient β_j (of equation 12 or β_j plus sg_j of equation 14) then β_j , the mean of all the β_j 's, is not necessarily equal to one. Consequently, equation 14 may assume an even more generalised form by substituting $(\beta + \beta'_j) a_i$ for $(1 + \beta'_j) a_i$. A graphical illustration of equation 14 is presented in fig. 1 when g_{ij} and its components were expressed as functions of the environment ($\mu + a_i$, when μ is the general mean). For the illustration a hypothetical specific genotype g_J was broken down at the arbitrarily chosen environment a_I . The various hypothetical regression lines were drawn and the accumulation of the components from μ to the full $g_{ij} + \mu$ are shown along the vertical axis. The wavy curve presents the actual ("true") functional relationship of the specific genotype g_j to $(\mu + a_i)$.

The independent measure of the environment (z) suggested by Freeman and Perkins (1971) could probably be applied to studies of competition if z is defined as a degree of crowding, proportion of other genotypes in a mixture, etc.

6. EXPERIMENTAL RESULTS

Relevant details of the ten experiments are given in table 2, while the results of separate ponds and mixed ponds are summarised, respectively, in tables 3 and 4. Full details of experiments 1 to 9 were given by Wohlfarth

TABLE 2
Details of the ten experiments

Experiment No.	Method of stocking the ponds	Number of ponds*	Pond's area in 1000 m ²	Number of fish per pond	Date of initiation of the test	Duration of the test (days)
1	Separate (E ₁) Mixed (E ₂)	10) 5)	1	175 or 250	2.ii.61	77
2	E ₁ E ₂	12) 6)	1	200	7.iii.61	76
3	E ₁ E ₂	12) 4)	1	252	6.vi.61	89
4	E ₁ E ₂	10) 5)	0.4	80	7.viii.61	97
5	E ₁ E ₂	20) 5)	0.4	90	5.viii.62	92
6†	E ₁ E ₂	20 6	1 5 to 30	250 100	9.ix.62 12.vii.62	70 101-140
7‡	E ₁ E ₂	8 4	0.2 15 to 25	37 30 to 120	18.vii.63 8.vii.63	86 98-139
8	E ₁ E ₂	10 4	0.4 15 to 25	100 55 to 120	9.vii.64 3.vii.64	76 90-141
9‡	E ₁ E ₂	18 5	0.2 15 to 25	40 175	28.vii.66 13.vii.66	110 109-135
10	E ₁ E ₂	17 4	0.4 20 to 30	85 50 to 200	28.vii.69 10.vii.69	119 115-144

* The number of separate ponds (E₁) stocked by each one of the two tested groups of each experiment was half the value recorded in this column. In experiment 10, one group was stocked into nine ponds and the second into eight ponds.

† In experiments 6 to 10 large ponds were used for the mixed stocking. These ponds were stocked with several groups of carp in addition to the pair dealt with here. The "number of fish" recorded in the table is an average per group of the two pertinent groups. When large variation existed, two values were recorded to cover the whole range of variation. The mixed ponds in these experiments were each stocked on the same day but emptied on different days. The two values for "duration of the tests" cover the whole range.

‡ In experiments 7 and 9, the unit of the "separate stocking" (E₁) was half a pond of 400 m² divided by wire netting.

TABLE 3
Summary of the results of separate ponds

Experiment No.	Initial weights (g)		Weight gains (g)*				
	Group A	Group B	Group A	Group B	Mean	$d_1 = (A-B)$	$d'_1 = T_1 d_1$
1	88	99	406	374	390	32	33
2	206	259	292	242	267	50	49
3	25	34	464	413	439	51	55
4	13	13	450	419	435	31	33
5	43	42	482	451	467	31	32
6	73	74	248	241	245	7	11
7	45	37	766	709	738	57	37
8	15	12	576	548	564	28	24
9	47	48	560	556	558	4	3
10	33	30	753	700	728	53	35

* d'_1 = difference in weight gain adjusted to an absolute weight of 500 g.

$T_1 = (500/W)$ when W = average absolute weight of all the separate ponds of a given experiment. Note that the results of separate ponds were not corrected for differences in initial weights (see text), hence the difference between this table and table 4.

TABLE 4
Summary of the results in mixed ponds

Experiment No.	Initial weights (g)		Weight gains (g)		Weight gains corrected for initial weights*			
	Group A	Group B	Group A	Group B	Group A	Group B	$(A-B) = d_2$	$d'_2 = T_2 d_2$
1	87	96	421	399	426	392	34	33
2	215	273	272	266	301	236	65	64
3	31	29	481	416	474	416	58	60
4	13	13	478	397	478	397	81	88
5	44	42	481	440	478	443	36	35
6	56	52	558	487	551	494	57	45
7	36	33	708	564	701	570	131	95
8	23	23	695	637	695	637	58	41
9	29	42	459	486	478	469	9	6
10	26	23	602	541	598	547	51	45

* d'_2 = difference in weight gains adjusted to an absolute weight of 500 g.
 $T_2 = (500/W_2)$, when W_2 = average absolute weight for a given mixed pond.

(1971). The difference in separate ponds between the two tested groups of each experiment was designated d_1 , and their difference in mixed ponds d_2 . Thus,

$$d_1 = (A_1 - B_1) \quad \text{and} \quad d_2 = (A_2 - B_2).$$

Table 5, A and B, show the differences in relative growth rate between the pairs of genetic groups in separate and mixed ponds for the ten experiments. The tables also provide estimates for the "error" variances due to

TABLE 5

Differences in weight gains in separate and mixed ponds, Standard Errors (SE) of these differences and the environmental (error) variances (s_E^2) of the ten experiments

(The indices 1 and 2 refer, respectively, to *separate* and *mixed* ponds. K = number of ponds. Values of d_1 , d_2 , d'_1 and d'_2 taken from tables 3 and 4.)

A. RESULTS NOT CORRECTED FOR ABSOLUTE WEIGHTS

Experiment No.	Difference in separate ponds (g) d_1	SE ₁ *	Difference in mixed ponds (g) d_2	SE ₂ *	K_1	$s_{E_1}^2$	K_2	$s_{E_2}^2$
1	32	11.9	34	7.4	5	714	5	277
2	50	7.7	65	9.0	6	353	6	487
3	51	9.2	58	9.9	6	507	4	396
4	31	16.6	81	9.2	5	1372	5	421
5	31	17.4	36	5.6	10	3032	5	154
6	7	5.0	57	15.3	10	252	6	1411
7	57	19.0	131	14.0	4	1437	4	788
8	28	14.0	58	7.2	5	982	4	210
9	4	8.8	9	8.3	9	704	5	345
10	53	19.3	51	3.8	8	2989	4	57
Mean	34.4	4.4	57.9	3.1	—	1234	—	455

B. RESULTS CORRECTED FOR ABSOLUTE WEIGHTS

Experiment No.	d'_1	SE ₁ *	d'_2	SE ₂ *	K_1	$s_{E_1}^2$	K_2	$s_{E_2}^2$
1	33	12.3	33	6.6	5	762	5	215
2	49	7.4	64	9.5	6	339	6	543
3	55	9.9	60	9.6	6	586	4	370
4	33	17.7	88	8.4	5	1566	5	358
5	32	17.1	35	5.6	10	2926	5	154
6	11	7.9	45	9.8	10	627	6	583
7	37	12.2	95	7.1	4	592	4	205
8	24	12.2	41	3.6	5	740	4	49
9	3	7.3	6	8.8	9	487	5	395
10	35	17.7	45	5.1	8	1296	4	104
Mean	31.2	3.9	51.2	2.5	—	992	—	297.6

* SE = Standard error of the mean = $(s_E^2/K)^{1/2}$. SE of the means of all the ten experiments = average $(s_E^2/K)/10)^{1/2}$.

environmental variation associated with the estimates of the genetic parameters. $s_{E_1}^2$ is the variance between the means of separate ponds stocked with the same genetic group, and $s_{E_2}^2$ is the variance between the deviations of the same group around the means of the mixed ponds. The group with the higher weight gain in *separate* ponds was designated A while the group with the lower weight gain was designated B. Thus, by definition, all the d_1 values

(tables 3 and 5) were positive. All ten d_2 values were also found to be positive (tables 4 and 5) showing that the rankings of the A and B groups were identical in the *separate* and *mixed* ponds of all the ten experiments.

It can be shown that the phenotypic correlation between weight gain in *separate* and *mixed* ponds is equal to the coefficient of correlation of d_1 and d_2 ($r_{d_1 d_2}$),

$$\hat{r}_{d_1 d_2} = \frac{\Sigma d_1 d_2}{\sqrt{(\Sigma d_1^2)(\Sigma d_2^2)}} \quad (15)$$

(see equations 13A, 14A and 15A in the appendix).

The estimates of this parameter obtained from the observed results and results corrected for absolute weights were very high, 0.92 and 0.91 respectively ($r_{d_1 d_2}$, table 6). This means that over 80 per cent (r^2 = the coefficient of determination) of the between-group variations in *mixed* ponds can be

TABLE 6

Estimates of genetic and phenotypic parameters of weight gain in mixed and separate ponds

Estimated parameter	Equations	Estimates*	
		1	2
$\sigma_{d_1}^2$	Variance between groups in <i>separate</i> ponds	13A	1495 1193
$\sigma_{d_2}^2$	Variance between groups in <i>mixed</i> ponds	14A	4299 3255
$\sigma_{d_1 d_2}$	Covariance of inter-group differences in <i>mixed</i> and <i>separate</i> ponds	15A and 20A	2312 1813
$r_{d_1 d_2}$	Correlation of group means in <i>mixed</i> and <i>separate</i> ponds	15	0.91 0.92
$\sigma_{g_1}^2$	Genetic variance between groups in <i>separate</i> ponds	18A	558 448
$\sigma_{g_2}^2$	Genetic variance between groups in <i>mixed</i> ponds	2 and 19A	2053 1568
$(\sigma_{g_2}^2 - \sigma_{g_1}^2)$	The difference between the genetic variance of <i>mixed</i> ponds and that of <i>separate</i> ponds	(18A-19A)	1495 1120
σ_{12}	Genetic covariance between groups in <i>mixed</i> and <i>separate</i> ponds	4 and 21A	1156 907
$\beta_g = 1 + \alpha$	Genetic regression of group means in <i>mixed</i> ponds on <i>separate</i> ponds	5 and 22A	2.07 2.02
ρ_g	Genetic correlation of group means of <i>mixed</i> and <i>separate</i> ponds	6	1.08 1.08
$\alpha(2 + \alpha)\sigma_{g_1}^2$	The variance component of genetic magnification	3	1833 1380
σ_I^2	The variance component of independent aggressiveness	9A	-338 -260

* The estimates in the columns headed by 1 and 2 were computed, respectively, from results not corrected for absolute weight and results corrected for an absolute weight of 500 g.

accounted for by variation in *separate* ponds. The residual variation in *mixed* ponds which is independent of variation in *separate* ponds is relatively small and could be due to experimental errors, or specific factors operating under one set of conditions but not the other (genotype-environment interactions). Thus, the very high value of the above correlation coefficient indicates that the second factor, composed of specific genotype-environment interactions is small or absent.

The estimate of the coefficient of regression of d_2 on d_1 was 1.5 ($\sigma_{d_1 d_2} / \sigma_{d_1}$, table 6). Its excess value over one indicated magnification of inter-group differences in mixed ponds, *i.e.* one unit difference in separate ponds increased to 1.5 units when the groups were co-stocked into the same mixed ponds.

The experimental results (tables 3, 4, and 5), analysed by the methods derived from the appendix, yielded estimates of the various phenotypic and genetic parameters. These estimates (table 6) constitute a summary of the results of the whole set of ten experiments. The following results are of particular interest:

(i) The inter-group genetic variance of growth rate in separate ponds ($\sigma_{g_1}^2$) under conditions prevailing in the tests was around 500 g^2 , while the inter-group genetic variance of growth rate of the same groups in mixed ponds ($\sigma_{g_2}^2$) was almost four times as high.

(ii) The estimate of the genetic correlation of growth rate in mixed and in separate ponds was one.

(iii) The estimate of the regression coefficient of growth rate in mixed ponds on growth rate in separate ponds was two, *i.e.* the value of the *amplification factor* α was one.

(iv) The estimate of the variance component of the *independent aggressiveness* (σ_I^2) was negative. It is assumed that the true value of this parameter is zero and that the negative value of the estimate was due to sampling and measurement "errors".

The biological interpretation of these results is that in each experiment the genetic group with the faster growth rate in separate ponds was more competitive in mixed ponds and therefore its relative growth rate was even higher in the presence of inter-group competition. This result fits our earlier findings that under competition larger carp grow faster through repression of smaller carp (Wohlfarth and Moav, 1972). They are also in agreement with Magnuson's (1962) observation that in the small Cyprinodont fish "Golden medaka" (*Oryzias latipes*) larger fish were dominant, grew faster and suppressed the growth of smaller fish when food supply was not abundant. Similar results were also reported by Greenberg (1947) in Sunfish and by Rose (1959, 1960) in guppies and in tadpoles.

The negative value of σ_I^2 indicates that competitiveness independent of growth capacity in separate ponds (yield capacity) was absent.

7. DISCUSSION

Three genetic parameters determine the genotype's performance (relative growth rate) under competition. These are, following equation 1, g_1 , α and I , or following equation 9, g_j , γ_i and I_{ij} . g is yield capacity, α is a population parameter determining the magnitude of the *amplification* by competition of inter-genotype differences and I is a measure of the only "true" interaction between the genotype and the environment; "true" in the sense that it is caused by specific adaptation. I is unpredictable, and is the only factor that reduces the value of the product-moment correlation coefficient below unity (Dickerson, 1962).

Many reports described experiments where different plant varieties or species were compared in pure stands and in mixtures. Some experiments were also conducted with insects, *e.g.* *Drosophila* (Ayala, 1969; Futuyma, 1970; Lewontin and Matsuo, 1963) and *Tribolium* (Park, 1954; Park *et al.*,

1964; Dawson, 1967). The many *pure-stand versus mixed planting* experiments in plants provided ample evidence for competition which is determined by *amplification* (αg_1) of differences in yield ability (g_1). But, at the same time, almost every experiment also provided evidence for *independent aggressiveness* (I of equation 1, or I' of equations 8, 9, 10 and 13) reducing, sometimes drastically, the correlation between growth rate under competition and performance in pure-stands (Christian and Gray, 1941; Suneson, 1949; Sakai, 1955, 1965; Roy, 1960; Allard, 1961; Harper, 1961, 1964; Jensen and Federer, 1965; Sammeta and Levins, 1970; Norrington-Davies, 1972; and others). The lack of correlation was sufficiently persistent and impressive to produce comments such as (Sakai, 1965): "That such characters as vigor, propagative capacity or fitness in pure-stand are not correlated with competitive ability is worthy of notice. Thus, the genotype, which is strong in competition but poor in fitness by itself could predominate in a population at the expense of another which is high in fitness but weak in competition." Translated into the present terminology this statement declares that independent aggressiveness (I) may be a more important contributor to the individual reproductive fitness than *efficiency* (g_1), therefore natural, or artificial selection, may increase I values at the expense of g_1 and this may result in reduced overall fitness of the population. The basic weakness of the above reasoning is that it overlooks the optimal solution of improving competitiveness by *amplification* (αg_1) rather than through increased independent aggressiveness (I).

Sakai (1965) suggested that in a natural population the simultaneous selection for *competitive ability* and for efficiency (which he calls fitness) should result in a genetic equilibrium, since the two characters are uncorrelated. This conclusion leads to the expectation that competitive ability should have a large genetic variance and a high heritability. However, this view is contrary to that of other workers such as Mather (1961), who claimed that a low heritability for competitive ability is to be expected for any species which has undergone selection and adjustment in nature or in cultivation.

In our opinion a durable simultaneous selection for competitiveness and efficiency of utilising external resources should result in an increased correlation rather than equilibrium between the two characters. That is, the two characters should be under a single genetic control and this could be achieved by increasing g_1 and α and eliminating I (equation 1).

The experiments described in this article provide empirical data on the correlation between growth rate of genetically diverse groups of carp in the presence and in the absence of inter-group competition. The results show a complete absence of competitiveness of the independent aggressiveness type ($\sigma_f^2 < 0$, table 6). This is compatible with the hypothesis that a single genetic control determines growth rate in the presence and absence of competition, and that competitiveness and its consequent effect on the variance of weight gain can be completely explained by variability in the weight of the fish (Wohlfarth and Moav, 1972). In other words, competitive ability independent of yield capacity, or of efficiency of exploiting external resources, is absent. However, competition does exist. It inflates the variance in mixed stocking through *amplification* of the genetic variability of yield capacity and it is expressed by the regression coefficient β_g (equation 5) having a value higher than one (table 6). This amplification could be caused by direct competition for food, "space", "growth factors" or "environmental

inputs", when the larger fish have an advantage over the smaller ones and they consume more than their "fair" share (de Wit, 1960; Harper, 1964). It could also be caused by social interaction (Allee *et al.*, 1948; Magnuson, 1962) or through excretion of growth inhibiting substances by the faster-growing individuals. Thus, Richards (1958) and Rose (1960) showed that in *tadpoles* accumulation of excreted substances caused growth inhibition, and that these water-borne inhibitors had a greater influence on smaller members of the population and resulted in a considerable increase in the variance of size (growth depensation).

Since the present results showed a complete correlation between competitive ability and yield capacity, they appear to be at variance with the results of many of the plant experiments mentioned above. This apparent difference could be due to real differences in the organisms under study, (cereals or legumes compared to carp), but it could also be due to a basic difference in the sampling of genotypes. Most of the competition experiments with plants were conducted with cultivated varieties developed in different locations and in response to varying requirements. This procedure is basically different from testing segregating genotypes of a single population that underwent natural selection and adjustment for many generations. In the latter case intra-population competitive ability is an integral major component of fitness and natural selection would be expected to increase its correlation with yield capacity (as described earlier). In the former case long-standing separate varieties, races or species are brought into competition by the experimenter. Inter-variety competition has then never been subjected to selection and its association with other fitness characters should be, to a large extent, coincidental and unpredictable. Hence, it is unjustified to extrapolate from inter-varietal differences to intra-population variability.

In the present experiment, assuming that the tested genotypes constituted a random sample of the population and that all the tested groups had the same intra-group genetic relationship, *i.e.* Full-sibs, then the inter-group genetic variance in this study should be half that of the population as a whole. Therefore, a rough estimate of the genetic variance in yield capacity, as well as, the coefficient of genetic determination of growth rate in a heterogeneous carp population, may be obtained from these results.

The genetic variance of growth rate in separate ponds for the entire population was $2\sigma_{g1}^2 = 1000 \text{ g}^2$ (table 6). Hence, one genetic standard-deviation in growth rate in separate ponds is 33 g, or 6-7 per cent of the mean final weight (500 g). Since weight gain in separate ponds is equivalent to yield capacity, a shift of one genetic SD should result in a 6-7 per cent change in yield.

Under the same assumptions the total genetic variance of carp of average weight of 500 g located in a single pond, under the general conditions of the present study, is roughly equal to twice the inter-group genetic variance in mixed ponds ($2\sigma_{g2}^2$), *i.e.* 3000 to 4000 g^2 . Since the total phenotypic variance of carp of this average weight and under the environmental conditions prevailing in the present experiments, is approximately 10,000 g^2 , it appears that the total genetic determination of the phenotypic variance is of the order of 30 to 40 per cent. It may be noted that most of the genetic variance in growth rate of carp was shown to be of the non-additive kind (Moav and Wohlfarth, 1966, 1973).

The above results and conclusions have direct applications to fish breeding. They imply that selection of individuals or groups for improving yield capacity may be carried out in mixed ponds. This is a very convenient feature because considerably higher selection intensities may be obtained when the units under selection are located within a single pond. For individual (mass) selection many thousands of fish can be grown in a single pond and therefore selection intensities can be very high. The alternative of growing each individual in a separate container not only requires a prohibitively large number of containers, it also compounds the container and genetic effects and introduces possible complications of genotype-environment interactions. Similarly group selection may be carried out in mixed ponds. We test each year 15-30 groups of carp for differences in growth rate in no more than five replicated mixed ponds (Wohlfarth and Moav, 1966). An equally powerful test in separate ponds would require hundreds of ponds. In mixed ponds the absence of compounding of genetic differences with pond differences plus the magnification of the inter-group differences due to competition makes the detection of relatively small differences between the groups much easier than in separate ponds. Another advantage of mixed ponds is that they do not have to be located in the same place or treated similarly.

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9. APPENDIX

Several methods for analysis of competition experiments have been suggested by various authors. For recent detailed procedures see, for

example, McGilchrist and Trenbath (1971) and Norrington-Davies (1972). The specific complications that these methods had to overcome were: correlations between observations within groups, scale effects, unequal structure and magnitude of "Error" variances, larger number of parameters than independent estimates and biologically meaningful partitioning of interaction terms (as described earlier in this report). None of the reported procedures was applicable to the present results. This was due to a combination of several factors, primary among them were an unsatisfactory partitioning of the genotype-environment interaction and differences in experimental lay-outs.

Following are details of two suggested procedures of analysis based on the models of equations 2 and 9. The first procedure is applicable to experiments testing three or more different groups (genotypes) simultaneously. It combines an Analysis of Variance (ANOVA) for *separate* (pure-stand) ponds, an ANOVA for *mixed* ponds and estimation of the covariation of the group means of the two stocking methods. The second procedure, which was employed for analysing the present experiments, is applicable to a series of separate tests each involving two groups only. The two procedures overcome the bothersome complication of different "Error" variances of *separate* and *mixed* stockings and provide legitimate estimates for the variances of the two competition factors, the *amplification* (αg_1) and independent aggressiveness (I) of equation 1.

1. ESTIMATION OF THE GENETIC PARAMETERS WHEN MORE THAN TWO GROUPS ARE TESTED IN A SINGLE EXPERIMENT

When J groups (genotypes) are tested, each in K_1 separate ponds (N_1 fish per pond), and together in K_2 *mixed ponds* (N_2 fish per group per pond), then weight gain of an individual fish (Y') is commonly presented as the following linear function:

$$Y'_{ijkn} = \mu + a_i + g_j + (ga)_{ij} + c_{ijk} + e_{ijkn}. \quad (1A)$$

When μ = grand mean over the two stocking methods (environments),

a_i = the "environment" effect, or the deviation of the mean of the i th stocking method from μ ,

g_j = the *average group* effect, or deviation of the j th group from its mean over the two environments,

$(ga)_{ij}$ = the *genotype-environment interaction*,

c_{ijk} = the "common-environment" effect of the k th sample (in separate ponds it includes the "pond effect") and,

e_{ijkn} = the "individual fish" effect.

Note that equation 1A is equal to equation 7 of the text with the addition of the general mean μ and the two "Error" terms c and e .

In the present study $(\mu + a_i)$ is of no interest, also the unit of measurement is not an individual fish but the mean of all the fish of the k th sample. Therefore, equation 1A was simplified into

$$Y_{ijk} = (Y'_{ijk} - (\mu + a_i)) = g_j + (ga)_{ij} + E_{ijk}. \quad (2A)$$

When dot in the index designates a mean value, and by definition:

$$E_{ijk} = c_{ijk} + e_{ijk}.$$

Analysis of variance (ANOVA) of weight gains in separate ponds (Environment 1)

Here $i = 1$, and equation 2A can be expressed as,

$$Y_{1jk} = g_{1j} + E_{1jk}. \quad (3A)$$

This describes a simple case of one-way ANOVA (randomised plot design) where an individual observation is the mean of a separate pond. Table 1A shows the two relevant Mean-Squares (MS) of the ANOVA and their

TABLE 1A

Analysis of variance (ANOVA) tables for analysis of differences between groups (genotypes) in separate and mixed stocking
(Y in the tables is equal to y' of equation 1A)

Source	d.f.	MS	E(MS)
<i>Separate ponds (i = 1)</i>			
Between groups (MS_{g_1})	$\bar{J}-1$	$\frac{1}{(\bar{J}-1)} \frac{1}{K_1} \sum_j (\sum_k Y_{1jk})^2 - \frac{(\sum_j \sum_k Y_{1jk})^2}{\bar{J}K_1}$	$K_1((1-\gamma)^2\sigma_g^2 + \sigma_{E_1}^2) + \sigma_{E_1}^2$ $= K_1\sigma_{g_1}^2 + \sigma_{E_1}^2$
Between ponds (MS_{E_1})	$\bar{J}(K_1-1)$	$\frac{1}{\bar{J}(K_1-1)} \sum_j \sum_k Y_{1jk}^2 - \frac{1}{K_1} \sum_j (\sum_k Y_{1jk})^2$	$\sigma_{E_1}^2$
<i>Mixed ponds (i = 2)</i>			
Between groups (MS_{g_2})	$\bar{J}-1$	$\frac{1}{(\bar{J}-1)} \frac{1}{K_2} \sum_j (\sum_k Y_{2jk})^2 - \frac{(\sum_j \sum_k Y_{2jk})^2}{\bar{J}K_2}$	$K_2((1+\gamma)^2\sigma_g^2 + \sigma_{E_2}^2) + \sigma_{E_2}^2$ $= K_2\sigma_{g_2}^2 + \sigma_{E_2}^2$
Pond-group interaction (MS_{E_2})	$(\bar{J}-1)(K_2-1)$	$\frac{1}{(\bar{J}-1)(K_2-1)} \sum_j \sum_k Y_{2jk}^2 - \frac{1}{K_2} \sum_j (\sum_k Y_{2jk})^2$ $- \frac{1}{\bar{J}} \sum_k (\sum_j Y_{2jk})^2 + \frac{(\sum_j \sum_k Y_{2jk})^2}{\bar{J}K_2}$	$\sigma_{E_2}^2$

expectations (E(MS)). Through these values an estimate of the inter-group (genetic) variance $\sigma_{g_1}^2$ can be computed,

$$\hat{\sigma}_{g_1}^2 = \frac{MS_{g_1} - MS_{E_1}}{K_1}. \quad (4A)$$

Note that the symbol $\hat{}$ signifies an estimate, and that $MS_{E_1} = \hat{\sigma}_{E_1}^2$.

ANOVA in mixed ponds (Environment 2)

Here $i = 2$,

$$Y_{2jk} = g_{2j} + c_{2k} + E_{2jk} \quad (5A)$$

and we have a two-way ANOVA (randomised blocks design). Note that the pond effect (c_{2k}) can be separated from E , hence the "error" components E_1 (Environment 1) and E_2 (Environment 2) are composed of different elements. From table 1A we may derive an estimate for $\sigma_{g_2}^2$,

$$\hat{\sigma}_{g_2}^2 = \frac{MS_{g_2} - MS_{E_2}}{K_2}. \quad (6A)$$

Also, $\hat{\sigma}_{E_2}^2 = MS_{E_2}$.

Analysis of the covariation between the two stocking methods (environments)

The mean performances of the j th group in Environments 1 and 2 are, respectively (following equations 3A and 5A, and equation 1 of the text),

$$Y_{1j} = g_{1j} + E_{1j}. \quad (7A)$$

and

$$Y_{2j} = (1 + \alpha)g_{1j} + I_j + E_{2j}. \quad (8A)$$

Note that Y_{2j} are defined as deviations around the means of individual mixed ponds, hence the absence of c_{2k} (of equation 5A) from the last equation. The covariance between the paired observations (Y_{1j} , Y_{2j}) is primarily of genetic origin and its expected value was presented in equation 4 of the text as a function of α and $\sigma_{g_1}^2$.

Similarly, the genetic regression (β_g) of g_2 on g_1 and their genetic correlation (ρ_g) were defined in equations 5 and 6. Substitution of estimates for σ_{12} , $\sigma_{g_1}^2$ and $\sigma_{g_2}^2$ (table 1A and equations 4A and 6A) in equations 5 and 6 provide estimates for β_g and ρ_g . Using the relationship $\beta_g = (1 + \alpha)$, and rearranging equation 2 (text) results in,

$$\sigma_I^2 = \sigma_{g_2}^2 - \beta_g^2 \sigma_{g_1}^2. \quad (9A)$$

Substitution of estimates for the factors on the right side of this equation provides an estimate for σ_I^2 .

2 ESTIMATION OF THE GENETIC PARAMETERS WHEN ONLY A SINGLE PAIR OF GROUPS (GENOTYPES) IS TESTED IN EACH ONE OF A SERIES OF SEVERAL EXPERIMENTS

Frequently, shortage of facilities does not permit the testing of more than one pair of groups in a single experiment, but several (M) experiments may be performed consecutively. This was the case in the present study. Under these circumstances, $I = 2$ (environments, stocking methods) and also $J = 2$ (tested groups in a single experiment).

The differences in the m th experiment, between the average performances of the pair of tested groups in separate ponds (d_{m1}) and mixed ponds (d_{m2}) are, respectively (following equation 7A and 8A),

$$d_{m1} = (Y_{m11} - Y_{m12}) = (g_{m11} - g_{m12}) + (E_{m11} - E_{m12}) \quad (10A)$$

and

$$d_{m2} = (Y_{m21} - Y_{m22}) = \beta_g(g_{m11} - g_{m12}) + (I_{m1} - I_{m2}) + (E_{m21} - E_{m22}). \quad (11A)$$

In general, the variable d was defined as $d = (Y_1 - Y_2)$ when, post-hoc, the index 1 was assigned to the larger of the Y 's ($Y_1 > Y_2$). When Y_1 and Y_2 are chosen independently and at random from a population of Y 's, then the variable d has an expectation of zero and its variance is,

$$\sigma_d^2 = E(d^2) = 2\sigma_y^2. \quad (12A)$$

The M experiments provide a series of M pairs (d_{m1} , d_{m2}) whose variances and covariances can be estimated directly from the experimental results.

$$\hat{\sigma}_{d_1}^2 = s_{d_1}^2 = \frac{1}{M} \sum_{m=1}^M d_{m1}^2 \quad (13A)$$

$$\hat{\sigma}_{d_2}^2 = s_{d_2}^2 = \frac{1}{M} \sum^M d_{m2}^2 \quad (14A)$$

and

$$\hat{\sigma}_{d_1 d_2} = s_{d_1 d_2} = \frac{1}{M} \sum^M d_1 d_2. \quad (15A)$$

Note that because of the assignment of $Y_1 > Y_2$ the above estimates can not be of the general form

$$s_{d_1 d_2} = (\Sigma(d_1 - d_1.)(d_2 - d_2.))/(m-1).$$

The mean variance between separate ponds of the same group (over all the experiments) may serve as an estimate for $\sigma_{E_1}^2$.

$$\hat{\sigma}_{E_1}^2 = s_{E_1}^2 = \frac{\sum^M \sum^J \left(\sum^{K_1} (Y_{1mjk} - Y_{1mj.})^2 \right)}{\sum^M \sum^J (K_{1jm} - 1)}. \quad (16A)$$

On the other hand, the estimate of $\sigma_{E_2}^2$ should be based on the variance of deviations of each group from the means of the individual mixed ponds,

$$\hat{\sigma}_{E_2}^2 = s_{E_1}^2 = \frac{\sum^M \sum^J \left(\sum^{K_2} (Y_{2mjk} - Y_{2m.k})^2 \right)}{\sum^M \sum^J (K_{2mj} - 1)}. \quad (17A)$$

Following equation 10A $\sigma_{d_1}^2$ can be expressed as,

$$\sigma_{d_1}^2 = 2(\sigma_{g_1}^2 + \sigma_{E_1}^2).$$

Rearrangement and substitution of estimates for $\sigma_{d_1}^2$ (equation 13A) and for $\sigma_{E_1}^2$ (equation 16A) provides an estimate for $\sigma_{g_1}^2$,

$$\hat{\sigma}_{g_1}^2 = \frac{s_{d_1}^2}{2} - \hat{\sigma}_{E_1}^2. \quad (18A)$$

Similarly,

$$\sigma_{d_2}^2 = 2(\beta_g^2 \sigma_{g_1}^2 + \sigma_I^2 + \sigma_{E_2}^2)$$

and

$$\hat{\sigma}_{g_2}^2 = \frac{s_{d_2}^2}{2} - \hat{\sigma}_{E_2}^2. \quad (19A)$$

The covariance $\sigma_{d_1 d_2}$ equals twice the genetic covariance σ_{12} ,

$$\sigma_{d_1 d_2} = 2\beta_g \sigma_{g_1}^2 = 2\sigma_{12}. \quad (20A)$$

Hence,

$$\hat{\sigma}_{12} = \frac{s_{d_1 d_2}}{2}. \quad (21A)$$

Substitution of estimates for $\sigma_{g_1}^2$ (equation 18A) and for σ_{12} (the last equation) and rearrangement of equation 20A provides an estimate for β_g ,

$$\hat{\beta}_g = \frac{\hat{\sigma}_{12}}{\hat{\sigma}_{g_1}^2} = \frac{s_{d_1 d_2}}{(s_{d_1}^2 - 2s_{E_1}^2)}. \quad (22A)$$

Finally, substitution of the estimates of equations 18A, 19A and 22A on the right side of equation 9A provides an estimate for σ_I^2 .