SEX-LINKAGE AND SEX-LIMITATION IN QUANTITATIVE INHERITANCE

II. DIALLEL CROSSES

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Received 4.iii.72

SUMMARY

For animal breeders the diallel cross has one advantage over all other mating designs in that it permits sex-linkage to be distinguished from other causes of reciprocal differences. In other designs it is necessary to assume that other causes of reciprocal differences are absent (Killick, 1971). Since the transmission of X chromosomes to female progeny follows the same pattern as that of autosomes, sex-linkage can cause no disturbance to the data on female progeny; equally sex-linked inheritance cannot be distinguished from autosomal inheritance when data from female progeny alone are available. When data from male progeny are analysed alone, the factorial and Hayman analyses of variance are very informative. The $(W_r + V_r)$ and $(W_r - V_r)$ analyses of variance are undisturbed by sex-linkage, but if reciprocal crosses are averaged the regression line will not pass through the origin of the graph even with complete dominance. Provided reciprocal crosses are not averaged, all possible parameters may be estimated from the data. When data from male and female progeny are pooled the components of variance are more complex and less informative. The factorial analysis of variance is of little real help but the Hayman analysis of variance remains useful. The W_r/V_r analyses retain their use but unless reciprocal crosses are averaged the $(W_r - V_r)$ analysis will be misleading. Not all the possible parameters can be estimated from the data. Thus the best approach will probably be to fit a simple model and then adjust and add to this until a satisfactory fit is obtained. Provided no more than 13 parameters are required, at least one degree of freedom will be available to test the adequacy of the model.

1. INTRODUCTION

THE diallel cross has been relatively rarely used by animal breeders and geneticists although Jinks (1954) and Dickinson and Jinks (1956) considered it to be a particularly economical method of investigating the inheritance of quantitative traits in animals because the first filial generation alone is required. The familiar analyses of the diallel table assume inheritance to be autosomal; one drawback to the animal geneticist is therefore his uncertainty over the distortion that may result from the segregation of sex-linked genes. Nevertheless, various diallel analyses do allow the recognition of reciprocal differences as in Hayman's (1954) analysis of variance or the factorial analysis of Jinks and Broadhurst (1963). Durrant (1965) considered two types of mechanism causing reciprocal differences in diallel tables. These he called α inheritance, where the contribution of a gene in both homozygotes and heterozygotes differs in male and female arrays and β inheritance where the dominance contribution of a gene differs in the two

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reciprocal crosses. Weardon (1964) gave the expected mean squares obtainable from a diallel table assuming either maternal or reciprocal effects (as might arise from sex-linkage). In the latter model he did not distinguish between the sex of progeny, which makes for ambiguity. Topham (1966) provided an analysis of the diallel table to detect maternal effects and their interactions.

In this paper the effect of sex-linkage and sex-limitation on the analysis and interpretation of data from diallel crosses will be considered. In the present context sex-limitation implies a differential effect of the same autosomal gene in males and females. This may range from a slight quantitative difference to cases where the character is expressed in one sex only (*e.g.* milk yield in cattle).

2. MODEL AND PARAMETERS

The diallel cross is constructed by crossing n inbred lines in all possible combinations as both male and female parents to give n^2 families. These consist of $\frac{n^2-n}{2}$ F_1 families, their $\frac{n^2-n}{2}$ reciprocal families and n "selfed" families resulting from matings within the inbred lines. Where inbred lines are not available herds or breeds may be used; the bias caused by the heterozygosity will not be too serious provided the genetic differences within such parental groups are much less than those between them (Jinks, 1954).

The model and parameters describing sex-linkage used in this paper are those of Mather and Jinks (1971). A locus on the X chromosome having two alleles, A and a is considered to have the following effects:

Females			Males	
AA	Aa	aa	A	a
d_x	h_x	$-d_x$	d'_x	$-d'_x$

Five parameters are required to specify the variation arising from sex-linked genes in randomly mating populations. These are:

$$\begin{aligned}
 D_{RX} &= \Sigma 4uv[d_x + (v-u)h_x]^2 \\
 H_{RX} &= \Sigma 16u^2v^2h_x^2 \\
 D'_X &= \Sigma 4uvd_x'^2 \\
 D'_{XX} &= \Sigma 4uvd_xd'_x \\
 F'_{XX} &= \Sigma 8uv(u-v)d'_xh_x
 \end{aligned}$$

With a parental population of inbred lines it is necessary to subdivide the D_{RX} parameter further, so we define:

$$\begin{aligned}
 D_X &= \Sigma 4uvd_x^2 \\
 H_{1X} &= \Sigma 4uvh_x^2 \\
 H_{2X} &= \Sigma 16u^2v^2h_x^2 \quad (= H_{RX}) \\
 F_X &= \Sigma 8uv(u-v)d_xh_x
 \end{aligned}$$

For some analyses it is necessary to redefine the F parameters to the form $\Sigma 4uvdh$. This is indicated in the appropriate tables.

The model describing sex-limitation is that given by Killick (1971). The familiar three parameters which specify the mid-parent and the additive

Method (a)

The transmission of X chromosomes to female progeny follows exactly the same pattern as the transmission of autosomes. Consequently the effects of the sex chromosomes cannot be separated from those of the autosomes, but equally sex-linkage cannot distort the analysis of female data. The additive and dominance effects of the combined autosomes and sex chromosomes can be separated since

$$\sigma_f^2 = \sigma_m^2 = \frac{1}{4}(D_{RX} + D_R)$$

and

$$\sigma_{mf}^2 = \frac{1}{4}(H_{RX} + H_R)$$

where the subscripts f , m and mf indicate the component of variance for female and male parent arrays and their interaction respectively. With unequal allele frequencies the additive parameters contain some dominance variation. If $\sigma_f^2 \neq \sigma_m^2$ the presence of some mechanism, other than sex-linkage, causing reciprocal differences may be detected. No other design permits this distinction to be made; in all the designs used for the analysis of randomly mating base populations, it is necessary to assume that sex-linkage alone is responsible for reciprocal differences (Killick, 1971).

Sex-linkage in male progeny causes σ_f^2 to exceed σ_m^2 . It is possible, therefore, to differentiate between the additive effect of the hemizygous X chromosome, the additive effect of the autosomes and the dominance effects of the autosomes by estimating the parameters representing each of these effects.

Method (b)

The expectation of the component of variance for sexes, σ_s^2 , is not given in table 1 as it is a fixed effect, not a true variance, having a complex interpretation not definable in terms of the parameters used in this paper. σ_s^2 will therefore be ignored in this and in subsequent analyses. This leaves six statistics from which nine parameters are to be estimated, so clearly full separation of the various effects is impossible. However, various combinations of the components do permit estimation of a few parameters. For example:

$$\begin{aligned}(\sigma_f^2 - \sigma_m^2) - (\sigma_{sf}^2 - \sigma_{sm}^2) &= \frac{1}{2}D'_{XX} - \frac{1}{4}F'_{XX} \\ (\sigma_f^2 - \sigma_m^2) + (\sigma_{sf}^2 - \sigma_{sm}^2) &= \frac{1}{2}D'_X\end{aligned}$$

but the standard errors of such estimates are bound to be large. Similarly $(\sigma_{mf}^2 - \sigma_{smf}^2) = \frac{1}{4}(H_R - H_{RS})$, an estimate of the degree of autosomal dominance, less the sex-limited dominance. Nevertheless, the main use of the factorial analysis of variance is the establishment of the significance of genetic effects rather than their estimation.

(ii) *The analysis of variance of Hayman (1954)*

Four main sources of variation are recognised in Hayman's analysis of variance. These, with their autosomal interpretations, are:

- "a" item   additive genetical variation
- "b" item   non-additive genetical variation subdivided into three parts

"b ₁ " item	overall potence
"b ₂ " item	potence due to particular parents
"b ₃ " item	residual non-additive variation
"c" item	reciprocal differences attributable to particular parents
"d" item	reciprocal differences not attributable to particular parents.

No distinction is made as to the origin of the reciprocal differences measured by the "c" and "d" items. Such a distinction can only be deduced from inspection of the data and not from the analysis of variance. The Hayman analysis was not intended for the estimation of parameters and is not suitable for this purpose. Its advantage lies in detecting significant genetical effects.

Method (a)

The expectations for the mean squares in the Hayman analysis are shown in table 2. When female progeny are analysed alone, each item

TABLE 2
Interpretation of the Hayman analysis of variance. Each mean square
is a function of the following parameters
Method (a), i.e. sexes separate

	Sex-linkage parameters				Autosomal parameters		
	Females		Males		Either sex		
	D_{RX}	H_{1X}	H_{2X}	D'_X	D_R	H_1	H_2
a	$\frac{1}{2}$	—	—	$\frac{1}{2}$	$\frac{1}{2}$	—	—
b	—	—	$\frac{1}{4}$	—	—	—	$\frac{1}{4}$
b ₁	$[(wh_x)^2]$			—	$[(wh)^2]$		
b ₂	—	1	—1	—	—	1	—1
c	—	—	—	$\frac{1}{2}$	—	—	—
d	—	—	—	—	—	—	—

	Sex-linkage parameters						Autosomal parameters					
	D_{RX}	H_{1X}	H_{2X}	D'_X	D'_{XX}	F'_{XX}	D_R	H_1	H_2	D_{RS}	H_{1S}	H_{2S}
	$\frac{1}{8}$	—	—	$\frac{1}{8}$	$\frac{1}{4}$	$-\frac{1}{8}$	$\frac{1}{8}$	—	—	—	—	—
a	$\frac{1}{8}$	—	—	$\frac{1}{8}$	$\frac{1}{4}$	$-\frac{1}{8}$	$\frac{1}{8}$	—	—	—	—	—
b	—	—	$\frac{1}{16}$	—	—	—	—	—	$\frac{1}{16}$	—	—	—
b ₁	$[(\frac{1}{2}wh_x)^2]$						$[(\frac{1}{2}wh)^2]$					
b ₂	—	$\frac{1}{4}$	$-\frac{1}{4}$	—	—	—	—	$\frac{1}{4}$	$-\frac{1}{4}$	—	—	—
c	—	—	—	$\frac{1}{8}$	—	—	—	—	—	—	—	—
d	—	—	—	—	—	—	—	—	—	—	—	—
S × a	$\frac{1}{8}$	—	—	$\frac{1}{8}$	$-\frac{1}{4}$	$\frac{1}{8}$	—	—	—	$\frac{1}{8}$	—	—
S × b	—	—	$\frac{1}{16}$	—	—	—	—	—	—	—	—	$\frac{1}{16}$
S × b ₁	$[(\frac{1}{2}wh_x)^2]$						$[(\frac{1}{2}wh)^2]$					
S × b ₂	—	$\frac{1}{4}$	$-\frac{1}{4}$	—	—	—	—	—	—	—	$\frac{1}{4}$	$-\frac{1}{4}$
S × c	—	—	—	$\frac{1}{8}$	—	—	—	—	—	—	—	—
S × d	—	—	—	—	—	—	—	—	—	—	—	—

continues to detect the effects as described above, whether the source is autosomal or sex-linked. Consequently, sex-linkage has no disturbing influence on the analysis. When male progeny are analysed alone, the additive effect of the sex chromosome makes an equal contribution to the "a" and the "c" items. The "b" item is unaffected since the hemizygous

X chromosome cannot exhibit dominance. The interpretation of the analysis remains valid since the "a" item detects additive gene action and the "c" item detects reciprocal differences. As with the factorial analysis of variance, this provides an unambiguous test for sex-linkage since if the "c" item is significant for male progeny but not for female progeny, then sex-linkage alone must be responsible. The difference between the "a" and "c" mean squares for male progeny indicates the relative proportions of additive gene effects on the autosomes and sex-chromosome.

Method (b)

With the exception of the "a" item all the main effects and their sex interactions in the analysis have the same expectation for sex-linked genes; differences between the main effects and their sex-interactions can be ascribed to autosomal variation (see table 2). It will be seen that each item continues to detect the type of gene action conventionally associated with it but the main effects are now compounded of sex-linked and autosomal effects while the sex interactions are compounded of sex-linked and sex-limited effects.

(iii) *Variances and covariances of arrays (Jinks, 1954)*

Jinks (1954) showed that in the absence of epistasis the difference between the covariance of array family means on their non-recurrent parents, W_r , and the variance of the array family means, V_r , should be constant for all arrays. The regression of W_r on V_r should be a line of unit slope with the most dominant parents giving rise to array points nearest the origin of the graph and the most recessive parents giving rise to array points furthest from the origin. If the line passes through the origin, dominance is complete; otherwise the distance from the origin to the point where the line intersects the W_r axis is equal to $\frac{1}{4}(D-H_1)$. If the point of intersection is on the positive side of the origin, dominance is incomplete, and if it is on the negative side, overdominance is to be suspected. If there is no dominance, all the array points centre at a single value of

$$V_r = \frac{1}{2}W_r = \frac{1}{4}V_p.$$

The significance of the dominance variation is shown by variation in values of $(W_r + V_r)$ for each array (Allard, 1956). The absence of epistasis and of correlated gene distributions, and the presence of dominance, are conveniently tested by an analysis of variance of $(W_r - V_r)$ and $(W_r + V_r)$ values using their block interactions as error (Mather and Jinks, 1971). It is usual to average reciprocal crosses before computing values of W_r and V_r , but here the expectations will be given for the data both before and after averaging over reciprocal crosses.

Method (a)

The expectations of the variances and covariances when sexes are analysed separately are shown in table 3. The expectations of the sex-linkage parameters in female progeny are identical to those of the autosomal parameters so, as might be expected, sex-linkage cannot distort any W_r , V_r analysis from female data; the interpretation covers both sex-linked and autosomal genes. The situation is a little different in male progeny. When the data are averaged over reciprocals, sex-linked genes contribute $\frac{1}{4}D'_X$

to the value of $(W_r - V_r)$ and $\frac{3}{4}D'_X$ to the value of $(W_r + V_r)$ for every array. Sex-linked genes therefore contribute nothing to the variance of arrays in either the $(W_r + V_r)$ or the $(W_r - V_r)$ analysis of variance and so do not bias the results. When the data are not averaged over reciprocal crosses the only contribution sex-linked genes make is $2D'_X$ to $(W_{r\delta} + V_{r\delta})$ for every array, so here again, provided male and female parent arrays are considered separately, sex-linkage causes no bias to the results. D'_X is estimated simply as half the average difference between

$$(W_{r\delta} + V_{r\delta}) - (W_{r\varphi} + V_{r\varphi})$$

for all arrays. The effect of sex-linkage may be separated from other causes of reciprocal differences in that it causes a difference between the statistics

TABLE 3
Variances and covariances of arrays
Method (a), i.e. sexes separate

	Sex-linkage parameters*				Autosomal parameters*		
	Females		Males		Either sex		
	D_X	H_{1X}	F_X	D'_X	D	H_1	F
Not averaged over reciprocal crosses:							
$V\varphi AA$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$
$V\varphi aa$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	—	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$
$V\delta A$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$	1	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$
$V\delta a$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	1	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$
$W\varphi AA$	$\frac{1}{2}$	—	$-\frac{1}{2}$	—	$\frac{1}{2}$	—	$-\frac{1}{2}$
$W\varphi aa$	$\frac{1}{2}$	—	$\frac{1}{2}$	—	$\frac{1}{2}$	—	$\frac{1}{2}$
$W\delta A$	$\frac{1}{2}$	—	$-\frac{1}{2}$	1	$\frac{1}{2}$	—	$-\frac{1}{2}$
$W\delta a$	$\frac{1}{2}$	—	$\frac{1}{2}$	1	$\frac{1}{2}$	—	$\frac{1}{2}$
Averaged over reciprocal crosses:							
$V AA$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$
$V aa$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$
$W AA$	$\frac{1}{2}$	—	$-\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	—	$-\frac{1}{2}$
$W aa$	$\frac{1}{2}$	—	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	—	$\frac{1}{2}$

$$* F_X = \Sigma 4wd_x h_x. \quad F = \Sigma 4wdh.$$

from male and female parent arrays in male progeny alone; other causes of reciprocal differences would create similar differences in female progeny also. Sex-linkage in the averaged male data will prevent the W_r , V_r regression line passing through the origin of the graph since the intercept will be $\frac{1}{4}(D - H_1 + D'_X)$ units from the origin and this will be positive even if all the autosomal genes are completely dominant. When reciprocal crosses are not averaged, and male and female parent arrays are considered separately, this effect on the intercept will not occur. In such cases

$$(W_{r\varphi} - V_{r\varphi}) = (W_{r\delta} - V_{r\delta}) = \frac{1}{4}(D - H_1)$$

for all arrays.

Method (b)

The expectations of array variances and covariances when male and female progeny are analysed jointly are shown in table 4. Considering

firstly the case where reciprocal families are averaged prior to computation of these statistics it can be seen that sex-linkage distorts neither the $(W_r - V_r)$ nor the $(W_r + V_r)$ analyses since

$(W_{AA} - V_{AA}) = (W_{aa} - V_{aa})$ and $(S \times W_{AA} - S \times V_{AA}) = (S \times W_{aa} - S \times V_{aa})$, where W_{AA} is the covariance of an array, the recurrent parent of which has

TABLE 4
Variances and covariances of arrays

Method (b), i.e. sexes pooled

	Sex-linkage parameters*						Autosomal parameters*					
	D_X	H_{1X}	F_X	D'_{XX}	D_{XX}	F'_{XX}	D	H_1	F	D_S	H_{1S}	F_S
Not averaged over reciprocal crosses:												
$V_{\bar{\sigma}} AA$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$	—	—	—
$V_{\bar{\sigma}} aa$	$\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	—	—	—
$V_{\bar{\sigma}} A$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$	—	—	—
$V_{\bar{\sigma}} a$	$\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	—	—	—
$W_{\bar{\sigma}} AA$	$\frac{1}{8}$	—	$-\frac{1}{8}$	—	$\frac{1}{8}$	$-\frac{1}{8}$	$\frac{1}{2}$	—	$-\frac{1}{2}$	—	—	—
$W_{\bar{\sigma}} aa$	$\frac{1}{8}$	—	$\frac{1}{8}$	—	$\frac{1}{8}$	$\frac{1}{8}$	$\frac{1}{2}$	—	$\frac{1}{2}$	—	—	—
$W_{\bar{\sigma}} A$	$\frac{1}{8}$	—	$-\frac{1}{8}$	$\frac{1}{4}$	$\frac{3}{8}$	$-\frac{1}{8}$	$\frac{1}{2}$	—	$-\frac{1}{2}$	—	—	—
$W_{\bar{\sigma}} a$	$\frac{1}{8}$	—	$\frac{1}{8}$	$\frac{1}{4}$	$\frac{3}{8}$	$\frac{1}{8}$	$\frac{1}{2}$	—	$\frac{1}{2}$	—	—	—
$SV_{\bar{\sigma}} AA$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	—	—	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$
$SV_{\bar{\sigma}} aa$	$\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	—	—	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$
$SV_{\bar{\sigma}} A$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	$\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{4}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$
$SV_{\bar{\sigma}} a$	$\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$
$SW_{\bar{\sigma}} AA$	$\frac{1}{8}$	—	$-\frac{1}{8}$	—	$\frac{1}{8}$	$-\frac{1}{8}$	—	—	—	$\frac{1}{2}$	—	$-\frac{1}{2}$
$SW_{\bar{\sigma}} aa$	$\frac{1}{8}$	—	$\frac{1}{8}$	—	$\frac{1}{8}$	$\frac{1}{8}$	—	—	—	$\frac{1}{2}$	—	$\frac{1}{2}$
$SW_{\bar{\sigma}} A$	$\frac{1}{8}$	—	$-\frac{1}{8}$	$\frac{1}{4}$	$-\frac{3}{8}$	$\frac{1}{8}$	—	—	—	$\frac{1}{2}$	—	$-\frac{1}{2}$
$SW_{\bar{\sigma}} a$	$\frac{1}{8}$	—	$\frac{1}{8}$	$\frac{1}{4}$	$-\frac{3}{8}$	$-\frac{1}{8}$	—	—	—	$\frac{1}{2}$	—	$\frac{1}{2}$
Averaged over reciprocal crosses:												
V_{AA}	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	$\frac{1}{16}$	$\frac{1}{8}$	$-\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$	—	—	—
V_{aa}	$\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	$\frac{1}{16}$	$\frac{1}{8}$	$\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	—	—	—
W_{AA}	$\frac{1}{8}$	—	$-\frac{1}{8}$	$\frac{1}{8}$	$\frac{1}{4}$	$-\frac{1}{8}$	$\frac{1}{2}$	—	$-\frac{1}{2}$	—	—	—
W_{aa}	$\frac{1}{8}$	—	$\frac{1}{8}$	$\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{8}$	$\frac{1}{2}$	—	$\frac{1}{2}$	—	—	—
SV_{AA}	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	$\frac{1}{16}$	$-\frac{1}{8}$	$\frac{1}{8}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{2}$
SV_{aa}	$\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	$\frac{1}{16}$	$-\frac{1}{8}$	$-\frac{1}{8}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$
SW_{AA}	$\frac{1}{8}$	—	$-\frac{1}{8}$	$\frac{1}{8}$	$-\frac{1}{4}$	$\frac{1}{8}$	—	—	—	$\frac{1}{2}$	—	$-\frac{1}{2}$
SW_{aa}	$\frac{1}{8}$	—	$\frac{1}{8}$	$\frac{1}{8}$	$-\frac{1}{4}$	$-\frac{1}{8}$	—	—	—	$\frac{1}{2}$	—	$\frac{1}{2}$

$$* F'_{XX} = \Sigma 4uvd'_x h_x. \quad F_X = \Sigma 4uvd_x h_x. \quad F = \Sigma 4uvdh. \quad F_S = \Sigma 4uvf_a f_h.$$

the increasing allele etc., *i.e.* the difference between the covariance and variance of every array is constant. Similarly

$$(W_{AA} + V_{AA}) - (W_{aa} + V_{aa}) = -\frac{1}{2}F_X - \frac{1}{2}F'_{XX} - F$$

so the dominance of both sex chromosomes and autosomes contributes to the variation between arrays. If the values of the corresponding sex interactions are considered, the effect of F_X opposes that of F'_{XX} and the analysis will be less effective since

$$(S \times W_{AA} + S \times V_{AA}) - (S \times W_{aa} + S \times V_{aa}) = -\frac{1}{2}F_X + \frac{1}{2}F'_{XX} - F_S.$$

Other combinations of statistics can be derived to test the significance of dominance. For example, the variation in values of $(W_r + S \times W_r)$ may be used since $(W_{AA} + S \times W_{AA}) - (W_{aa} + S \times W_{aa}) = \frac{1}{2}F_X - F - F_S.$

Even with complete autosomal dominance the W_r , V_r regression line will not pass through the origin of the graph but cut the W_r axis at a point $\frac{1}{16}D'_X + \frac{1}{8}D'_{XX}$ from it. The $S \times W_r$, $S \times V_r$ regression line will cut the $S \times W_r$ axis at $\frac{1}{16}D'_X - \frac{1}{8}D'_{XX}$.

In cases where reciprocal families are not averaged prior to computing the statistics a different situation obtains. The analysis of variance of $(W_r - V_r)$ using either male or female parent arrays, and either the main effects or sex interactions can not be interpreted in the normal way. This is because F'_{XX} contributes to the variation between arrays which cannot therefore be ascribed reliably to epistasis or correlated gene distributions. The $(W_r + V_r)$ analysis is quite satisfactory when performed on either male parent or female parent arrays, and detects both sex-linked and autosomal dominance. The contribution of F'_{XX} to the variation between male parent arrays is three times that in female parent arrays. The former are therefore more efficient in detecting sex-linked dominance.

(iv) *Estimation of parameters*

Whilst some parameters may be estimated from the factorial analysis of variance the method of estimation described by Jinks (1954) is more efficient. Four statistics are used; mean variance within arrays, $\bar{V}_r$; variance of array means, V_r ; variance of parents, V_p ; and the covariance of parents and offspring, $\bar{W}_r$. Jinks states that the last statistic may be calculated in either of two ways; as the mean of the covariance of array members on their non-recurrent parents or as the covariance of array means on the recurrent parent of each array. However, this identity applies only to symmetrical tables as when reciprocal crosses are averaged prior to computation and does not apply when reciprocal differences exist. In the latter case the mean covariance of members of female parent arrays on their non-recurrent (*i.e.* male) parents is algebraically equivalent to the covariance of male parent array means on the recurrent parent of those arrays. To avoid any confusion reference will only be made to the covariance of array means on the recurrent parent of the arrays designated $W_{r\bar{2}}$ for female parent array means and $W_{r\bar{3}}$ for male parent array means.

Method (a)

The coefficients of the sex-linkage parameters for female progeny are identical to the corresponding autosomal parameters whether the data are averaged over reciprocal crosses or not (see table 5). Consequently no separation of sex-linked and autosomal parameters is possible, but their joint effects can be estimated. Because the expectations of male parent arrays are the same as those of female parent arrays the data must be averaged over reciprocal crosses to facilitate estimation of these parameters. For male progeny D'_X is completely correlated with D in the averaged data, so these two parameters cannot be separated. When the data are not averaged over reciprocals all five parameters can be estimated.

Method (b)

Table 6 shows the expectations of statistics when the sexes of progeny are combined. When the data are averaged over reciprocal crosses before computation of the statistics, only eight statistics can be derived although

TABLE 5
Expectation of statistics from a diallel table

Method (a), i.e. sexes separate

	Sex-linkage parameters					Autosomal parameters			
	Females				Males	Either sex			
	D_X	H_{1X}	H_{2X}	F_X	D'_X	D	H_1	H_2	F
Averaged over reciprocal crosses:									
V_p	1	—	—	—	1	1	—	—	—
$\bar{V}_r$	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$
$V_{\bar{r}}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$
W_r	$\frac{1}{2}$	—	—	$-\frac{1}{4}$	$\frac{1}{2}$	$\frac{1}{2}$	—	—	$-\frac{1}{4}$
Not averaged over reciprocal crosses:									
V_p	1	—	—	—	1	1	—	—	—
$\bar{V}_{r\varnothing}$	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$	—	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$
$\bar{V}_{r\delta}$	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$	1	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$
$V_{\bar{r}\varnothing}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$	1	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$
$V_{\bar{r}\delta}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$
$W_{r\varnothing}$	$\frac{1}{2}$	—	—	$-\frac{1}{4}$	1	$\frac{1}{2}$	$\frac{1}{4}$	—	$-\frac{1}{4}$
$W_{r\delta}$	$\frac{1}{2}$	—	—	$-\frac{1}{4}$	—	$\frac{1}{2}$	—	—	$-\frac{1}{4}$

TABLE 6
Expectations of statistics from a diallel table

Method (b), sexes combined

	Sex-linkage parameters							Autosomal parameters							
	D_X	H_{1X}	H_{2X}	F_X	D'_X	D'_{XX}	F'_{XX}	D	H_1	H_2	F	D_S	H_{1S}	H_{2S}	F_S
Averaged over reciprocals:															
V_p	$\frac{1}{4}$	—	—	—	$\frac{1}{4}$	$\frac{1}{2}$	—	1	—	—	—	—	—	—	—
$\bar{V}_r$	$\frac{1}{16}$	$\frac{1}{16}$	—	$-\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	$-\frac{1}{16}$	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$	—	—	—	—
$V_{\bar{r}}$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{16}$	$-\frac{1}{16}$	$\frac{1}{16}$	$\frac{1}{8}$	$-\frac{1}{16}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$	—	—	—	—
W_r	$\frac{1}{8}$	—	—	$-\frac{1}{16}$	$\frac{1}{8}$	$\frac{1}{4}$	$-\frac{1}{16}$	$\frac{1}{2}$	—	—	$-\frac{1}{4}$	—	—	—	—
$S \times V_p$	$\frac{1}{4}$	—	—	—	$\frac{1}{4}$	$-\frac{1}{2}$	—	—	—	—	—	1	—	—	—
$S \times \bar{V}_r$	$\frac{1}{16}$	$\frac{1}{16}$	—	$-\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	$\frac{1}{16}$	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$
$S \times V_{\bar{r}}$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{16}$	$-\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{8}$	$\frac{1}{16}$	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$
$S \times W_r$	$\frac{1}{8}$	—	—	$-\frac{1}{16}$	$\frac{1}{8}$	$-\frac{1}{4}$	$\frac{1}{16}$	—	—	—	—	$\frac{1}{2}$	—	—	$-\frac{1}{4}$
Not averaged over reciprocals:															
V_p	$\frac{1}{4}$	—	—	—	$\frac{1}{4}$	$\frac{1}{2}$	—	1	—	—	—	—	—	—	—
$\bar{V}_{r\varnothing}$	$\frac{1}{16}$	$\frac{1}{16}$	—	$-\frac{1}{16}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$	—	—	—	—
$\bar{V}_{r\delta}$	$\frac{1}{16}$	$\frac{1}{16}$	—	$-\frac{1}{16}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$	—	—	—	—
$V_{\bar{r}\varnothing}$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{16}$	$-\frac{1}{16}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$	—	—	—	—
$V_{\bar{r}\delta}$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{16}$	$-\frac{1}{16}$	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$	—	—	—	—
$W_{r\varnothing}$	$\frac{1}{8}$	—	—	$-\frac{1}{16}$	$\frac{1}{4}$	$\frac{3}{8}$	$-\frac{1}{16}$	$\frac{1}{2}$	—	—	$-\frac{1}{4}$	—	—	—	—
$W_{r\delta}$	$\frac{1}{8}$	—	—	$-\frac{1}{16}$	—	$\frac{1}{8}$	$-\frac{1}{16}$	$\frac{1}{2}$	—	—	$-\frac{1}{4}$	—	—	—	—
$S \times V_p$	$\frac{1}{4}$	—	—	—	$\frac{1}{4}$	$-\frac{1}{2}$	—	—	—	—	—	1	—	—	—
$S \times \bar{V}_{r\varnothing}$	$\frac{1}{16}$	$\frac{1}{16}$	—	$-\frac{1}{16}$	—	—	—	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$
$S \times \bar{V}_{r\delta}$	$\frac{1}{16}$	$\frac{1}{16}$	—	$-\frac{1}{16}$	$\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{8}$	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	—	$-\frac{1}{4}$
$S \times V_{\bar{r}\varnothing}$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{16}$	$-\frac{1}{16}$	$\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{8}$	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$
$S \times V_{\bar{r}\delta}$	$\frac{1}{16}$	$\frac{1}{16}$	$-\frac{1}{16}$	$-\frac{1}{16}$	—	—	—	—	—	—	—	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$-\frac{1}{4}$
$S \times W_{r\varnothing}$	$\frac{1}{8}$	—	—	$-\frac{1}{16}$	$\frac{1}{4}$	$-\frac{3}{8}$	$\frac{1}{16}$	—	—	—	—	$\frac{1}{2}$	—	—	$-\frac{1}{4}$
$S \times W_{r\delta}$	$\frac{1}{8}$	—	—	$-\frac{1}{16}$	—	$-\frac{1}{8}$	$\frac{1}{16}$	—	—	—	—	$\frac{1}{2}$	—	—	$-\frac{1}{4}$

15 parameters are specified. Since the coefficient of D_X is equal to that of D_X^* in every statistic these two parameters cannot be separated, but even allowing for their combination separation of the remaining effects is impossible.

If the data are not averaged over reciprocal crosses, 14 statistics are available and there are no obvious correlations between parameters. Thus a solution is only possible if one parameter is omitted from the model. The particular parameter to be omitted will depend on the particular circumstances. Thus if gene frequencies are roughly equal or dominance is negligible the appropriate F type parameters may be omitted or the H_1 and the H_2 type parameters may be combined. If autosomal sex-limitation is absent, or complete, the autosomal parameters can be reduced. The decision as to which course of action is least damaging may be gauged by consideration of the analyses of variance described earlier. The assumptions made in estimating these parameters include absence of epistasis and correlated gene distributions. For autosomal genes it has been shown that failure of these assumptions could lead to distortion of the estimates obtained if either effect is very marked (Coughtrey and Mather, 1970).

Acknowledgments.—I am indebted to Dr M. J. Kearsey for his help and advice during the course of this work and for constructive criticism of the manuscript. The work was done whilst in receipt of a Science Research Council Studentship.

4. REFERENCES

- ALLARD, R. W. 1956. The analysis of genetic environmental interactions by means of diallel crosses. *Genetics*, **41**, 305-318.
- COMSTOCK, R. E., AND ROBINSON, H. 1952. Estimation of average degree of dominance of genes. In *Heterosis*, pp. 494-516. Iowa State College Press.
- COUGHTREY, A., AND MATHER, K. 1970. Interaction and gene association and dispersion in diallel crosses where gene frequencies are unequal. *Heredity*, **25**, 79-88.
- DICKINSON, A. G., AND JINKS, J. L. 1956. A generalised analysis of diallel crosses. *Genetics*, **41**, 65-78.
- DURRANT, A. 1965. Analysis of reciprocal differences in diallel crosses. *Heredity*, **20**, 573-607.
- HAYMAN, B. I. 1954. The analysis of variance of diallel tables. *Biometrics*, **10**, 235-244.
- JINKS, J. L. 1954. The analysis of continuous variation in a diallel cross of *Nicotiana rustica*. *Genetics*, **39**, 767-788.
- JINKS, J. L., AND BROADHURST, P. L. 1963. Diallel analysis of litter size and body weight in rats. *Heredity*, **18**, 319-336.
- KILLICK, R. J. 1971. Sex-linkage and sex-limitation in quantitative inheritance. I. Random mating populations. *Heredity*, **27**, 175-188.
- MATHER, K., AND JINKS, J. L. 1971. *Biometrical Genetics*. Chapman and Hall, London.
- TOPHAM, P. 1966. Diallel analysis involving maternal and maternal interaction effects. *Heredity*, **21**, 665-674.
- WEARDON, S. 1964. Alternative analyses of the diallel cross. *Heredity*, **19**, 669-680.