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THE EXPECTED DISTRIBUTION OF MARKER-LINKED QUANTITATIVE EFFECTS IN CROSSES BETWEEN INBRED LINES

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Summary

The *a priori* theoretical distribution of marker-linked quantitative effects in crosses involving inbred lines, and the number of quantitative loci contributing to such effects was obtained by numerical analysis, and examined as a function of the number and effects of the quantitative loci differentiating the lines, the location of the marker relative to the chromosome ends, and the mapping function. Over a wide range of assumptions it was found that the probability that a given marker will have an associated linked quantitative effect equal in magnitude to 0·2 phenotypic standard deviations or greater is of the order of 0·10 or more. In most of these cases it can be expected that the marker-linked effects will be due to one, or at the most two, quantitative loci in the vicinity of the marker.

1. Introduction

Marker-linked quantitative effects have been demonstrated in a variety of organisms (Sax, 1923; Rasmusson, 1935; Thoday, 1961; Fasoulas and Allard, 1962; Law, 1967; Lieberman *et al.*, 1972; Chai, 1975) and Soller *et al.* (1976) have pointed out that crosses between inbred lines differing in their marker genotypes provide a potentially useful method of demonstrating such effects. The purpose of the present study is to examine *a priori* probabilities of finding marker-linked quantitative effects of a given magnitude in crosses involving inbred lines. The composition of such effects in terms of the expected number of quantitative loci involved was also investigated.

2. Theory

In crosses between inbred lines differing at a marker-locus, the quantitative difference between homozygous marker genotypes in the F_2 generation, due to the effects of n quantitative loci in the vicinity of the marker (henceforth: marker-linked effects) will equal

$$\sum_{i=1}^{n} (1-2r_i)(d_{1i}-d_{2i}) \qquad (1)$$

where,

r_i is the probability of recombination between the marker locus and the ith quantitative locus (of the n quantitative loci in the vicinity of the marker);

d_{1i} is the effect (including sign) of the ith quantitative locus in inbred line 1 (*i.e.*, $+d_i$ if the inbred line is homozygous for the plus allele at the

ith quantitative locus, and $-d_i$ if the line is homozygous for the minus allele); and

d_{2i} is the effect (including sign) of the ith quantitative locus in inbred line 2.

Thus, $(d_{1i}-d_{2i})$ is the difference in quantitative value between the two inbred lines at the ith locus, while $(1-2r_i)$ is the proportion of this difference that remains associated with the linked marker genotypes in the F_2 generation. From (1) it is clear that only quantitative loci for which $r<0{\cdot}5$ and for which d_1 and d_2 have opposite sign will contribute to quantitative effects showing linkage with a particular marker. Such loci are termed " differentiating loci ", and respective differences, $(d_{1i}-d_{2i})$ are termed " contrasts ".

The expected distribution of marker-linked effects was calculated according to the following procedure. The distribution of marker-linked effects due to a single differentiating locus in the neighborhood of a marker was calculated assuming a particular mapping function and array of gene effects. This distribution will be denoted Φ. The distribution Φ_n of linkage effects due to contributions from n differentiating loci in the neighbourhood of the marker ($n = 1, 2, \ldots, N$) will then be given by the distribution of the sum of n independent variables each having the distribution Φ_1. Assuming a random distribution of quantitative loci over the genome, the probability, P_n, of finding n quantitative loci in the vicinity of a given marker will have a Poisson distribution with parameter, $\lambda = kL$, where k is the mean number of differentiating loci per Morgan in the genome as a whole (henceforth: the density of differentiating loci), and L is the map length in Morgans of the region in the vicinity of a marker within which quantitative loci will produce a marker-linked effect (henceforth: the extent of the marker neighbourhood). The overall distribution of marker-linked quantitative effects is then given by a mixture of the sum of N independent Φ_n variables, using a Poisson mixing distribution with parameter λ *i.e.*, by combining the N independent Φ_n distributions according to their respective probabilities, P_n.

In practice the calculations were carried out numerically according to the following assumptions and procedures.

(i) *Scale*

The ability to establish marker-linked effects in crosses between inbred lines depends on the relative magnitude of the marker-linked effects as compared to the phenotypic standard deviation in the F_2 generation of the cross (Soller *et al.*, 1976). For this reason, gene effects and marker-linked effects in this study are given in units of the F_2 standard deviation (σ). Clearly σ will vary according to the number of loci differentiating the lines, gene effects at these loci, and the environmental variance affecting the F_2 progeny. In calculating the Φ distribution, gene effects are given in units of the F_2 phenotypic standard deviation σ_0 of a cross between two inbred lines with differentiating loci and environmental variance as given in section (ii).

For other arrays of gene effects and for other environmental variances, σ will differ from σ_0, and this will require appropriate changes in the scale of the Φ distribution. These are detailed in section (vi) and table 3.

(ii) *Gene effects*

In formulating arrays of gene effects for numerical calculations it should be noted that the heritability (h^2) of the trait under consideration in the F_2 generation places strong restraints on the total number, m_f, of differentiating loci, over the entire genome, that have any particular magnitude of effect, d_f. In particular, the additive genetic variance generated in the F_2 by a single differentiating locus of effect d_f will be $\frac{1}{2}d_f^2$ (Falconer, 1960); that generated by m_f such loci will $\frac{1}{2}m_f d_f^2$; and that generated by all differentiating loci will be $\sum_f \frac{1}{2}m_f d_f^2$. If the d_f are expressed in units of σ, then it can readily be shown that

$$h^2 = \sum_f \tfrac{1}{2}m_f d_f^2 \tag{2}$$

Thus, even large numbers of loci of small effect will make only a small contribution to h^2, while there cannot be more than a few loci having a large magnitude of effect, unless heritability is very high. In our case we assumed residual and additive genetic variances in the F_2 generation to be equal so that $h^2 = 0{\cdot}5$.

Two arrays of differentiating loci were now defined, (i) an exponential array, in which the probability of a given gene effect decreases in proportion to its magnitude, and (ii) a uniform array, in which all gene effects are equal. In practice the exponential array was approximated by an array consisting of five classes of loci, each class contributing one-fifth of the total F_2 additive genetic variance, with m_f = 1, 2, 4, 8 and 16, respectively. By (2) $d_f = (2h^2/m_f)^{\frac{1}{2}}$, thus for $h^2 = 0{\cdot}5$, the corresponding $d = (0{\cdot}2/m_f)^{\frac{1}{2}}$ are equal to 0·45, 0·33, 0·22, 0·16 and 0·11, respectively. These values are given at the top of table 1. For a uniform array with the same total number,

TABLE 1

Marker-linked effects for a single differentiating locus in the marker neighbourhood, according to gene effect and gene location, assuming an exponential array of gene effects and the theroretical mapping function [1,4].

Gene location		Gene array					
Segment boundaries [2]	Recombination percentage [3] (r_j)	Gene effect: (d_f) No. of loci: (m_f)	0·45 1	0·33 2	0·22 4	0·16 8	0·11 16
0-0·10	0·045		0·82	0·60	0·40	0·29	0·20
0·10-0·20	0·128		0·67	0·49	0·33	0·24	0·16
0·20-0·30	0·195		0·55	0·40	0·27	0·20	0·13
0·30-0·40	0·251		0·45	0·33	0·22	0·16	0·11
0·40-0·50	0·296		0·37	0·27	0·18	0·13	0·09
0·50-0·60	0·333		0·30	0·22	0·15	0·11	0·07
0·60-0·70	0·363		0·25	0·18	0·12	0·09	0·06
0·70-0·80	0·388		0·20	0·15	0·10	0·07	0·05
		Proportion of all loci (b_f)	0·0323	0·0625	0·1290	0·2581	0·5161
		Frequency of effect (p_{fj})	0·0040	0·0081	0·0161	0·0323	0·0645

[1] See text for details.
[2] Measured in Morgans from the marker.
[3] Calculated as the mean of the recombination percentages at the segment boundaries, using the theoretical mapping function. See text for additional details.
[4] In units of the phenotypic standard deviation in the F_2 generation of the crosses between the two inbred lines. See text for additional details.

T = Σm_f = 31 of differentiating loci and the same F_2 genetic variance, each locus will have an effect, d = 0·18. Assuming a total map length of 30 Morgans, T = 31 corresponds to a density, k, of 1·04 loci/Morgan. Some variations on these assumptions will be discussed in section (vii).

(iii) *Mapping functions*

Two mapping functions were considered, (i) the theoretical mapping function, $r = (1-e^{-2x})/2$, where x is the distance, in Morgans, from marker to locus (Stahl, 1969) and (ii), an empirical mapping function based on fig. 17-4 of Strickberger (1968, p. 338).

(iv) *Distribution of marker-linked effects given* n *loci in the marker neighbourhood*

(a) *Distribution of marker-linked effects, due to a single differentiating locus in the marker neighbourhood, (the Φ_1 distribution).*

For the exponential array of gene effects, the distribution of marker-linked effects due to a single locus in the marker neighbourhood (the Φ_1 distribution was calculated as follows:

(1) The marker neighbourhood was divided into consecutive chromosome segments of length 0·1 Morgan. A locus of effect d_f falling in the jth segment, will produce a marker-linked effect a_{fj} of magnitude $a_{fj} = (1-2r_j)(2d_f)$ where r_j is the probability of recombination with the marker (r_j was calculated as the mean of the recombination percentages at the two boundaries of the segment as given by the appropriate mapping function). Since the theoretical mapping function extends to infinity, the marker neighbourhood was terminated at the distance x, for which r = 0·40. For the theoretical mapping function this was at x = 0·8, (L = 1·6 for a centrally located marker) and for the empirical mapping function at x = 0·5 (L = 1·0 for a centrally located marker). The r_j for the theoretical mapping function are given at the left of table 1. The various marker-linked effects (a_{fj}) according to the effect of the locus (d_f) and its probability of recombination with the marker (r_j) are given in the body of table 1.

(2) Given a single locus in the marker neighbourhood, the expected frequency, p_{fj} of each a_{fj} was calculated by $p_{fj} = b_f(0{\cdot}1/L)$, where $b_f = m_f/T$ is the proportion of loci having effect d_f, and $0{\cdot}1/L$ is the probability that the locus falls in the *jth* segment (*e.g.*, if L = 1·6 Morgan, and there is a single locus in the marked neighbourhood, the probability that the locus falls in any particular segment of width 0·1 Morgan, is 0·1/1·6.

(3) The distribution of marker-linked effects due to a single locus was approximated by grouping the a_{fj} of table 1 into classes of width $0{\cdot}04\sigma_0$, and summing the corresponding p_{fj} to obtain the expected frequency of each class of single-locus marker-linked effect. This gives the distribution of marker-linked effects due to a single locus, Φ_1, for a marker at the end of the chromosome, or for a centrally located marker remote from both chromosome ends. For a subterminally located marker, certain a_{fj} will be included twice in the frequency table, others only once, depending on whether the corresponding chromosome segments are found to one side or to both sides of the marker, and the corresponding distribution will have to be calculated separately.

(b) *The distribution of marker-linked effects due to two or to three or more differentiating loci in the marker neighbourhood.*

The distribution of marker-linked effects due to two loci in the vicinity of a marker Φ_2 was obtained by taking the sum and expected frequencies of all possible two-way combinations of single-loci marker-linked effect by classes. Sums for each combination were calculated as the sum of the respective single-locus class midpoints. The expected frequency of each combination was calculated as the product of the respective single-locus class frequencies. The two-locus marker-linked effects were grouped in a frequency table with the same class widths as the single-locus effects and their respective frequencies summed to give the expected frequency of each class of two-locus marker-linked effect.

For more than two loci in the marker neighbourhood, the distribution of marker-linked effects was obtained by assuming that the sum of n single-locus marker-linked effects will be normally distributed with parameters, $u_n = n\bar{d}$ and $\sigma_n^2 = n\sigma_d^2$ where $\bar{d}$ and σ_d^2 are the mean and variance of the Φ_1 distribution. For the values of k, n and h^2 investigated, P_n were very small for $n = 5$ or more, so that there was no need to calculate the corresponding Φ_n.

The distribution of marker-linked effects for $n = 1, 2, 3$, or 4, as calculated from the Φ_1 distribution, are shown in table 2. Similar distributions (not shown) were calculated for a subterminal marker ($L = 0{\cdot}8+0{\cdot}3$), for a uniform array of gene effects, and for the empirical function.

TABLE 2

Distribution of marker-linked effects for n = 1, 2, 3 *or* 4 *loci in the marker neighbourhood*

Effect[1]	Frequency of effect			
	$n = 1$	$n = 2$	$n = 3$	$n = 4$
0·00-0·039	—	—	0·010	0·003
0·04-0·079	0·226	—	0·007	0·002
0·08-0·119	0·210	—	0·010	0·004
0·12-0·159	0·137	0·051	0·015	0·005
0·16-0·199	0·121	0·095	0·022	0·007
0·20-0.239	0·125	0·106	0·029	0·010
0·24-0·279	0·061	0·112	0·038	0·014
0·28-0·319	0·036	0·126	0·047	0·018
0·32-0·359	0·024	0·113	0·057	0·026
0·36-0·399	0·004	0·091	0·066	0·031
0·40-0·439	0·024	0·073	0·069	0·037
0·44-0·479	0·004	0·052	0·078	0·044
0·48-0·519	0·008	0·043	0·080	0·054
0·52+	0·020	0·139	0·472	0·745

[1] In units of the phenotypic standard deviation in the F_2 generation. See text for details.

(v) *The Poisson parameter and mixing procedure*

As described at the beginning of this section, the expected proportion of marker neighbourhoods that include $n = 1, 2, 3$ or 4 quantitative loci is given by a Poisson distribution with Poisson parameter $\lambda = kL$, where k and L depend on the specific situation investigated. For the theoretical mapping function, exponential gene array, the densities investigated were

$k = 1{\cdot}04$, $0{\cdot}52$ and $0{\cdot}26$ and L was variously taken as $0{\cdot}8$ for a terminal marker, $1{\cdot}1$ for a subterminal marker and $1{\cdot}6$ for a centrally located marker. For the theoretical mapping function, uniform gene array, k was taken as $0{\cdot}52$ and L as $0{\cdot}8$ and $1{\cdot}6$. For the empirical mapping function, exponential array, k was taken as $0{\cdot}52$ and L as $0{\cdot}5$ and $1{\cdot}0$ for a terminal marker and a centrally located marker, respectively. Values of L were determined by the $r = 0{\cdot}40$ cut-off point as described in section (iva). Values of k were taken to cover the interesting situation where quantitative loci are relatively few, so that total effect of all quantitative loci is relatively small, and probabilities of marker-linked effects of significant magnitude are also small. Higher densities, at the given heritability, imply many quantitative loci, having total effects large enough, so that the probability of a significant marker-linked effect is high.

(vi) *The effect of density of differentiating loci and of environmental variance on the distribution of marker linked effects.*

The effect of the density of differentiating loci and environmental variance on the distribution of marker-linked effects was investigated by varying k and environmental variance while keeping the total map length constant. For given map length, changes in the density of differentiating quantitative loci do not change the shape of the Φ_1 distribution, but will change the total number of segregating loci, and hence the genetic variance, heritability and phenotypic standard deviation in the F_2. Thus, after carrying out the mixing procedure the scale of the resultant frequency distribution must be multiplied by an appropriate factor in order to express marker-linked effects in terms of the new F_2 phenotypic standard deviation. Similar considerations apply when environmental variance is changed. Table 3 gives appropriate scale factors for the various situations investigated.

TABLE 3

Scaling factors according to density of quantitative loci (k) and environmental variance [1]

Density (k) (loci/Morgan)	Genetic variance	Environmental variance	Phenotypic variance	Scale factor	h^2
1·04	0·500	0·500	1·000	1·000	0·50
0·54	0·250	0·500	0·750	1·157	0·33
0·26	0·125	0·500	0·625	1·265	0·20
0·52	0·250	1·000	1·250	0·894	0·20
0·52	0·250	0·0625	0·3125	1·789	0·80

[1] All variances in units of the standard phenotypic variance, σ_0^2, see text for details.

For example, genetic variances for $k = 0{\cdot}54$ and $0{\cdot}26$ are only $0{\cdot}5$ and $0{\cdot}25$ of genetic variance for the standard situation, $k = 1{\cdot}04$. This will reduce phenotypic standard deviation in these situations to $0{\cdot}87\ \sigma_0$ and $0{\cdot}79\ \sigma_0$ respectively, and the appropriate scale factors will be $1{\cdot}157$ and $1{\cdot}265$.

3. RESULTS

Fig. 1 shows the cumulative distribution of marker-linked effects of given magnitude or greater according to the density of quantitative loci in the genome (k), and the extent of the marker neighbourhood (L), assuming a

total map length of 30 Morgans, the theoretical mapping function, and the exponential array of gene effects. The probability of an effect of a given magnitude or greater varied over a wide range, showing a fairly direct proportionality to density, and within densities to the extent of the marker neighbourhood. That is, probabilities of a given magnitude of effect or greater were proportional to the expected number of loci in the marker neighbourhood. This is reasonable. At constant total map length, densities

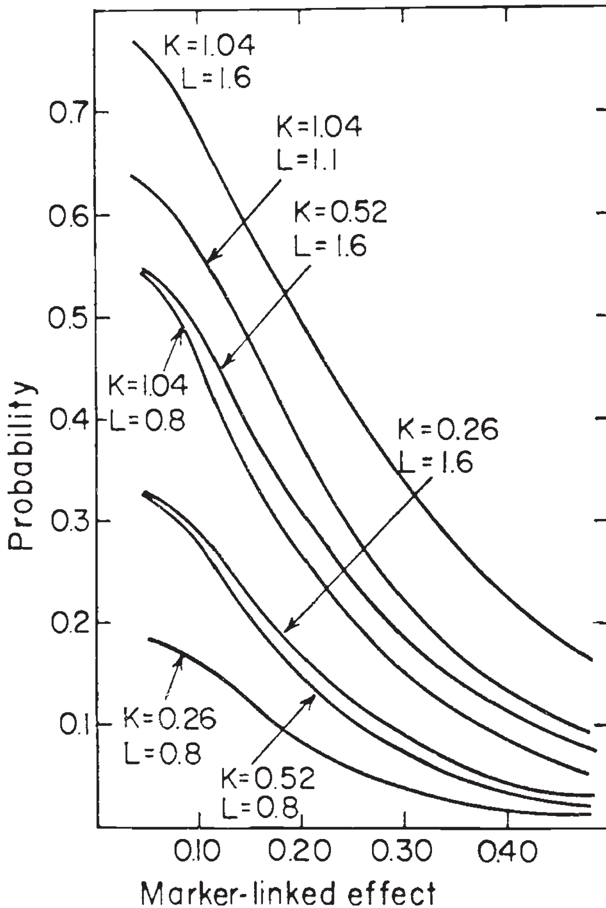

Fig. 1.—Cumulative distribution of marker-linked effects of given magnitude or greater, according to the density of quantitative loci in the genome (k), and the extent of the marker neighbourhood (L), for the theoretical mapping function and exponential array of gene effects, assuming $h^2 = 0{\cdot}50$ (see text for details).

of $k = 0{\cdot}52$ and $0{\cdot}24$ involve a proportional reduction in the genetic variance, relative to the standard situation ($k = 1{\cdot}04$) and a corresponding (though not proportional) reduction in phenotypic variance. This results in a scale effect as discussed in sections 2(i) and 2(vi) and is the reason that the curve for $k = 0{\cdot}52$, $L = 1{\cdot}6$ is slightly higher than the curve for $k = 1{\cdot}04$, $L = 0{\cdot}8$, even though the expected number of loci in the marker neighbourhoods are the same in both cases.

Fig. 2 shows the effect of changes in the environmental variance on the distribution of marker-linked effects for $k = 0{\cdot}52$. Other assumptions are as in fig. 1. For marker-linked effects of intermediate magnitude ($0{\cdot}1 - 0{\cdot}3\ \sigma$) doubling the environmental variance ($h^2 = 0{\cdot}2$) reduced probabilities by about one-third, while an eight-fold reduction in environmental variance ($h^2 = 0{\cdot}8$), increased probabilities by about one-half.

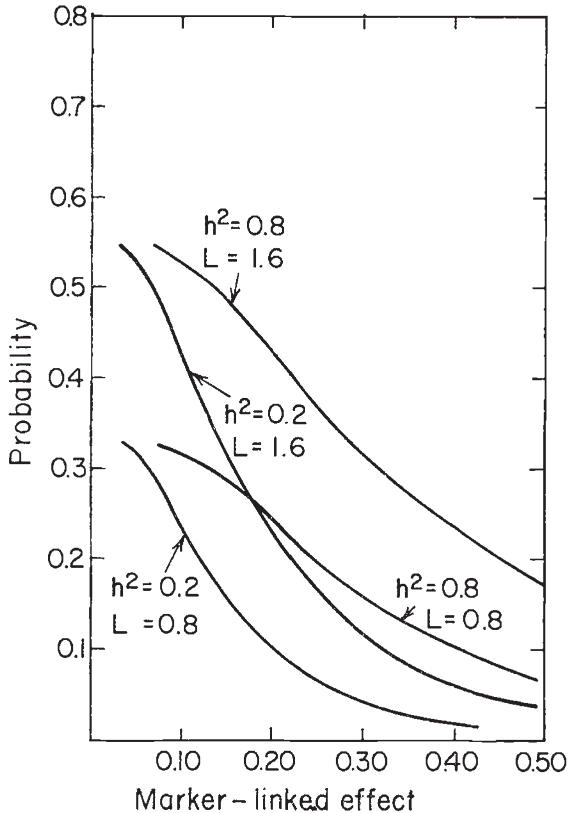

Fig. 2.—Cumulative distribution of marker-linked effects of given magnitude or greater, according to the heritability (h^2) and extent of the marker neighbourhood (L), for the theoretical mapping function and exponential array of gene effects, assuming $k = 0{\cdot}52$ (see text for details).

Fig. 3 shows the effect of the mapping function and the array of gene effects on the distribution of marker-linked effects for $k = 0{\cdot}52$. Other assumptions are as in fig. 1. For the uniform array of gene effects, probabilities were somewhat greater than for the corresponding exponential array. For the empirical mapping function, probabilities were less than for the corresponding theoretical function, but this can be explained almost entirely by the difference in neighbourhood extent, being 1·0 Morgan for the empirical mapping function, and 1·6 Morgan for the theoretical mapping function.

Table 4 shows the proportion of marker-linked effects of magnitude $0{\cdot}2\ \sigma$ or greater that are due to one or two loci, under the various situations examined. In all cases the bulk of effects of $0{\cdot}2\ \sigma$ or greater can be attributed

to no more than two loci, and for the lower densities and smaller marker neighbourhoods, over half of the effects are due to one locus only.

(i) *Other experimental possibilities*

The distribution of marker-linked effects given above can be considered to refer to the expected results in a cross between two inbred lines out of opposite selection lines derived from a single base population segregating at k quantitative loci per Morgan. In the cross between two such selection-line inbreds, except for drift effects, the expected density of differentiating

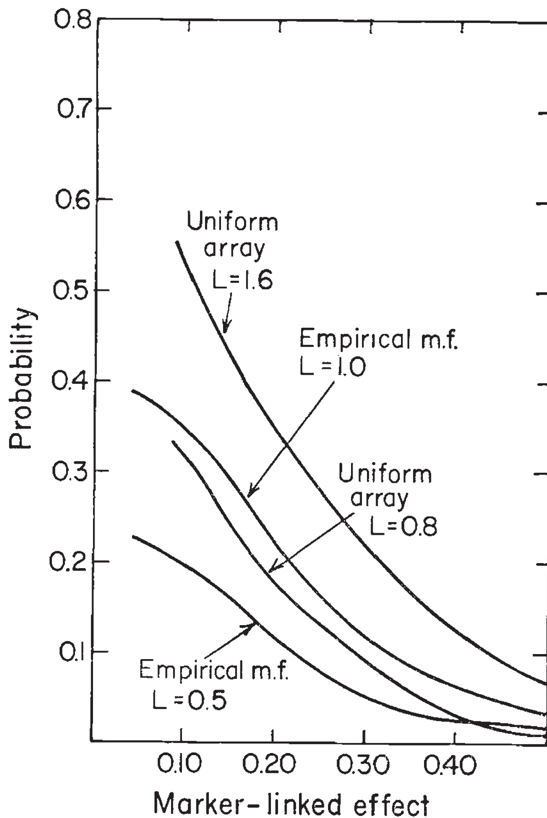

Fig. 3.—Cumulative distribution of marker-linked effects of given magnitude or greater, for the empirical mapping function (m.f.) and uniform array of gene effects, according to the extent of the marker neighbourhood (L), assuming $k = 0{\cdot}52$ (see text for details).

loci will also be k, and almost all contrasts will have the same sign. Distribution of marker-linked effects for experiments based on lines or individuals bearing other relationships to each other or to the base population will differ from the above distributions in the expected density of differentiating loci and in the uniformity of contrast sign. Lack of uniformity in contrast sign, however, will have only a small effect on the distribution of marker-linked effects. It will not affect the distribution of single-lows marker-linked effects at all, and trial calculations show that it will have only a minor effect

TABLE 4

Probability of a marker-linked effect of magnitude 0·2 σ or greater, and the proportion of effects of such magnitude attributable to one locus or to two loci in the marker neighbourhood, according to distribution of gene effects, mapping function, density of differentiating loci (k), *heritability* (h²), *and the extent of marker neighbourhood* (L). *Total map length constant, at 30 Morgans.*

Gene effects	Mapping function	k	h^2	L	Probability of effects	Proportion due to 1 locus	Proportion due to 2 loci
Exponential	Theoretical	1·04	0·50	0·8	0·28	0·40	0·46
				1·1	0·39	0·36	0·45
				1·6	0·50	0·19	0·45
Exponential	Theoretical	0·52	0·33	0·8	0·15	0·68	0·32
				1·6	0·31	0·44	0·43
Exponential	Theoretical	0·26	0·20	0·8	0·09	0·84	0·16
				1·6	0·17	0·70	0·30
Exponential	Theoretical	0·52	0·20[2]	0·8	0·10	0·60	0·40
				1·6	0·23	0·35	0·49
Exponential	Theoretical	0·52	0·80[3]	0·8	0·24	0·78	0·22
				1·6	0·43	0·57	0·34
Exponential	Empirical	0·52	0·33	0·5	0·12	0·80	0·20
				1·0	0·23	0·67	0·33
Uniform	Theoretical	0·52	0·33	0·8	0·18	0·69	0·31
				1·6	0·35	0·47	0·42

[1] See text for details.
[2] Environmental variance increased by a factor of 2·0, genetic variance unchanged.
[3] Environmental variance decreased by a factor of 0·125, genetic variance unchanged.

on the distribution two-locus marker-linked effects (note that in this case both contrasts will still have the same sign half the time). Thus the distribution of marker-linked effects for other situations can be obtained from figs. 1-3, after taking into account effects on the density of differentiating contrasts. Lack of uniformity in contrast sign can generally be neglected, except to perhaps subtract a percentage point or two from the appropriate probabilities in figs. 1-3. For example, in a cross between two inbred lines, both derived independently from the same, unselected, base population, the proportion of differentiating loci will be $2\overline{pq}\, k$, when $2\overline{pq}$ is the mean value of $2pq$ over all segregating loci. Hence if the base population were segregating at k loci, the density of differentiating loci would be no more than $k/2$ (probably closer to $k/2{\cdot}5$) and the values of figs. 1-3 apply, according to the appropriate density. These values should however be reduced somewhat since contrasts in this case can be both positive and negative: *e.g.*, assuming $k = 1{\cdot}04$ in the base population, the probabilities of a marker-linked effect of magnitude 0·2 σ or more will be 0·10 to 0·15 for this situation, compared to 0·28 for a cross involving two selection-line inbreds.

Table 5 gives densities and the state of uniformity of signs of contrasts for various experimental possibilities assuming $2pq = 0{\cdot}5$ (the maximum possible). The cross between an inbred line and a single outbred individual is of particular interest since potentially it would enable a variety of outbred populations to be sampled and compared with respect to the presence of useful quantitative loci. In this case marker-linked effects of loci heterozygous in the outbred individual will be small so that any marker-linked effects will be produced primarily by quantitative loci for which the outbred

TABLE 5

Density of differentiating loci[1] and uniformity of contrast signs for various experimental crosses

Parent 1	Parent 2	Density	Contrast sign
Inbred out of selection line	Inbred out of opposite direction selection line from same base population	k	Uniform
Inbred out of selection line	Inbred out of same base population, no selection	$k/2$	Uniform
Inbred out of unselected base population	Inbred, independently derived from same unselected base population	$k/2$	Not uniform
Inbred out of unselected base population	Single outbred individual from same base population[2]	$k/4$	Not uniform

[1] Density of segregating quantitative loci in the unselected base population denoted by k, $\overline{2pq}$ assumed equal 0·5 (see text for details).
[2] Not including any potential contribution from loci heterozygous in the outbred.

individual is homozygous for different alleles than the inbred. Neglecting heterozygotes, the density of differentiating loci for an inbred line and outbred individual, both of the same base population, will be at most $k/4$, and the differentiating contrasts will be either positive or negative in sign. Because of the low density of differentiating loci in this design, frequencies of marker-linked effects of magnitude 0·2 σ or greater will be low, and the design is therefore not too encouraging. If, however, the outbred individual comes from a population that differs from the inbred base population, frequencies of such effects will be greater. Also, if a number of outbred males from the same population are tested independently, then for a given k the overall frequencies of marker-linked effects of given magnitude turning up in at least one of the tested males will approach those for a cross between inbred lines. Of course this will require a considerable increase in the number of offspring scored.

4. DISCUSSION

The results of this study show that under the given assumptions, experiments with good power against effects of magnitude 0·2 σ appear to have reasonable expectation of detecting marker-linked effects over a wide range of assumptions as to the number and distribution of quantitative loci. In theory, at least, experiments of this power would require no more than a few hundreds of offspring per marker genotype in the F_2 generation (Soller *et al.*, 1976) and thus could be carried out in many plants and some animals. The results of this study also suggest that in many cases observed marker-linked effects will be primarily due to single quantitative loci. Once identified, such loci may be suitable for physiological or genetic studies, or for controlled introgression in plant and animal improvement (Soller and Plotkin-Hazan, 1978). Various plant species, particularly selfers such as barley, tomato, and some tetraploid wheats are prime candidates for such analyses in view of their economic significance and the potential usefulness in their further improvement of controlled introgression of evaluated chromosome segments from wild populations. Among agricultural animals, poultry seem the most likely candidate for such studies (Soller *et al.*, 1976).

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