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Choice of method for identifying germplasm with superior alleles

2. Computer simulation results *

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Summary. An accurate and efficient method of screening the many germplasm sources available for their ability to improve elite, adapted germplasm is needed. The superiority measure (SX) of a population (P) was defined as the product of the frequency and relative superiority of the alleles in P that are more favorable than the best in an elite, adapted reference single cross I1 × I2. A computer simulation was done to determine the correlations between various screening methods and the SX. The genetic model used included multiple alleles, no linkage, two types of non-epistatic gene action (additive and complete dominance) and two types of epistatic gene action (complementary and duplicate). Genetic variances in the populations and a statistic proposed by Dudley (SD = {[P × I1 − I1] [I1 × I2 − I2] − [P × I2 − I2] [I1 × I2 − I1]}/{2[I1 − I2]}) were inconsistently correlated with the SX over all types of gene action on the basis of rank correlations. The testcross to the single cross (TC[SC] = P × [I1 × I2]) and the upper bound on the SX (UBND = minimum [P × I1 − I1, P × I2 − I2]) were both consistently highly genetically correlated with the SX. In the set of populations simulated, there were positive correlations between products of allelic frequencies and effects at different classes of loci. The UBND usually had a higher rank correlation coefficient with the SX than did the TC(SC). The differences between their correlation coefficients were often insignificant. Although the TC(SC) gives no indication as to which inbred the population is more closely related, its ease of use and expected lower standard error compared with the UBND indicate that it would be an appropriate choice of screening method for identifying superior populations in the sense defined.

Key words: Exotic germplasm – Computer simulation – Testcross – Upper bound – Germplasm choice

Introduction

Choice of germplasm lays the foundation for an effective breeding program. Recently, there has been concern that the genetic base of our major crops may have been seriously eroded by intense selection of superior genotypes. The adapted, elite germplasm currently being utilized for any crop certainly does not contain all the most desirable alleles available in a species or breeding pool (Brown 1983). Immediate utilization of non-elite or unadapted germplasm is frequently hindered by low mean performance, photoperiod sensitivity or other undesirable agronomic traits. Adapted, elite germplasm is the preferred source for favorable alleles because it is least likely to contain undesirable alleles. In using unadapted or non-elite germplasm, a breeder is primarily interested in material that can contribute alleles more favorable than those available in elite germplasm.

There are tremendous numbers of accessions of important crop species in germplasm banks [e.g., 400,000 of wheat (*Triticum* species), 200,000 of rice (*Oryza* species), 175,000 of barley (*Hordeum* species) and 77,000 of maize (*Zea mays*), according to Plucknett et al. 1983]. A relatively simple, resource-efficient method of screening these accessions and other potentially useful germplasm is needed. The method used depends on breeding goals. For traits exhibiting little interaction with specific environments, evaluation of populations per se for the trait of interest is desirable and straightforward. For a quantita-

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tively inherited trait such as grain yield, the choice of a screening method is not nearly so obvious. Several have been used: (1) evaluation per se (Wellhausen 1956; Burton and Davies 1984); (2) crossing to other populations in diallels (Eberhart 1971; Josephson 1982); (3) crossing to an elite single cross (Kramer and Ullstrup 1959; Stuber 1978); and (4) crossing to elite inbreds (Burton and Davies 1984). The amount of genetic variance in the population has also been suggested as a consideration in choosing populations (Hallauer and Miranda 1981). Dudley (1984) suggested estimating the potential contribution of a population by using a function of measurements of elite inbreds, their single cross and testcrosses to the inbreds.

Computers have been used to elucidate genetic problems since Fraser (1957a, b) reported on their use in attempting to understand the effect of linkage on response to mass selection. Comparing different screening methods for identifying populations that can contribute favorable alleles to elite germplasm is complicated by the fact that allelic frequencies and values at loci controlling a quantitative trait are not known. Results from different screening methods can be compared, but true superiority of real populations cannot be easily determined. For this reason, theoretical or computer methods are helpful in objectively evaluating screening methods. The estimator suggested by Dudley, the testcross to the single cross, and an upper bound on the measure of superiority have been compared on the basis of their expected genetic correlation with a measure of superiority (Gerloff and Smith 1988). The objective of the present research was to consider the effects of several different multi-allelic models of gene action, including epistatic models, on choice of a method to identify populations that contain alleles more favorable than those present in an elite single cross. A computer-assisted approach was chosen because of the difficulties in mathematically describing a complex model involving multiple alleles and epistasis.

Theory

Using a model of two alleles per locus in a diploid species with regular Mendelian inheritance, Dudley (1984) subdivided the loci affecting a quantitative trait into four classes based on frequencies of the favorable allele in homozygous inbreds I1 and I2, which represented the parents of an elite single cross. Both inbreds were favorable and unfavorable at loci in classes i and l, respectively. At class j loci, I1 was favorable and I2 was unfavorable. At class k loci, I2 was favorable and I1 was unfavorable. The population to be evaluated, P, had average frequencies of the favorable allele at the i-th, j-th, and k-th classes of loci equal to $\bar{p}$ and at the l-th class equal to $\bar{p}_l$ (Table 1). The number of loci in their respective classes were denoted by i, j, k, and l. Half the difference between the values of the two homozygotes was denoted by u (Comstock and Robinson 1948). All loci were assumed to have the same value of u, and dominance was complete with no epistasis. The elite single cross was considered to contain the highest concentration of favorable alleles available in elite, adapted germplasm. The statistic (which we call SD) equal to $[(P \times I1 - I1)(I1 \times I2 - I2) - (P \times I2 - I2)(I1 \times I2 - I1)]/[2(I1 - I2)]$ estimates $l\bar{p}_l u$ for the population when the measurements for the trait of interest replace the designations for P, I1, I2 and their crosses. The product lu is a constant from population to population, given a specific reference single cross I1 × I2. Comparison of estimates of $l\bar{p}_l u$ for different populations compares their $\bar{p}_l$ values. These estimates of $l\bar{p}_l u$ are then relative measures of the frequency of the favorable allele in populations at loci where only the unfavorable allele is available in the elite single cross. If dominance is in the favorable direction, $l\bar{p}_l u$ will be greater than or equal to zero. If equal to zero, the population has no favorable alleles to contribute that are not already present in the reference single cross.

Table 1. Average frequencies of the favorable allele in homozygous inbreds I1 and I2 and population P at four classes of loci [from Dudley (1984) with minor notational changes]

Class	Frequency of the favorable allele		
	I1	I2	P
i	1.0	1.0	$\bar{p}$
j	1.0	0.0	$\bar{p}$
k	0.0	1.0	$\bar{p}$
l	0.0	0.0	$\bar{p}_l$

Gerloff and Smith (1988) considered a genetic model with two alleles per locus, complete dominance, no epistasis, unequal average frequencies of the favorable allele at each class of loci and unequal allelic effects at each locus controlling a quantitatively inherited trait. The superiority measure of a population was defined as $\sum_l p_l u_l$, where p_l was the frequency of the favorable allele in a population and u_l was the value of u at the l-th locus in class l. This represents the frequencies of the favorable alleles weighted by their effects at loci where the favorable allele must be sought outside the elite single cross. An upper bound (UBND) could be placed on $2\sum_l p_l u_l$ by choosing, for each population, the minimum of the difference between the testcross of the population to each of the inbred parents of the reference single cross and their respective inbred values (i.e., minimum$[P \times I1 - I1, P \times I2 - I2]$). The relatively simple statistics UBND, SD and the testcross to the reference single cross were compared on the basis of their theoretical genetic correlation with $\sum_l p_l u_l$. This was done by assuming no correlations

in a set of tested populations between the sums of products of frequencies and effects of alleles at the j, k and l classes of loci defined by Dudley (1984). That is, it was assumed that correlations between $\sum_j p_j u_j$, $\sum_k p_k u_k$ and $\sum_l p_l u_l$ were zero, where the subscripts denote the locus and p and u are the frequency of the favorable allele and half the difference between the values of the homozygotes, respectively. The testcross to the reference single cross, TC(SC), was expected to have the highest correlation with the superiority measure. It was predicted that multiple alleles would give the same results. The effects of epistasis and correlations between the different classes of loci in the populations to be screened were unpredictable.

Materials and methods

Model

Simulations were done using the VS FORTRAN programming language. The basic steps in the computer simulation of genotypic means in each replication were:

1. Generation of two sets of gametes from each population and tester. Thirty gametes were generated per population or tester in each set of gametes for each of four replications.
2. Combining of gametes to form zygotes. Populations were crossed using the first set of gametes for all crosses. The second set of gametes was used to make within-population crosses (i.e., to determine population per se means).
3. Determination of the genotypic value of each individual on the basis of specified type of gene action.
4. Determination of the mean of the 30 individuals representing a particular cross, resulting in the value of the entry for that replication. Means over all 4 replications were equivalent to means of 120 individuals per entry.

Forty loci affected the trait, with six possible alleles per locus. This resulted in 21 unique genotypes at each locus because parentage of an allele was ignored.

Values for the 6 alleles at each of the 40 loci were generated pseudo-randomly from a normal (0,1) distribution by using the subroutine GGNPM of International Mathematical and Statistical Libraries (IMSL). Values for the alleles were ranked within a locus so that allele 1 (A_1) at a locus had the lowest value and allele 6 (A_6) at that locus had the highest value. All random numbers needed from a uniform (0,1) distribution were generated by using the GGUBS subroutine of IMSL.

Genotypic value of an individual was based on four different models of gene action: two non-epistatic (additive and dominant) and two epistatic (complementary and duplicate).

For the non-epistatic models, let v_i^n = value of the i-th allele at the n-th locus, let y_{ij}^n = contribution of the n-th locus to total genotypic value in an individual with alleles i and j, and let Y = genotypic value of an individual. When gene action was additive,

$$y_{ij}^n = v_i^n + v_j^n \quad \text{and} \quad Y = \sum_n y_{ij}^n .$$

Values of alleles were additive within and between loci. When gene action was dominant,

$$y_{ij}^n = 2[\text{maximum}(v_i^n, v_j^n)] \quad \text{and} \quad Y = \sum_n y_{ij}^n .$$

There was complete expression of the more favorable allele at a locus and no interactions between loci.

In the epistatic models, dominance was assumed to be complete within a locus. Loci interacted in sequential pairs. Locus 1 interacted with locus 2, locus 2 with locus 3 (and with 1), and so on, with locus 40 interacting with both loci 39 and 1. The genotypic value of an individual was determined by summing the contributions of the 40 interacting pairs of loci. The epistatic gene actions were multiple allele extensions of the definitions for two allele models. For complementary gene action, the genotypic contribution of the r-th interacting pair of loci (loci a and b) in an individual with alleles i, j and k, l at loci a and b, respectively, was minimum (y_{ij}^a, y_{kl}^b). On a biological level, either locus in an interacting pair might control the rate of a limiting step in the same pathway. For duplicate gene action, the genotypic contribution of the r-th pair was maximum (y_{ij}^a, y_{kl}^b). This might be demonstrated by the products of different interacting loci each facilitating the same biochemical reaction; alleles at the loci would vary in efficiency of catalyzing the reaction.

Population derivation

Eight of the 40 loci, every fifth one, were considered to be adaptive loci. The remaining 32 loci were called trait loci. Populations or testers that were adapted had only one or two of the three most favorable alleles (A_4, A_5 or A_6) present at the adaptive loci. Unadapted populations had only one or two of the three least favorable alleles (A_1, A_2 or A_3) at adaptive loci. If two alleles were present in the population at adaptive loci, they had equal frequencies. Hence, unadapted populations never had a better allele at an adaptive locus than any allele at that same locus in adapted entries. This automatically put non-adapted populations at a disadvantage relative to adapted populations that had the same allelic frequencies at trait loci. This was done to ensure that, when gene action was not additive, some unadapted populations might have a low mean performance per se but could perform reasonably well in testcrosses, as was observed by Stuber (1978).

The first one or two letters of the population code (Table 2) indicate: (1) a relationship to an inbred (A, B or C) or hybrid (A × B = AB, A × C = AC, or B × C = BC); (2) a population with multiple alleles per locus (M); (3) a population with two alleles per locus (T) formed by pooling alleles in a multi-allelic population; or (4) a population with low frequencies of the most favorable alleles at many loci (X). The numeral in the code designates a particular type of relationship (i.e., type 1 or 2 to inbreds, type 3 or 4 to hybrids) or a parameter set (e.g., type 5 for a particular set of frequencies at trait loci). All populations designated by the last letter U are unadapted; those with A for the last letter are adapted.

Three source populations were specified (A1A, B1A and C1A) such that inbreds derived from them (A, B and C, respectively) would exhibit heterosis when all possible single crosses were produced and gene action was not additive. The source populations always contained either A_1 or A_2 at trait loci. There was an equal probability of a non-zero frequency for each of the other four alleles. Because a large amount of heterosis was desired among inbreds derived from these populations, allelic frequencies in one population complemented those of another population for one-third of the loci. Thus, one source population complemented the two other source populations at a total of two-thirds (26 or 27) of the loci. Actual frequencies were chosen for one population by rounding off a series of pseudo-random numbers from a uniform (0, 1) distribution to the nearest 0.1 and assigning that number (p) as the frequency of alleles 1 or 2. Choice of A_1 or A_2 was made independently, with each having an equal chance of being selected. Frequency of A_3, A_4, A_5 or A_6 was set at 1-p, and choice of allele was again independent with each of the four having an equal chance of occurrence. In

Table 2. Summary of simulated populations

Unadapted	Trait loci description	Adapted
A1U	Source population for inbred A	A1A
B1U	Source population for inbred B	B1A
C1U	Source population for inbred C	C1A
A2U	$p=0.6$ for allele fixed in inbred A	
B2U	$p=0.6$ for allele fixed in inbred B	
C2U	$p=0.6$ for allele fixed in inbred C	
AB3U	p_5 or $p_6=0.3$ at 1/2 loci where single cross AB has only A_1, A_2 or A_3	AB3A
AC3U	p_5 or $p_6=0.3$ at 1/2 loci where single cross AC has only A_1, A_2 or A_3	
BC3U	p_5 or $p_6=0.3$ at 1/2 loci where single cross BC has only A_1, A_2 or A_3	
AB4U	p_5 or $p_6=0.8$ at 1/4 loci where single cross AB has only A_1, A_2 or A_3	
AC4U	p_5 or $p_6=0.8$ at 1/4 loci where single cross AC has only A_1, A_2 or A_3	AC4A
BC4U	p_5 or $p_6=0.8$ at 1/4 loci where single cross BC has only A_1, A_2 or A_3	
M5U	Multiple alleles/locus	M5A
T5U	2 allele/locus version of M5U	
M6U	Multiple alleles/locus	
T6U	2 allele/locus version of M6U	
X7U	p_5 or $p_6=0.1$; p_1, p_2 or $p_3=0.9$	X7A

the population complementing this population at that locus, the frequency of A_1 or A_2 was 1-p with choice of allele independent of the allele present in the first population and independent of frequency. Frequency of one allele from the set A_3, A_4, A_5 and A_6 was specified as p.

Inbreds A, B and C were derived from their respective source populations by fixing the inbred for the most common allele at each locus in the source population. Hence, all inbreds were adapted. When the frequency of both alleles was 0.5 in the source population, the fixed allele in the inbred was a random choice between the two.

The single-cross testers were produced by making all possible crosses between the inbred testers, excluding reciprocal crosses.

At trait loci, populations A2U, B2U and C2U had allelic frequencies of 0.6 for the allele fixed in inbreds A, B and C, respectively. The allelic frequency was 0.4 for another allele at that locus, with every other allele having an equal probability of being the second allele.

For half the trait loci (determined at random) where single crosses AB, AC and BC, respectively, had only the most unfavorable alleles (A_1, A_2 or A_3), the populations AB3U, AC3U and BC3U had the frequency of A_5 or A_6 (p_5 or p_6) equal to 0.3. Choice of either A_5 or A_6 was random. The frequency of A_1, A_2 or A_3 was 0.7 at these loci, with choice of a particular allele made at random. At all other loci, these populations had one or two of the three least favorable alleles. If two of the alleles were present, they both had a frequency of 0.5.

Unadapted populations AB4U, AC4U and BC4U had a high frequency (0.8) of a favorable allele (A_5 or A_6) at only one-fourth the loci where the respective single cross contained only A_1, A_2 or A_3. The frequency of A_1, A_2 or A_3 at those loci was 0.2. At all other loci, they had only one or two alleles from the set A_1, A_2 or A_3.

M5U and M6U were unadapted populations whose trait loci were not restricted to having only two alleles per locus, nor were the frequencies of alleles restricted to particular values. T5U and T6U corresponded to M5U and M6U, respectively, except that they never had more than two alleles at a locus. For loci where M5U and M6U had multiple alleles, alleles were grouped with number of alleles grouped and choice of designated allele, chosen from among those grouped together, being random and independent between populations. The frequency of the designated allele was the sum of the frequencies of the alleles in the group.

X7U had a low frequency (0.1) of a very favorable allele (either A_5 or A_6) and a high frequency (0.9) of an unfavorable allele (A_1, A_2 or A_3) at all trait loci.

For all trait loci, the unadapted populations had the same allelic frequencies as their adapted counterparts. At adaptive loci, they had allelic frequencies chosen as for other unadapted or adapted populations, respectively. Choice of allelic frequencies at adaptive loci was independent for each population and was not dependent on the frequencies in their counterparts. A1U, B1U and C1U were unadapted versions of source populations A1A, B1A and C1A, respectively. AB3U, AC4U, M5U and X7U were unadapted versions of AB3A, AC4A, M5A and X7A, respectively. Not all populations had corresponding adapted and unadapted versions.

Analysis

SD estimates were made by using backcrosses $BC1=(I1\times I2)\times I1$ and $BC2=(I1\times I2)\times I2$ by noting that, using the genetic model of two alleles per locus, complete dominance, no epistasis, equal allelic effects at each locus and equal average frequencies of the favorable allele at loci in the i-th, j-th, and k-th classes:
1. $2(BC1-BC2)$ estimates $2(j-k)u$, the genotypic value of $I1-I2$;
2. $2(BC1)-(I1\times I2)$ estimates $(i+j-k-l)u$, the mean of I1; and
3. $2(BC2)-(I1\times I2)$ estimates $(i-j+k-l)u$, the mean of I2.
Appropriate substitutions were then made in the SD estimating function. Calculations of SDs using inbred data are denoted by ISD; those where backcross information was used are denoted by BCSD.

Upper bounds (UBNDs) were calculated by taking the minimum of $(P\times I1-I1,\ P\times I2-I2)$ for each population with respect to each reference single cross.

The superiority index (SX) used for the measure of superiority was calculated using the frequencies of alleles and their values and varied with type of gene action. For the non-epistatic cases (additive and dominant), let p_i^n = frequency of the i-th allele at the n-th locus in a particular population, v_i^n = value of the i-th allele at the n-th locus and γ_s^n = value of the most favorable allele in single cross s at the n-th locus. The superiority index with reference to single cross s (SX[s]) for this population was defined as $\sum_n \sum_{i:\, v_i^n > \gamma_s^n} p_i^n (v_i^n - \gamma_s^n)$. This is an indication of how much better an allele is than the best available in the elite, adapted reference single cross ($v_i^n - \gamma_s^n$) weighted by the probability, at a locus, of that particular allele occurring (p_i^n). When there are only two alleles per locus and if p_n = frequency of the favorable allele at the n-th locus, this index simplifies to:

$$\sum_l p_l[u_l-(-u_l)]=2\sum_l p_l u_l,$$

where the set of l loci are those where the single cross is fixed for the unfavorable allele and u_l is half the difference between the values of the two homozygotes at the l-th locus. This is twice the superiority measure used by Gerloff and Smith (1988).

In the epistatic cases, let $p_i^{r,a}$ = frequency of the i-th allele at the a-th locus of the r-th interacting pair of loci in a particular

population and $v_i^{r,a}$ = value of the i-th allele at the a-th locus of the r-th interacting pair. When gene action is complementary, let $\gamma_s^{r,a}$ = value of the most favorable allele present in single cross s at the a-th locus of the r-th interacting pair. Let γ_s^r = minimum $(\gamma_s^{r,1}, \gamma_s^{r,2})$, where r,1 and r,2 are the interacting loci. This is a relative measure of the maximum value attainable in a single-cross s-derived inbred for the r-th interacting pair. With the type of complementary gene action defined, the maximum value of alleles at a locus determined the value of the locus, but the minimum value of the loci in an interacting pair determined the value of the pair. Then the SX (s) for a population was defined as:

$$\sum_r \Big[\sum_{i:\, v_i^{r,1} > \gamma_s^r} p_i^{r,1} \Big(\sum_{x:\, v_x^{r,2} > v_i^{r,1}} p_x^{r,2} \Big)(v_i^{r,1} - \gamma_s^r) + \sum_{x:\, v_x^{r,2} > \gamma_s^r} p_x^{r,2} \Big(\sum_{i:\, v_i^{r,1} \geq v_x^{r,2}} p_i^{r,1} \Big)(v_x^{r,2} - \gamma_s^r) \Big],$$

where i and x both range from 1 to the number of alleles. In the first term, for alleles at the first locus in the r-th pair with values greater than the best in the single cross, the difference between these values is multiplied by the frequency of that allele at the r,1 locus and also by the sum of the frequency of all alleles in the population at the second member of the pair (r,2) that have values greater than the value of the particular allele at the r,1 locus. This product of $p_i^{r,1}$ and $\sum_{x:\, v_x^{r,2} > v_i^{r,1}} p_x^{r,2}$ is the probability of the i-th allele at r,1 determining the value for the r-th interacting pair of loci for that population. Similarly, the second term involves sums over the superior alleles at the second locus and sums over alleles at the first locus that have values greater than or equal to the one at the second locus. The strict inequality $(x: v_x^{r,2} > v_i^{r,1})$ in the first term but not the second $(i: v_i^{r,1} \geq v_x^{r,2})$ ensured that the frequency of all allelic combinations superior to the best in the single cross were considered only once.

In duplicate gene action cases, the SX was analogous to that for complementary gene action but was changed to account for the fact that the value of an interacting pair of loci was determined by the value for the most favorable, rather than the least favorable, locus. So in this case, we let γ_s^r = maximum $(\gamma_s^{r,1}, \gamma_s^{r,2})$. The SX (s) was:

$$\sum_r \Big[\sum_{i:\, v_i^{r,1} > \gamma_s^r} p_i^{r,1} \Big(\sum_{x:\, v_x^{r,2} < v_i^{r,1}} p_x^{r,2} \Big)(v_i^{r,1} - \gamma_s^r) + \sum_{x:\, v_x^{r,2} > \gamma_s^r} p_x^{r,2} \Big(\sum_{i:\, v_i^{r,1} \leq v_x^{r,2}} p_i^{r,1} \Big)(v_x^{r,2} - \gamma_s^r) \Big].$$

The SX was calculated from the specified frequency of alleles in the population rather than from the frequency in the sample of gametes and thus was calculated without sampling error. Genetic variances were calculated according to Kempthorne (1969), also using the population rather than the sample frequencies. The statistics ISD, BCSD and UBND were calculated using entry means over the four replications.

The correlations of different types of testcrosses with the SX were done by using means of the entries over the four replications. Rank correlations were used because of the lack of fit to normality of the testcross and per se values as determined by examining the W-statistic of Shapiro and Wilk (1965). Approximate significance levels for the rank correlation coefficients were taken from Snedecor and Cochran (1980). Correlation coefficients were compared to determine if they were significantly different from each other by using a two-tailed test based on the test for differences among product-moment correlation coefficients (Steel and Torrie 1980). This test was considered an appropriate approximation to a randomization test because product-moment correlation coefficients were similar in magnitude and pattern to those for rank correlations, indicating that lack of normality was not a major problem. Rank correlations were calculated for the same performance criterion (per se or testcross mean, SD, UBND or SX) between dominant gene action and the three other types of gene action.

Table 3. Rank correlation coefficients for performance criteria between dominant and additive, complementary, and duplicate gene action

Dominant gene action performance criterion	Additive gene action	Complementary gene action	Duplicate gene action
per se	0.96[a]	0.93	0.98
TC(A)	0.57	0.98	0.95
TC(B)	0.66	0.96	0.90
TC(C)	0.69	0.94	0.97
TC(AB)	0.76	0.96	0.97
TC(AC)	0.74	0.98	0.97
TC(BC)	0.73	0.93	0.96
ISD(AB)	0.35	0.93	0.95
ISD(AC)	−0.03	0.94	−0.86
ISD(BC)	0.38	0.93	−0.75
BCSD(AB)	0.34	0.95	0.94
BCSD(AC)	−0.12	0.96	−0.82
BCSD(BC)	0.37	0.92	−0.73
UBND(AB)	0.48	0.94	0.93
UBND(AC)	0.53	0.89	0.92
UBND(BC)	0.68	0.93	0.96
SX(AB)	1.00	0.90	0.93
SX(AC)	1.00	0.85	0.95
SX(BC)	1.00	0.78	0.95

[a] Correlation coefficients with absolute values greater than 0.40 and 0.52 were significant at the 5% and 1% levels, respectively

All testcrosses are indicated by TC with the tester parent in parentheses (i.e., A, B, C, AB, AC and BC). Letters in parentheses after SDs (ISDs and BCSDs), UBNDs and SXs identify the reference single cross.

Results and discussion

Overall means were lowest for complementary, followed by additive, dominant and duplicate gene action. Rank correlations for per se and testcross means, UBNDs and SXs between dominant and the two types of epistatic gene action were highly significant ($P \leq 0.01$), ranging from 0.78–0.98 (Table 3). Correlations for the per se, testcrosses and UBND values between dominant and additive gene action were lower, although significant ($P \leq 0.05$). There was a perfect correlation between the SXs for additive and dominant gene action because they were defined to be equal in the non-epistatic cases. Correlations for the SDs (both inbred and backcross) between additive and dominant gene action were sometimes negative and never significantly different from zero. Correlations for SD(AB)s were significant and positive between dominant and both epistatic types of gene action. Correlations for SD(AC)s and SD(BC)s were significant and positive between dominant and complementary gene action cases, but significant and negative between the domi-

Table 4. Ranks for superiority indexes, per se and testcross means and test statistics in the dominant gene action case

Population	Superiority index with reference to			Per se	Testcross to						ISD with reference to			UBND with reference to		
	AB	AC	BC		A	B	C	AB	AC	BC	AB	AC	BC	AB	AC	BC
A1A	23	18	4	4	23	5	2	14	13	4	24	1	4	23	19	2
A1U	23	24	5	15	24	9	6	15	15	6	23	2	7	24	24	6
A2U	20	19	2	10	20	3	4	11	12	2	22	3	6	20	14	4
B1A	13	5	13	3	7	23	8	10	5	14	1	19	1	17	5	13
B1U	13	7	16	11	9	18	11	9	7	15	2	21	3	14	8	12
B2U	17	6	10	13	10	15	9	12	8	12	3	12	2	11	6	10
C1A	3	11	15	2	4	6	16	5	10	10	7	24	24	4	13	16
C1U	6	12	17	14	5	10	18	6	11	11	6	23	23	7	16	18
C2U	1	10	9	8	2	7	13	3	6	9	4	22	22	5	10	11
AB3A	9	17	19	12	13	14	22	17	19	18	14	20	21	10	21	22
AB3U	12	23	20	21	18	17	24	20	23	22	18	18	20	16	23	24
AB4U	19	21	21	23	22	20	20	24	24	21	20	8	17	22	18	20
AC3U	22	22	24	20	19	22	21	22	21	23	16	13	18	18	20	21
AC4A	16	13	21	16	14	21	17	18	17	20	8	16	13	15	15	17
AC4U	18	16	23	24	17	24	23	21	22	24	11	17	19	19	22	23
BC3U	21	20	18	22	21	19	18	23	20	19	21	5	15	21	16	18
BC4U	10	15	11	19	16	16	15	19	18	17	15	11	14	13	12	15
M5A	3	1	6	1	1	4	3	2	2	5	5	15	5	3	2	3
M5U	6	4	7	7	8	8	7	7	4	7	9	14	11	6	4	7
T5U	8	8	8	17	11	11	10	8	9	8	19	7	12	8	7	8
M6U	3	3	3	5	6	2	5	4	3	3	12	6	9	2	3	5
T6U	2	2	1	6	3	1	1	1	1	1	13	4	8	1	1	1
X7A	11	9	12	9	12	12	12	13	14	13	10	9	10	9	9	9
X7U	15	14	14	18	15	13	14	16	16	16	17	10	16	12	11	14

nant and duplicate cases. The differences between the inbred parents (I1 and I2) and backcrosses (BC1 and BC2) were of different sign for single crosses AC and BC in the dominant as opposed to the duplicate cases. Because testcrosses were positively correlated over the two types of gene action, the effect of changing sign on the scaling factor that appears in the denominator of the SDs was to produce a negative correlation for these traits. These results indicate that, in future studies of this type, the complementary gene action case need not be simulated because results would be expected to be similar for the dominant and complementary cases. This is in accordance with the findings of Gill (1965), who found similar results for selection under his dominant and complementary factor cases in a model with two alleles per locus.

When gene action was additive, rank of the population was the same no matter which tester or function of testcross information (SD or UBND) was used. This ranking was very close to the ranking based on per se means. Because of the high correlations for the indexes and testcrosses across all types of gene action, only the ranks for the various means and statistics for the dominant case are shown (Table 4). Product-moment correlations between ISDs and BCSDs within the same reference single cross ranged from 0.98–1.00. Therefore, only the ranks for the ISDs are included in Table 4. For dominant, complementary and duplicate types of gene action, the ranks of the populations when crossed to the various testers were reasonably consistent over testers except for populations when crossed to their related inbreds (A1A, A1U and A2U crossed to A; B1A, B1U and B2U crossed to B; C1A, C1U and C2U crossed to C). The change in rank was less when the populations were crossed to a single cross that had the related inbred as one parent.

There were no consistent average differences between unadapted versions of source populations (A1U, B1U and C1U) and the unadapted populations related to the same inbred (A2U, B2U and C2U, respectively). A2U was, in most instances, better-performing than A1U; C2U was usually better-performing than C1U. B2U and B1U were inconsistently ranked. Although these pairs of populations were related to the same inbreds, gene frequencies were not controlled to ensure that either the group 1 populations (i.e., A1U, B1U and C1U) or group 2 populations were superior.

Comparisons between groups 3 and 4 were also not consistent over testcrosses. Group 4 populations had the frequency of A_5 or A_6 equal to 0.8 at only one-fourth the loci where the respective single cross had none of the three best alleles. Group 3 had frequencies of 0.3 at half the same group of loci. All other frequencies being equal, one would expect group 4 populations to outperform

those in group 3. These groups did not have the same allelic frequencies at other loci, however. They had alleles A_1, A_2 or A_3 at other loci, with choice of alleles being independent between populations. It was not surprising that the effects of higher specified average frequency at a few loci should, in some cases, be obscured by the effects of inferior alleles at other loci even though, on the average, the populations would be expected to have approximately equal contributions to performance at most loci. Obviously, small differences at many loci can overshadow greater differences at a few loci. If the superiority index had not been used to objectively define the value of these populations, it would have been difficult to draw conclusions about their relative value.

No attempt was made in the conversion from multiple-allele to two-allele populations (M5U to T5U and M6U to T6U) to ensure that the average allelic effects remained the same or nearly so. No consistent results between these two pairs would therefore be expected. M5U was nearly always better-performing than T5U; M6U was nearly always worse than T6U. Because the M and their respective T versions were usually close in rank, it would seem that it might be unnecessary to simulate multiple alleles within a population in the future.

The X7 populations (X7A and X7U) were consistently near the median for all testcross and per se means and test statistics. Even though they had one of the two most favorable alleles at every trait locus and thus would be sources of the most favorable alleles available at those loci, these favorable alleles were in such low frequency (0.1) that they were not able to result in better than average performance for these populations. In the definition of the SX, differences in allelic values were weighted by frequency. This resulted in the X7 populations having SXs near the median, also. All testcross or per se measurements contain terms in their genotypic expectation that weight allelic values by their frequencies for at least some loci. If the superiority measure was changed with the intention of identifying populations that had the most favorable alleles regardless of their frequency, no estimator of superiority based on performance data would be very highly correlated with this type of superiority measure.

Averaged over all pairs, differences in per se performance between adapted and their counterpart unadapted populations were more than twice as great as differences in testcross performance (Table 5). Unadapted and their corresponding adapted populations were closer in rank for testcross than per se performance in all but the additive case. This indicates that testcrossing can be effective in allowing evaluation of populations without unnecessarily penalizing them for their unadaptedness. For per se performance, duplicate gene action produced the greatest average difference between the two groups, followed by additive, dominant and complementary. The effect of the adaptive loci was greatest in the case of duplicate gene action because each adaptive locus interacted with two other loci, and the maximum value of a locus determined the value of the interacting pair. The adapted populations had more favorable alleles than those in unadapted populations at the adaptive loci. These alleles had the potential to determine the value of the interacting pair of loci for two pairs of loci when duplicate gene action existed, because they were often of higher value than the alleles at the trait loci with which they interacted. The more favorable adaptive alleles in adapted populations had the least effect in the complementary case because, in most of the populations used, trait loci had low frequencies of very favorable alleles. The least favorable locus determined the value of the interacting pair of loci for complementary gene action; very favorable alleles at the interspersed adaptive loci were unlikely to be expressed. In testcross performance, duplicate gene action again resulted in greater differences between adapted and unadapted populations than dominant gene action, which produced greater differences than complementary gene action. Additive gene action resulted in the greatest differences between the two groups in testcross performance, however. There was some masking effect of the tester in all except the additive case, which dampened the differences that would otherwise have been produced. There were six instances of an unadapted population having a higher testcross mean over four replications than its adapted counterpart. Four of these cases were when gene action was complementary and one each in the dominant and duplicate cases. Four of the situations involved population B1A and its unadapted counterpart B1U; A1A, A1U, C1A and C1U were involved in such situations only once each, in the complementary case. Because of the sampling involved in the simulation, this could

Table 5. Average differences between adapted populations and their unadapted counterparts for per se and testcross means and superiority indexes

Performance criterion	Gene action			
	Additive	Dominant	Complementary	Duplicate
Per se	31.44	30.95	22.77	39.13
TC(A)	15.59	4.13	2.06	6.20
TC(B)	15.59	1.94	0.24	3.63
TC(C)	15.59	3.89	1.60	6.19
TC(AB)	15.59	2.64	0.65	4.64
TC(AC)	15.59	3.85	1.46	6.19
TC(BC)	15.59	3.15	0.86	5.43
SX(AB)	0.14	0.14	1.83	0.27
SX(AC)	1.20	1.20	0.96	1.55
SX(BC)	0.39	0.39	1.30	0.71

occur due to chance if the sampled gametes from the unadapted population were superior at trait loci to those from the adapted population. In most cases, the effect of this superior set was unlikely to be large enough to offset the beneficial effects of more favorable alleles at adaptive loci in the adapted populations.

Many negative values of ISD and BCSD occurred. Under conditions of the appropriate model of two alleles per locus, equal gene effects, equal average frequencies at the j-th and k-th classes of loci and complete dominance, these statistics will be non-negative. Values above zero would indicate that the population can make a potential contribution to the reference single cross. That negative values occurred when the genetic model was extended to more than two alleles per locus, unequal allelic effects at each locus, and unequal allelic frequencies at each class of loci indicates that, under these conditions, at best they can be used to indicate relative merit of the populations screened. It is then of interest to note how these estimators of superiority of populations and other possible estimators compare with the superiority index defined. Rank correlations between all SXs, testcrosses, per se means and genetic variances were calculated; correlations between SXs and ISDs, BCSDs and UBNDs were calculated within a type of gene action only for the corresponding reference single cross. These correlations are, of course, genetic correlations because no environmental variable was added in the simulations. The correlations involving testcrosses, per se means or test statistics reflect some sampling during formation of gametes. The same set of single-cross tester gametes was used for all populations within a replication, which resulted in less sampling variation than in a biological system. Errors in estimation of genotypic value due to environmental and genotype × environment interaction effects would be expected to decrease the correlation between the SX and any per se or testcross mean or statistic calculated from field data.

When gene action was additive, per se performance, testcross performance, ISD, BCSD and UBND all had correlations with the SXs that were significant but, within a reference single cross, were equal or not significantly different from each other. The rank correlation coefficient (r_s) for these criteria and SX(AB) was 0.46. For SX(AC) and SX(BC), the rank correlation coefficients for per se performance were 0.58 and 0.63, respectively; for the testcrosses and statistics, r_s was equal to 0.59 for SX(AC) and 0.62 for SX(BC). For two of the three reference single crosses, the additive variance in the population was more highly correlated with the superiority index than was per se or testcross performance (Table 6). However, the differences in correlation coefficients were not significant at the 5% level. Given the greater difficulty in estimating genetic variances compared with estimating testcross or per se performance and the small increase in correlation obtained through the greater effort, per se or testcross performance would be more reasonable methods for screening superior populations than estimating the genetic variances in the populations.

The epistatic genetic variances were small relative to the total genetic variation in the complementary and duplicate cases (not shown). This is not surprising owing to the nature of the model used to fit the epistatic effects such that additive and dominant effects are fit before epistatic effects (Stuber and Moll 1971). Epistatic effects were exact opposites for these two types of gene action so that, when squared to determine the variances, the epistatic variances were equal for each population across the two types. Correlations between the dominance variance in the populations and the SXs were significant or highly significant for the cases of dominant and duplicate gene action. Correlation coefficients for additive variance and the SX in the dominant gene action case and additive × dominance and dominance × dominance variances in the duplicate case were also significant or highly signif-

Table 6. Rank correlation coefficients between superiority indexes and genetic variances for four types of gene action. Correlation coefficients greater than 0.40 and 0.52 were significant at the 5% and 1% levels of probability, respectively

Genetic variance	Gene action											
	Additive			Dominant			Complementary			Duplicate		
	Superiority index with reference to											
	AB	AC	BC	AB	AC	BC	AB	AC	BC	AB	AC	BC
Additive	0.54	0.81	0.63	0.40	0.66	0.42	0.14	0.67	0.54	0.58	0.69	0.22
Dominance	–[a]	–	–	0.42	0.68	0.54	0.18	0.71	0.46	0.52	0.71	0.62
Additive × additive	–	–	–	–	–	–	0.40	0.65	0.36	0.48	0.63	0.37
Additive × dominance	–	–	–	–	–	–	0.41	0.79	0.37	0.54	0.74	0.49
Dominance × dominance	–	–	–	–	–	–	0.26	0.74	0.38	0.42	0.61	0.64

[a] The corresponding genetic variance does not exist

icant. Other correlations between variances and SXs in the complementary and duplicate cases were significant for some, but not all, single crosses. Because of inconsistent correlations over all types of gene action and all single crosses and the difficulty in obtaining accurate estimates of the genetic variances in a population, other criteria for selecting populations should be chosen when screening for superior alleles.

For the three non-additive types of gene action, the correlations between SXs and BCSDs and ISDs were never significantly different from each other (Table 7). Neither the BCSD nor the ISD was consistently more highly correlated with the SX. More measurements go into the estimate of BCSD than ISD, each with an error associated with it under field conditions. The accuracy of the estimates of backcross performance (BC1 and BC2) must be greater than the accuracy of the estimates of inbred performance (I1 and I2) for the backcross method to be superior to the inbred method.

Of the correlations calculated between the SX and per se means, testcross means and test statistics for the dominant, complementary and duplicate gene action cases, the only non-significant ones occurred between some SXs and testcrosses to the inbred that was not a parent of the reference single cross, some ISDs and some BCSDs (Table 7). This indicates that these types of testcrosses and statistics would be poor choices as ranking criteria for choosing superior populations on the basis of the superiority index defined.

In the four types of gene action, there were only two situations where the testcross to a single cross was not also significantly or highly significantly correlated with the SX for the other two reference single crosses (TC [BC] with SX [AB] for dominant and complementary gene action). This was not surprising because a particular single cross in this study had one parent in common with the two other single crosses. Testcrosses to the non-parental inbred were significantly correlated with the superiority index in only one of the possible nine correlations for the three non-additive types of gene action. Testcrosses to different single crosses were highly significantly correlated with each other within a type of gene action (not shown). Testcrosses to the inbreds were highly significantly correlated in the additive and duplicate cases. In the dominant and complementary cases, only testcrosses to B and C were significantly or highly significantly correlated. For unrelated single crosses, we would not expect as high a correlation as observed between testcrosses to non-reference single crosses and the superiority index.

Testcrosses to the parental inbreds were significantly or highly significantly correlated with the superiority index. For the non-additive cases, crossing to one of the parental inbreds resulted in a correlation that was sometimes significantly lower than crossing to the other, but the choice of inbred that would produce the greater correlation was not obvious a priori. It was not consistently the lower nor the higher parent, nor was it the parent whose testcrosses had a higher or lower standard devia-

Table 7. Rank correlation coefficients between superiority indexes and per se and testcross means and test statistics. Correlation coefficients greater than 0.40 and 0.52 were significant at the 5% and 1% levels of probability, respectively

Performance criterion	Gene action								
	Dominant			Complementary			Duplicate		
	Superiority index relative to								
	AB	AC	BC	AB	AC	BC	AB	AC	BC
per se	0.50	0.66	0.65	0.59	0.75	0.62	0.61	0.74	0.66
TC(A)	0.89	0.86	0.27	0.84	0.63	−0.03	0.90	0.91	0.39
TC(B)	0.48	0.38	0.84	0.47	0.36	0.70	0.55	0.52	0.87
TC(C)	0.13	0.54	0.93	−0.04	0.62	0.80	0.40	0.65	0.92
TC(AB)	0.75	0.80	0.68	0.75	0.69	0.42	0.81	0.82	0.72
ISD(AB)	0.54	–[a]	–	0.44	–	–	0.64	–	–
BCSD(AB)	0.54	–	–	0.49	–	–	0.59	–	–
UBND(AB)	0.94	–	–	0.83	–	–	0.94	–	–
TC(AC)	0.61	0.88	0.73	0.62	0.86	0.45	0.71	0.88	0.76
ISD(AC)	–	−0.20	–	–	0.08	–	–	0.44	–
BCSD(AC)	–	−0.30	–	–	−0.01	–	–	0.64	–
UBND(AC)	–	0.93	–	–	0.79	–	–	0.97	–
TC(BC)	0.36	0.50	0.95	0.31	0.53	0.82	0.44	0.56	0.95
ISD(BC)	–	–	0.58	–	–	0.68	–	–	−0.18
BCSD(BC)	–	–	0.59	–	–	0.70	–	–	−0.15
UBND(BC)	–	–	0.94	–	–	0.82	–	–	0.92

[a] Correlation coefficients replaced by – were not calculated

tion over the sample of populations. Testcrosses to the reference single cross were always more highly correlated with the superiority index than testcrossing to at least one of the parents, sometimes significantly so. Correlation coefficients for the testcross to the single cross were sometimes higher and sometimes lower than the correlation coefficients for the testcross to the inbred parent that had the higher correlation coefficient with the SX. The differences between these correlation coefficients were never significant. TC(SC) and the UBND were always more highly correlated with the SX than was per se performance for these three types of gene action. When gene action was dominant or duplicate, correlation coefficients with SX for UBND were significantly different at the 1% or 5% levels from those for per se performance with reference to all single crosses. The correlation between SX(BC) and TC(BC) was significantly different at the 1% level from the correlation between SX(BC) and the per se mean for both dominant and duplicate gene action. When gene action was complementary, the correlation of the TC(SC) or the UBND with the SX was not significantly different from the per se mean correlation with the same SX.

Consistently high correlations occurred between the appropriate SX and the UBNDs and testcrosses to the reference single cross (TC[SC]s). Correlation coefficients for the UBNDs were usually higher than those for the testcross to the reference single cross. These differences were significant at the 5% level for duplicate gene action relative to AB and AC and for dominant gene action relative to AB. On the basis of no correlations between products of frequencies and effects at different loci over the populations screened, the TC(SC) should be more highly correlated with the SX in the case of dominant gene action (Gerloff and Smith 1988). In the set of populations specified, the relative variances for the TC(SC)s, UBNDs and SDs agreed with the predictions based on no correlations between loci. Positive covariances occurred between the sums of products of frequencies and effects for the various classes of loci in this sample of populations (i.e., $\sum_j p_j u_j$, $\sum_k p_k u_k$ and $\sum_l p_l u_l$). This resulted in covariances between SDs and the SX being greater than those between the UBND and the SX, which were greater than those between the TC(SC) and the SX. This increased the correlations between the SX and UBND relative to the SX and TC(SC) because the proportional increase in the covariance between the UBND and SX was usually greater than the proportional increase in the square root of the variance of the UBND. For the SDs, their variance was enough greater to result in lower product-moment (and rank) correlations between the SDs and SX as compared with the TC(SC) and the UBND. Further research could be done to evaluate the effects of these covariances. It is unclear what sorts of covariances might actually occur in real populations. The covariances between the terms $\sum_j p_j u_j$, $\sum_k p_k u_k$ and $\sum_l p_l u_l$ will be inestimable from field data for a sample of populations, where allelic frequencies and values are not known.

Linkage certainly will be involved for loci governing a quantitative trait. The populations simulated were in initial linkage equilibrium. If they had not been, unpredictable correlations between alleles at various loci would occur. Again, these correlations would not be estimable from field data. Their effect on actual screening of populations will be difficult to assess.

The correlations of the UBNDs and TC(SC)s with the SXs were equal in the additive case. Choice of either of these methods over the other would hence be of no benefit or harm if gene action is additive. Per se performance would be equally as good as testcross performance in the additive case as long as the populations could be evaluated accurately in the target environment and would have the benefit of not requiring the formation of testcross progenies. For grain yield in maize, estimates of level of dominance have been partial to complete (Robinson et al. 1958). As the level of dominance increases from none to complete, the superiority of the TC(SC) or UBND over per se testing for screening populations for superior alleles not already available in a particular elite single cross increases. Gene action affecting a quantitative trait is expected to involve many kinds of interactions. The loci affecting the trait very likely behave and interact in different ways. If the gene action is a mixture of the kinds simulated, the TC(SC) or the UBND would be favored over other methods because all methods were nearly equal in the additive case, and these two were more consistently highly correlated with the superiority measure than others in the dominant, complementary and duplicate cases.

Calculation of the UBND requires growing testcrosses to the two inbreds and the inbreds themselves. Testcrossing to the single cross requires growing one testcross to evaluate each population. There is more sampling variation when sampling gametes from a single cross than an inbred, but testing resources required for the UBND are at least doubled compared with testcrossing to the single cross. In a field situation where environmental variance may play a large role, this can be critical. Also, a single cross tester is expected to be a better pollen or seed parent than an inbred, which will facilitate making the testcrosses. Accurate estimation of inbred performance, also needed for calculation of the UBND, may be extremely difficult because of the low levels of vigor and potentially different interactions with fertility and density of inbreds compared with testcrosses. For these reasons, screening populations by testcrossing to the single cross would seem the most logical choice. However, testcrossing to the single cross as opposed to crossing to the

inbreds (as needed for calculating the UBND) provides no estimate of which heterotic group the population falls into. This information may be important to some breeders. The TC (SC) and UBND did not often have genetic correlation coefficients with the SX that were significantly different from each other. Choice between the TC (SC) and the UBND for identifying superior populations in the sense defined here will most likely be based on other considerations, such as efficiency in use of resources or other information provided by the screening methods.

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