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Effects of random and non-random errors on phenotypic selection in autotetraploids*

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Summary. A theoretical investigation was made to ascertain the effects of random and non-random deviations, called errors, of phenotypic from genotypic values on population means and on the response to phenotypic recurrent selection. The study was motivated as a selection experiment for disease resistance where there was either variability in the inoculation or environment (the random errors) or where the inoculation was above or below the the optimum rate where genetic differences in resistance are maximized (the non-random errors). The study was limited to the genetics at a diallelic locus (alleles B and b) in an autotetraploid population in random mating equilibrium. The response to selection was measured as the covariance of selection and compared to the exact covariance which was the covariance of selection without errors in phenotype. The random errors were modeled by assuming that a given percentage (α) of the population was uniformly distributed among the five possible genotype classes independent of their true genotypes. This model was analyzed numerically for a theoretical population with the frequency of the B allele (p) ranging from 0.0 to 1.0 and assumed errors of $\alpha = 0.1$ and 0.5 for the following six types of genic action of the B allele: additive, monoplex dominance, partial monoplex dominance, duplex dominance, partial duplex dominance, and recessive. The effect of random error was to consistently reduce the response to selection by α percentage independent of the type of genic action at the locus. The effect on the population mean was an upward bias when p was low and a downward bias when p approached unity. In the non-random error model below optimum inoculations altered the phenotypes by systematically including α percentage of susceptible genotypes into one or more other genotype classes with more genetic resistance (a positive shift). With above optimum inoculations, some resistant genotypes are classed with the non-resistant genotypes (a negative shift). The effects on the covariance of selection were found by numerical analysis for the same types of genic action and α's as investigated for random error. With a negative shift and a low p, the covariance of selection was always reduced, but for an increasing p the covariance approached and exceeded the exact covariance for all types of genic action except additive. With a positive shift and a low p, response to selection was greatly improved for three types of genic action: duplex dominance, partial duplex dominance, and recessive. The effect of a non-random error on population means was to greatly bias the means upwards for a low p and positive shift, but with increasing p the bias decreased. A relatively slight decrease in the mean occurred with a negative shift. This study indicated check varieties commonly used to monitor selection pressures in screening programs are very responsive to positive non-random shifts, but are relatively unresponsive to negative shifts. The interaction of selection pressure, types of genic action, and genotypes in the class shift models was suggested as a partial explanation for the lack of response to increasing selection pressures observed in some breeding programs.

Key words: Phenotypic recurrent selection – Selection response – Check varieties

Introduction

The objectives of recent theoretical genetic research on cross-pollinated autotetraploid species, such as alfalfa

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(*Medicago sativa* L.) have been to elucidate the differences in expected gains for various breeding schemes (Hill and Haag 1974; Rowe and Hill 1981) and to determine what effects the gametic disequilibrium (DSE) have on response to selection (Rowe 1982) and on mean genotypic values of synthetic varieties (Rowe and Hill 1984). This study expands the theoretical research on autotetraploids to include some problems encountered in evaluating and selecting populations for disease resistance and other traits. Though model development was made with reference to pest resistance, it may be equally applicable to other selection pressures in the environment, artificial or natural.

The precision of evaluations and effectiveness of screenings for resistance to diseases are dependent upon uniform exposure of each of the alfalfa plants to the disease causing organism at an inoculation rate in an environment which will allow expression of differences in levels of genetic resistance among plants. The researcher tries to attain uniform application of the inoculum through use of environmental controls and experimental designs. Despite these efforts, the patchiness of the inoculum or environment may be a persistent problem which leads to some clones being classified as resistant only because they were not exposed to the pest (the escapes), and symmetrically, other individuals appear susceptible which have genetic resistance but were exposed to excessive levels of inoculum.

Even when the inoculation is uniform for all plants, the rate is critical (Russell 1978). When inoculation rates are too low, plants and populations will have apparent resistances where there is little or no genetic resistance and when the dosage rate is too high, even those individuals with genetic resistance will appear susceptible. Thus the phenotype of a population or individual may not reflect its genotype due to a patchiness of inoculation or environment (a random error) or to a uniform inoculation at a rate which does not result in a one-to-one relationship of genotype to phenotype (a non-random error).

This theoretical research investigated the effects of random and non-random errors on population means and on rate of gain with phenotypic recurrent selection as measured by the covariance of selection. The covariance of selection with phenotypic recurrent selection is the covariance between the frequency of the allele controlling resistance (B) in a clone to its apparent genotypic value, the phenotype.

Calculations

Genotypes

This study was restricted to the genetics at a single, diallelic locus in an autotetraploid population. The frequencies of alleles B and b are p and q, respectively. Allele B was assumed to code for disease resistance while b did not. Random chromosome inheritance was assumed and frequencies of the alleles were assumed to change only in response to selection. The theoretical population in which evaluations and selections were to be made was assumed to be in random mating equilibrium. Thus, expected frequencies of the five genotypes at the B locus is given by expansion of the binomial $(p+q)$ to the fourth power (Table 1).

Models of resistance

Six types of genic action with monotone increasing levels of resistance for an increasing number of b alleles were investigated. The types of genic action were additive (ADD), monoplex dominance (MD), partial monoplex dominance (PMD), duplex dominance (DD), partial duplex dominance (PDD), and recessive (REC). The genotypic values of each genotype in each model of genic action are expressed in Table 2 in terms of the arbitrary constant indicating resistance "h".

Errors in phenotypes

The phenotype of an individual was its resistance score in either a screening or evaluation test. All scoring by a researcher was assumed to reflect the phenotype accurately. The deviation of phenotype from the genotype, for these modeling purposes, was assumed to be a random error caused by patchiness of inoculation or environment or to be a non-random error caused by consistently exposing plants to non-optimum rates of the disease causing factor. The optimum rate of inoculation was defined as that rate for which phenotype was identical to genotype.

Table 1. The genotypes, genotype class indicators, genotype frequencies, frequencies of B allele, and cross codes for genotypic values for calculation of means and covariance of selection

Genotype	Genotype class indicator i	Frequency of genotype f_i	Frequency of 'B' B_i	Genotypic value code V_i
BBBB	4	p^4	1	G4
BBBb	3	$4p^3q$	3/4	G3
BBbb	2	$6p^2q^2$	1/2	G2
Bbbb	1	$4pq^3$	1/4	G1
bbbb	0	q^4	0	G0

Table 2. The genotypic values and cross-reference codes for six models of genic action

Genotypes	Genotypic values (h)[a]						Cross-reference code
	ADD	MD	PMD	DD	PDD	REC	
BBBB	1	1	1	1	1	1	G4
BBBb	0.75	1	1	1	1	0	G3
BBbb	0.50	1	1	1	0.5	0	G2
Bbbb	0.25	1	0.5	0	0	0	G1
bbbb	0	0	0	0	0	0	G0

[a] h is an arbitrary constant of the trait

Thus, the individual phenotype was the sum of its genotypic value and a bias. A single type of random error and four types of non-random errors were investigated.

For the random error a subset of size αN, where α was a given percent and N was the number of individuals in the population, was uniformly distributed among the five genotype classes independent of their true genotypes. Thus, when $\alpha = 0.0$, there was no error, and when $\alpha = 1.0$ the genotypes were uniformly distributed independent of their true values. Thus the subset αN consisted of a constant proportion of each genotypic class, i.e. $\alpha N = \sum_{i=0}^{4} \alpha f_i N$ where f_i was the frequency of the i^{th} genotype of Table 1. The subset was randomly sampled with (1/5) of the subset assigned to each of the five genotypes. Thus, the apparent frequency of the i^{th} genotype was $f_i(1-\alpha)+(\alpha/5)$, see column f_{iRAND} of Table 3. The frequency of the i^{th} genotype in the subset αN was $f_i(\alpha N)$ of which (1/5) was expected to be assigned to the i^{th} genotype. Thus, the frequency of plants erroneously classified in the i^{th} genotype was $((4/5)f_i\alpha)/(f_i(1-\alpha)+\alpha/5)$ and the frequency of misclassified plants in the population was $(4\alpha)/5$. The frequency of B alleles in the i^{th} genotype class with random error was $[(if_i(1-\alpha)+4p(\alpha/5)]/[4(f_i(1-\alpha)+\alpha/5)]$ where i was the number of B alleles in the i^{th} genotype class in Table 1, see column B_{iRAND} of Table 3.

The non-random errors were viewed as the interaction of genotype and inoculation rate. When the inoculation rate was below optimum, some of the plants which were genetically susceptible were shifted into resistant genotype classes and the effect was named a positive class shift. When inoculation rate was above optimum the apparent resistance was lower than the level of genetic resistance and this was called a negative class shift. The class shifts, whether positive or negative, were assumed to be generated via an accumulative or non-accumulative mechanism as explained below.

With the non-accumulative class shifts individuals deviated by only one class from their correct genotype class. Thus, with positive non-accumulative class shift a given percentage, α, of the classes i = 0, 1, 2, and 3 was moved into classes i = 1, 2, 3, and 4, respectively (column $f'_{i(POS)}$ of Table 4). If the shift were negative, α of the classes i = 1, 2, 3, and 4 was put into classes i = 0, 1, 2, and 3, repectively (column $f'_{i(NEG)}$ of Table 4). When $\alpha = 0.0$, there was no class shift errors, and when $\alpha = 1.0$ with a positive shift, there were no plants in class 0 and each of classes i = 1, 2, 3 had the original frequencies of classes i = 0, 1, and 2, respectively, and the frequency of class 4 was $f_3 + f_4$. The percentage of clones classed erroneously in a population was $\alpha(1-f_4)$ with a positive shift and $\alpha(1-f_0)$ with a negative shift.

With accumulative class shift there was a progressive, directed shifting from one class into one or more classes. With a positive accumulative class shift the class i = 1 was increased by αf_0 and decreased by α percentage without regard to genotype, i.e. $\alpha(f_i + \alpha f_0)$. Thus, the apparent frequency of class i = 1 was $f_1 - \alpha f_1 + \alpha f_0 - \alpha^2 f_0$. Then class i = 2 has a frequency increased by αf_1 and $\alpha^2 f_0$ and decreased by α of $(f_2 + \alpha f_1 + \alpha^2 f_0)$ and the changes progress through the i = 4 (column $f''_{i(POS)}$, Table 5). In the population the percentage of plants improperly classified was the same as with the non-accumulative class shift. If the class shift were negative accumulative, the same progressive shifting described above would begin with i = 4 and continue towards i = 0 (column $f''_{i(NEG)}$ of Table 5). When $\alpha = 0.0$, the phenotype was the same as genotypic value. With positive accumulative shift and $\alpha = 1.0$, all clones were assigned to class i = 4, i.e. appeared resistant. With a negative accumulative shift and $\alpha = 1.0$, the entire population was in the susceptible class i = 0. The frequency of B alleles was found by multiplying each f_i term by i in the numberator of $f'_{i(POS)}$ (Table 5) and dividing by 4.

Population means, exact or biased, were calculated as $\sum_{i=0}^{4} f_i^a V_i$ where f_i^a is apparent frequency f_i, $f'_{i(POS)}$, $f'_{i(NEG)}$, $f''_{i(POS)}$, or $f''_{i(NEG)}$ of the i^{th} class as found in Table 1, 4, or 5, and V_i is the corresponding value for the i^{th} class for a specified type of genic action (Table 2).

Table 3. The class frequencies and frequencies of B allele in classes with random error in terms of f_i of Table 1 and percentage error (α)

Genotype class i	Genotypic value V_i	Class frequency f_{iRAND}	Frequency of 'B' B_{iRAND}
4	G4	$f_4(1-\alpha)+(\alpha/5)$	$[4f_4(1-\alpha)+4p(\alpha/5)]/4[f_4(1-\alpha)+(\alpha/5)]$
3	G3	$f_3(1-\alpha)+(\alpha/5)$	$[3f_3(1-\alpha)+4p(\alpha/5)]/4[f_3(1-\alpha)+(\alpha/5)]$
2	G2	$f_2(1-\alpha)+(\alpha/5)$	$[2f_2(1-\alpha)+4p(\alpha/5)]/4[f_2(1-\alpha)+(\alpha/5)]$
1	G1	$f_1(1-\alpha)+(\alpha/5)$	$[1f_1(1-\alpha)+4p(\alpha/5)]/4[f_1(1-\alpha)+(\alpha/5)]$
0	G0	$f_0(1-\alpha)+(\alpha/5)$	$4p(\alpha/5)]/4[f_0(1-\alpha)+(\alpha/5)]$

Table 4. Genotypic value codes, class frequencies, and frequencies of B allele for positive and negative non-accumulative class shift errors in terms of f_i from Table 1 and percentage shift (α)

Genotype class	Genotypic value code	Positive shift		Negative shift	
		Class frequency	Frequency of 'B'	Class frequency	Frequency of B
i	V_i	f'_{iPOS}	$B'_{i(POS)}$	f'_{iNEG}	$B'_{i(NEG)}$
4	G4	$f_4+\alpha f_3$	$[(4f_4+3\alpha f_3)/4]f'_{4\,(POS)}$	$f_4(1-\alpha)$	$[4f_4(1-\alpha)/4]f'_{4\,(NEG)}$
3	G3	$f_3(1-\alpha)+\alpha f_2$	$[[3f_3(1-\alpha)+2\alpha f_2]/4]f'_{3\,(POS)}$	$[f_3(1-\alpha)+\alpha f_4]$	$[[3f_3(1-\alpha)+4\alpha f_4]/4]f'_{3\,(NEG)}$
2	G2	$f_2(1-\alpha)+\alpha f_1$	$[[2f_2(1-\alpha)+\alpha f_1]/4]f'_{2\,(POS)}$	$[f_2(1-\alpha)+\alpha f_3]$	$[[2f_2(1-\alpha)+3\alpha f_3]/4]f'_{2\,(NEG)}$
1	G1	$f_1(1-\alpha)+\alpha f_0$	$[[f_1(1-\alpha)/4]f'_{1\,(POS)}$	$[f_1(1-\alpha)+\alpha f_2]$	$[[f_1(1-\alpha)+2\alpha f_2]/4]f'_{1\,(NEG)}$
0	G0	$f_0(1-\alpha)$	0	$f_0+\alpha f_1$	$(\alpha f_1/4)f'_{0\,(NEG)}$

Table. 5. Genotypic value codes, class frequencies, and frequencies of B allele for positive and negative accumulative class shift errors in terms of f_i from Table 1 and percentage shift (α)

Genotype class	Genotypic value code	Positive shift		Negative shift	
i	V_i	Class frequency $f''_{i(POS)}$	Frequency of 'B' $B''_{i(POS)}$	Class frequency $f''_{i(NEG)}$	Frequency of 'B' $B''_{i(NEG)}$
4	G4	$\sum_{i=0}^{4} \alpha^{4-i} f_i$	$\left(\left(\sum_{i=0}^{4} i\,\alpha^{(4-i)} f_i\right)/4\right) f''_{4(POS)}$	$f_4(1-\alpha)$	$(4 f_4(1-\alpha)/4)\, f''_{4(NEG)}$
3	G3	$\sum_{i=0}^{3} \alpha^{3-i} f_i(1-\alpha)$	$\left(\left(\sum_{i=0}^{3} i\,\alpha^{(3-i)} f_i(1-\alpha)\right)/4\right) f''_{3(POS)}$	$\sum_{i=3}^{4} \alpha^{(i-3)} f_i(1-\alpha)$	$\left(\left(\sum_{i=3}^{4} i\,\alpha^{(i-3)} f_i(1-\alpha)\right)/4\right) f''_{3(NEG)}$
2	G2	$\sum_{i=0}^{2} \alpha^{2-i} f_i(1-\alpha)$	$\left(\left(\sum_{i=0}^{2} i\,\alpha^{(2-i)} f_i(1-\alpha)\right)/4\right) f''_{2(POS)}$	$\sum_{i=2}^{4} \alpha^{(i-2)} f_i(1-\alpha)$	$\left(\left(\sum_{i=2}^{4} i\,\alpha^{(i-2)} f_i(1-\alpha)\right)/4\right) f''_{2(NEG)}$
1	G1	$\sum_{i=0}^{1} \alpha^{1-i} f_i(1-\alpha)$	$\left(\left(\sum_{i=0}^{1} i\,\alpha^{(1-i)} f_i(1-\alpha)\right)/4\right) f''_{1(POS)}$	$\sum_{i=1}^{4} \alpha^{(i-1)} f_i(1-\alpha)$	$\left(\left(\sum_{i-1}^{4} i\,\alpha^{(i-1)} f_i(1-\alpha)\right)/4\right) f''_{1(NEG)}$
0	G0	$f_0(1-\alpha)$	0	$\sum_{i=0}^{4} \alpha^{i} f_i$	$\left(\left(\sum_{i=0}^{4} i\,\alpha^{i} f_i\right)/4\right) f''_{0(NEG)}$

Covariance of selection

The genetic gain with selection was assumed to be proportional and constant to the covariance of selection (Falconer 1981) which is the covariance of the phenotypic value of observed units and the frequency of B alleles in corresponding selected units. The covariance of selection was calculated for a single cycle of phenotypic recurrent selection in which selections of individuals were based on their phenotypes, and the selections were randomly intercrossed to produce the improved population. The base population was always in RME. The errors in the phenotypes affected the frequency of B alleles in the selection. The calculation of covariance of selection with random error was made as follows with values from Table 3.

$$\mathrm{Cov}_{RAND} = \sum_{i=0}^{4} f_{iRAND} V_i B_{iRAND} - p\left(\sum_{i=0}^{4} f_{iRAND} V_i\right). \quad (1)$$

Where f_{iRAND} was the apparent frequency of i^{th} class, V_i was the genotypic value of the i^{th} class which was cross coded to a type of genic action found in Table 2, and B_i was the frequency of B alleles in the i^{th} class. The random error will bias the mean of the population, but does not change the frequency of allele B in the population, $\Sigma f_{iRAND} B_{iRAND} = p$.

The calculations of covariance of selection with non-accumulative (NONACC) positive shifts and accumulative (ACC) positive shifts were:

$$\mathrm{Cov}_{NONACC} = \sum_{i=0}^{4} f'_{i(POS)} V_i B'_{i(POS)} - p \sum_{i=0}^{4} f'_{i(POS)} V_i \quad (2)$$

$$\mathrm{Cov}_{ACC} = \sum_{i=0}^{4} f'_{i(POS)} V_i B''_{i(POS)} - p \sum_{i=0}^{4} f''_{i(POS)} V_i, \quad (3)$$

using values derived from Tables 4 and 5, respectively. The $f'_{i(POS)}$ was frequency of class i with non-accumulative positive shift, V_i was the phenotypic value of the i^{th} class, and $B'_{i(POS)}$ was the frequency of B alleles in the i^{th} class (Table 4). Similarly, for equation (3), $f''_{i(POS)}$ was the apparent frequency of the i^{th} class, V_i was the genotypic value of the i^{th} class, and $B'_{i(POS)}$ was frequency of B alleles in the i^{th} class (Table 5).

The calculation of covariance of selection with non-accumulative negative shifts or accumulative negative shifts was as follows from Tables 4 and 5, respectively:

$$\mathrm{Cov}_{NONACC} = \sum_{i=0}^{4} f'_{i(NEG)} V_i B'_{i(NEG)} - p \sum_{i=0}^{4} f'_{i(NEG)} V_i \quad (4)$$

$$\mathrm{Cov}_{ACC} = \sum_{i=0}^{4} f'_{i(NEG)} V_i B''_{i(NEG)} - p \sum_{i=0}^{4} f'_{i(NEG)} V_i \quad (5)$$

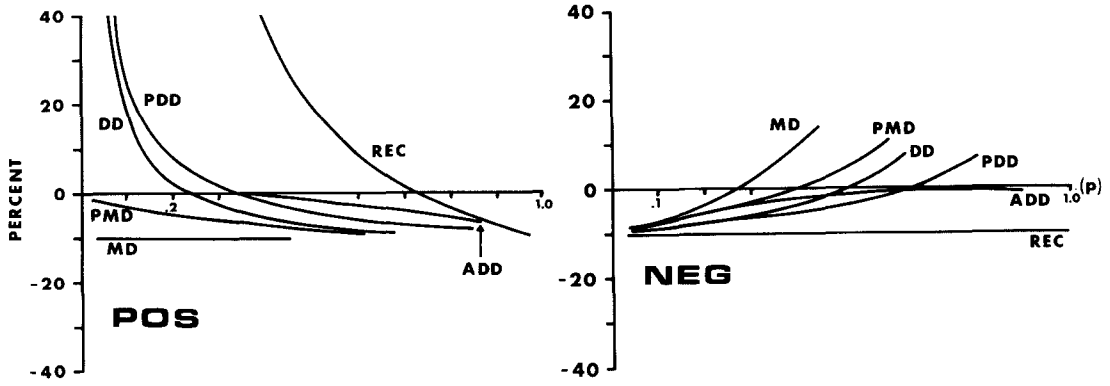

Fig. 1. Response curves of percentage bias of exact covariance of selection with $\alpha = 0.1$, six types of genic action, and positive (*POS*) or negative (*NEG*) class shifts

The terms for these equations were defined for negative shift as they were for positive shift with obvious referencing to columns of values for negative shifts (NEG).

Responses

The covariance calculated for each type of error (the error covariances) were compared to the exact covariance, i.e. $\alpha=0.0$. The effect of the errors on the covariance of selection was the difference, exact covariance minus error covariance, expressed as a percentage of the exact covariance. The biases in covariance caused by class shift errors appear as response curves in Figs. 1–3 for p values from 0 to the frequency where the base population had a mean genotypic value of 0.9 h. For the p values where the base population had resistance of 0.9 h to 1.0 h, the covariance was very small and often the effect of errors were large. In a practical sense, this region would not likely be of interest. Thus the error covariances were numerically compared to exact covariances for p values from near 0.0 to 0.9 for ADD, 0.45 for MD, 0.63 for PMD, 0.7 for DD, 0.8 for PDD, and 0.97 for REC with α percentage fixed at 0.1 and 0.5. At $\alpha=0.1$ the difference between accumulative and non-accumulative class shift errors was very slight and Fig. 1 was accurate for both types of class shift errors. The effects of random and non-random errors on the population means was the difference (correct mean minus biased means) expressed as a percentage of the correct mean (Table 6).

Results

The covariance of selection was affected by an interaction for all four variables considered in this study: error model, value of α, type of genic action, and allele frequency. The response curve plots across the p-axis indicated a different response curve for each type of genic action with a class shift error. With random error the covariance was a constant (1-α) percentage of the exact covariance independent of the type of genic action.

When there was a negative class shift, some symmetry appeared in the response curves associated with levels of dominance (Figs. 1–3). The genic models MD, PMD, DD, and PDD, which differ from each other by a sequential subtraction of 0.5 h from one genotype (Table 2) had similar response curves shifted along the p-axis. The ADD and REC genic models, which were modeled very differently, had unique response curves.

When there was a positive frame shift, the symmetry in response curves could no longer be associated with levels of dominance. The dissimilar genic models REC, DD, and PDD had similar response curves shifted along

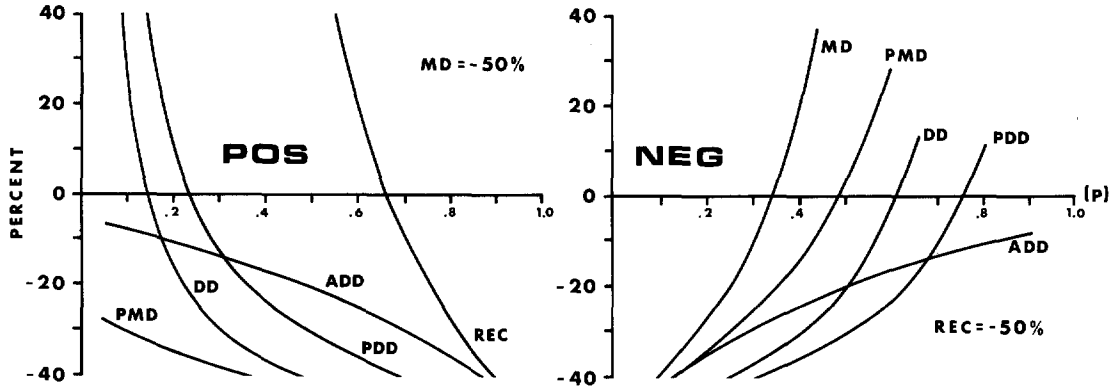

Fig. 2. Response curves of percentage bias of exact covariance of selection with $\alpha=0.5$, six types of genic action, and positive (*POS*) or negative (*NEG*) accumulative class shift

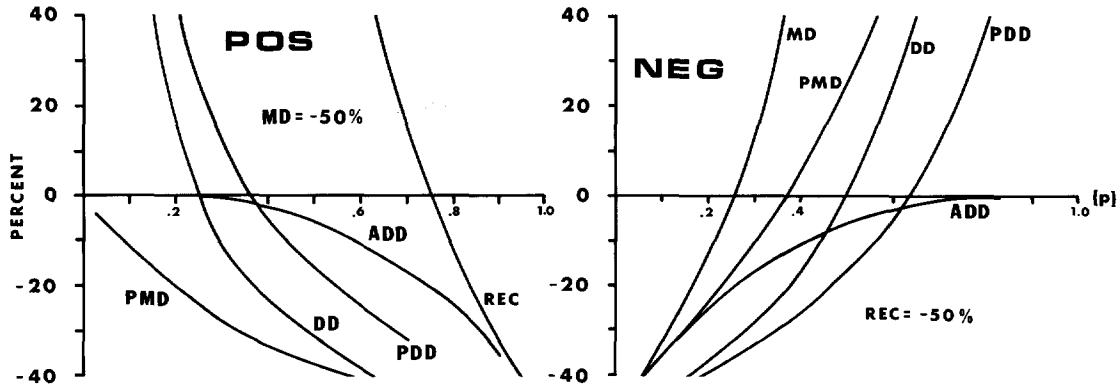

Fig. 3. Response curves of percentage bias of exact covariance of selection with $\alpha=0.5$, six types of genic action, and positive (*POS*) or negative (*NEG*) non-accumulative class shift

Table 6. Percent change in population means with error rate of $\alpha = 0.1$ and random error or accumulative class shift error calculated on base populations of 0.1 and 0.9 h for six types of genic action

Genic action	Accumulative class shift ($\alpha = 0.1$)				Random error ($\alpha = 0.1$)	
	Positive		Negative			
means (h) =	0.1	0.9	0.1	0.9	0.1	0.9
			Error (%)			
ADD	28	1	−9	−2	40	−5
MD	93	1	−16	−4	105	−1
PMD	54	1	−9	−3	60	−2
DD	38	1	−9	−4	47	−3
PDD	31	1	−9	−4	38	−4
REC	32	2	−10	−10	10	−8

the p-axis. The response curves for ADD, MD, and PMD were unique.

The results shown in the figures indicated that with a negative class shift in a base population with low resistance (low p), the covariance of selection will be reduced for every type of genic action. But with an increasing p in the base population, the error covariance eventually exceeds the exact covariance for MD, PMD, DD, and PDD genic action.

With a positive class shift the covariances of selection for REC, DD, and PDD were enormously increased for low p values, but were decreased below the exact covariance when p was increased. The covariance of selection with PMD and ADD genic action was near the exact covariance when p was low, but became negative with increasing p. The positive class shift effect on MD genic action was to reduce the covariance by a constant α percentage.

The effect of class shift error on the population mean was to bias it in the direction of the shift: a negative shift decreased the mean and a positive shift increased the mean. But with a random error the population mean was increased for a low p and decreased for a high p and at some frequency of p the random error had no effect on the base population mean (Table 6). In general, the effect of a random or non-random error was much greater for low values of p than for high values of p. This was particularly true with the positive class shift error. The population mean was much more responsive to a positive shift error than the negative shift error at the same α.

Discussion

Resistant and susceptible check varieties are commonly used to monitor the rate of inoculation in either an evaluation of, or selection for disease resistance. The results of this study indicated a highly asymmetrical response in the population means to non-random errors. The means of such check varieties will be very sensitive to inoculations at lower than optimum rates and comparatively insensitive to inoculations at higher than optimum rates. Inoculation at rates above the optimum (the negative class shift) reduces the covariance of selection in a population with low levels of resistance. Thus the practice of increasing the rate of inoculation to reduce the frequency of escapes could reduce the covariance of selection to the same level as it was with the escapes.

The two models of class shift error, accumulative and non-accumulative, differed little in predicted responses at low values of α, but had significant differences for large values of α. Conceptually, it is difficult to see how the accumulative class shift would occur in nature, but the non-accumulative produces illogical results. For instance, with a positive class shift and $\alpha = 1.0$, the non-accumulative error model has non-zero frequencies in all classes except $i = 0$. Thus in the situation of plants not exposed to the disease causing agent, the model predicts a portion of the population will show a susceptibility reaction except for MD genic action or the trivial case of $p = 0.0$. With the accumulative class shift this discrepancy does not occur because frequency of class $i = 4$ is 1.0. A similar illogical result occurs with negative non-accumulative class shift and $\alpha = 1.0$.

If the class shift model is extended to all loci affecting some trait and it is assumed that there are no epistatic effects among the loci, the interactions of alleles at every locus and selection pressure could be constant, and there would be a single selection pressure for expressing the genetic differences among plants and populations. However, if the interaction varied among the loci, then for varying selection pressures, genes at different loci would respond. Thus with increasing selection pressure there is a response, but the genetic factors effecting the response are expected to be different. With increasing selection pressure there will be a point where α will approach 1.0 and then none of the loci affecting a trait will be subjected to selection pressure. This phenomenon might be a partial explanation for the lack of response to intense selection pressure when there is a response to moderate selection pressure (reviewed in Chapter 2, Namkoong 1979).

This theoretical study does not predict responses to selection in a given breeding program but does indicate the complex interaction of some factors which might affect such a response. The errors in population means calculated from the simplistic random and non-random error models suggest a source of variation for experimental estimates based on population means, such as estimates of rate of gain with selection and realized

heritabilities, in addition to the sources of variation considered by Rowe (1982) and Rowe and Hill (1984).

Though the models of random and non-random errors were presented separately, a joint occurrence is easily visualized as the summation of effects of the non-random error followed by the random error. For the covariance of selection, the effects of the two errors are antagonistic if the effect of the non-random error was to increase the covariance but complementary if the non-random error had decreased the covariance, since the random error always reduces the covariance. From Table 6 it is apparent that the effects of simultaneous random and non-random errors on the phenotypic mean is dependent upon a complex interaction of every factor manipulated in this study: type of model, level of α, and type of genic action.

References

Falconer DS (1981) Introduction to quantitative genetics. Longman, New York

Hill RR Jr, Haag WL (1974) Comparison of selection methods for autotetraploids. 1. Theory. Crop Sci 14:587–590

Namkoong G (1979) Introduction to quantitative genetics in forestry. USDA Techn Bull 1588

Rowe DE (1982) Effect of gametic disequilibrium on selection in an autotetraploid population. Theor Appl Genet 64:69–74

Rowe DE, Hill RR Jr (1981) Inter-population improvement procedures for alfalfa. Crop Sci 21:392–397

Rowe DE, Hill RR Jr (1984) Effects of gametic disequilibrium on means and genetic variances of autotetraploid synthetic varieties. Theor Appl Genet 68:69–74

Russell GE (1978) Plant breeding for pest and disease resistance. Butterworths, London