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SELECTION AND ADAPTATION IN IRRADIATED PLANT AND ANIMAL POPULATIONS: A REVIEW

SELECTION ET ADAPTATION DES POPULATIONS ANIMALES ET VEGETALES IRRADIEES: UNE CRITIQUE

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RESUME

On examine la littérature existante relative aux effets du rayonnement ionisant sur les vitesses de mutation, la variabilité et les réactions d'adaptation à la sélection des populations animales et végétales exposées. La variabilité à l'état accumulé et, par conséquent, les différentiels de sélection possibles, peuvent être augmentés de nombreuses fois du fait de la mutation provoquée. La dose de rayonnement qui maximise la mutation provoquée varie beaucoup d'une espèce, d'une variété, ou d'un système génétique, à l'autre. La variabilité provoquée tend à augmenter la réaction à la sélection mais la réduction initiale de l'héritabilité de la variation provoquée peut retarder ou empêcher cet effet. Les effets nuisibles des mutations nocives chez les populations irradiées peuvent dépasser d'une façon importante les effets bénéfiques de la sélection en ce qui concerne les caractéristiques d'adaptation. La sélection pour la résistance au rayonnement peut se produire à des doses de rayonnement égales ou inférieures à la dose mortelle, mais les rapports entre les doses sont très variables.

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ABSTRACT

Available literature on the effects of ionizing radiation on mutation rates, variability and adaptive responses to selection in exposed plant and animal populations is reviewed. Accumulated variability, and hence potential selection differentials, may be increased by many times due to induced mutation. The radiation dose that maximizes induced mutation varies greatly among species, strains and genetic systems. Induced variability tends to enhance the response to selection, but this effect may be delayed or prevented by an initial reduction in the heritability of induced variation. Significantly, the detrimental effects of harmful mutations in irradiated populations may exceed the beneficial effects of selection for adaptive characteristics. Selection for radioresistance may occur at lethal or sub-lethal radiation doses but dose relationships are highly variable.

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CONTENTS

1. INTRODUCTION

The mutagenic properties of ionizing radiation have been studied intensively since Müller (1927) and Haldane (1937) first drew attention to the possible effects of radiation-induced mutation. Reviews of radiation genetics have concentrated upon the mechanisms of genetic damage (Wolff, 1967) and the frequency of mutation (Selby, 1977; Searle, 1974; Russell, 1965, 1972). However, the long-term significance of radiation in exposed populations has received less attention. The role of mutation in reducing individual reproductive potential and population mean fitness has been discussed in general reviews by Crow (1957), Crow and Abrahamson (1965) and Woodwell and Sparrow (1965), with particular reference to mammals by Green (1968) and Grahn (1972), and with reference to human populations by Newcombe (1962). These authors have dealt primarily with the ecological impact of lethal or disabling mutations rather than the evolutionary significance of viable mutations that increase population variability. Viable mutations are probably more common, but more difficult to detect, than obvious deleterious ones (BEIR, 1980). Radiation effects upon population variability were discussed briefly by Berry (1972) and may be of greater importance than has previously been recognized (IAEA, 1976). Their relationship to the process of natural selection in irradiated populations is discussed in reviews of population genetics by Dubinin (1964) and Dubinin, Shevchenko and Pomerantseva (1973). Very little experimental work on this aspect of population genetics has been done at the low radiation doses that have been predicted for natural populations, as a possible consequence of man's activities.

Adaptive responses to irradiation are of major interest in crop improvement programs where successive generations are exposed to induce desirable variability for selective propagation (IAEA, 1972). They are also of considerable interest in pest control programs where radiation is applied to successive generations to increase the incidence

of sterility and to reduce the reproductive potential of pest populations (IAEA, 1971). Finally, adaptation to long-term irradiation may be an important consideration in understanding populations and ecosystems exposed to above-normal dose rates (Odum, 1965). The importance of this consideration will depend upon the number of generations exposed, the increase in mutation rate and the strength of selective forces acting on the population (Woodwell and Sparrow, 1965). The present review examines available evidence on the effects of ionizing radiation on mutation rate, population variability and adaptive responses to selection over many generations.

2. RADIATION-INDUCED MUTATION

The genetic effects of ionizing radiation result from mutations in the germ cell chromosomes, which are passed on to successive generations in sexual reproduction. Therefore, long-term effects on a population's genetic composition depend on the relative rates at which mutations are induced and eliminated at specific genetic loci. Specific locus mutation rates can be determined precisely only for clearly defined characters of simple genetic origin, such as albinism in humans, coat colour in mice and eye colour in insects. Representative estimates of these rates suggest minor differences in the maximum rates that may be produced in different groups of organisms.

Most estimates of induced mutation at single loci have been derived from the study of laboratory mammals. Spontaneous rates in mice average about 1×10^{-5} mutations/locus (Searle, 1974; Russell, 1965, 1972). Russell (1965) induced mutation rates averaging 1.9×10^{-4} mutations/locus in female mice (*Mus musculus*) irradiated during oogenesis with X-ray doses of 400 R at 90 R/min. Fission neutrons produced average mutation rates of 1.2×10^{-4} mutations/locus after an acute dose of 60 rads at 75 rads/min (Russell, 1972). Mutation rates in male mice

averaged 1.3×10^{-4} mutations/locus after 600 R of X-rays during spermatogenesis (Russell, 1965) and 1×10^{-4} mutations/locus after 60 rads of fission neutrons (Russell, 1972). Higher acute doses are less effective, due apparently to killing of the mutant germ cells (Searle, 1974).

Greater rates of mutation, up to 7.3×10^{-4} mutations/locus, may be induced in male mice by fractionating large X-ray doses of 10^3 rads* into consecutive doses of 500 rads separated by a 24-hour time interval (Lyon and Morris, 1969). However, dose protraction over larger time intervals reduces mutagenic effectiveness by allowing time for significant repair (Searle, 1974). Chronic γ-radiation doses (0.009 R/min) up to 860 R in male and 400 R in female mice have produced average mutation rates no greater than 7×10^{-5} and 2×10^{-5} mutations/locus, respectively (Russell, 1965, 1972). Female mice apparently exceed males in their capacity for genetic repair at these low dose rates.

The single-locus mutation rates of insects are less well known than those of mammals. Generally, much larger radiation doses, on the order of 1 to 10 kR, are tolerated by insect populations and greater mutation rates can be induced, although fewer mutations/rad are produced. Kayhart (1956) irradiated wasps (*Mormoniella vitripennis*) during oogenesis with an acute X-ray dose of 5.7 kR (854 R/min) to produce eye colour mutants of five distinct types in the haploid male offspring. The resulting mutation rates, assuming that five loci were involved, averaged 2.2×10^{-3} mutations/locus as compared to a spontaneous rate of 7×10^{-5} mutations/locus. Baldwin and Cross (1966) produced eye colour mutants of four distinct types in the wasp, *Dahlbominus fuscipennis*. Assuming that four loci were involved, acute γ-radiation and neutron doses of 750 rads yielded 6.3×10^{-4} and 8.3×10^{-4} mutations/locus, respectively. Oster (1963) induced mutation rates of 5×10^{-3} mutations/locus in male fruit flies (*Drosophila melanogaster*) irradiated with 4 kR of γ-radiation (3 kR/min) prior to mating. Inagaki et al. (1977) irradiated male fruit flies with 1.5 kR of X-rays (115 R/min) during spermatogenesis and

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* 1 rad = 0.01 Gy = 0.97 R X- or γ-radiation (Holm and Berry, 1970)

found average rates ranging from 1×10^{-4} to 2.1×10^{-3} mutations/locus, depending upon the exact time of mating.

The single-locus mutation rates of plants are well known only for agricultural species which have been investigated in crop improvement studies. Chlorophyll mutations have been studied intensively. Investigators have recognized five mutation types which are controlled in barley (*Hordeum vulgare*) by recessive alleles at some 300 loci (Gustafsson, 1972). Aastveit (1968) irradiated barley plants, as embryos in the seed, with 10 kR of X-rays and counted chlorophyll mutations in their progeny. Homozygous mutants occurred with sufficient frequency to suggest an approximate mutation rate of 9.5×10^{-3} mutations/locus. Results after irradiation with 100 kR (Kawai, 1969) indicated a mutation rate of 1.2×10^{-2} mutations/locus. Spontaneous mutation rates at these loci (Doll and Sandfaer, 1969) are from one to three orders of magnitude lower. Rana and Swaminathan (1967) irradiated wheat (*Triticum aestivum*) seeds with 25 kR of X-rays; their results indicated a chlorophyll mutation rate of 5.7×10^{-3} mutations/locus, if the numbers of loci operating in wheat and barley are assumed to be the same. These estimates must be considered approximate since they are based upon rough estimations of the number of loci involved. Nevertheless, they do suggest that very high mutation rates may be induced in plants by irradiation of seeds with doses on the order of 10 to 100 kR.

Lower organisms, such as fungi and bacteria, are relatively insensitive to irradiation and yield even fewer mutations/rad than insects (Abrahamson et al., 1973). Mutability, expressed on a per rad basis, appears to decrease with the DNA content of the cell. However, increased radiation tolerance in lower organisms may compensate for this effect so that mutation rates approaching those of higher plants and animals can be induced. Webber and de Serres (1965) induced mutations for adenine dependence in the red bread mold (*Neurospora crassa*) by irradiating cultures with X-rays. Acute doses of 40 kR (1 kR/min) produced average rates of 1.2×10^{-4} mutations/locus. Demerec and Latarjet

(1946) irradiated bacteria (*Escherichia coli*) cultures with X-ray doses of 100 kR (2 kR/min) and induced mutations for T_1 phage resistance with a frequency of 2.0×10^{-4} mutations/locus. Survival is greatly reduced by such large radiation doses but reproductive potential is sufficient to permit rapid population recovery in successive generations.

Radiation-induced mutation rates are dose dependent and usually increase with radiation dosage until a maximum rate is reached. The optimum dose, at which this occurs, varies greatly among species, strains and different genetic loci (Venkateswarlu et al., 1978). In addition it depends upon the organism's age and physiological condition at exposure, the exposure rate, and the type of radiation used (Pawar, Thakare and Joshua, 1978). Dose dependence in plants has been reviewed by Nilan and Konzak (1961). Extensive reviews of dose dependence in animals include those of Searle (1974) and Russell (1972), and more recently the BEIR (1980) and UNSCEAR (1977) reports.

3. DIRECT EFFECTS OF MUTATION ON RESPONSE TO SELECTION

The direct effects of induced mutation on the genetic composition of a population may be understood by examination of genetic models incorporating the fundamental processes of evolution - mutation and selection. Mutation acts to transform genetic alleles in the germ cells from one form to another; the new alleles are passed on to the next generation where they are usually detrimental. Selective environmental forces act to eliminate the unfit individuals carrying and expressing detrimental alleles before they can reproduce and transmit those alleles to their offspring. The genetic composition of the population is therefore determined by the balance between these opposing forces, respectively generating and eliminating mutant alleles.

The proportion of each new generation lost due to the action of selective environmental forces upon mutant individuals is referred to as the population's genetic load. Recurring mutant alleles are normally maintained in the population at very low frequencies. However, when the environment changes, some of the mutant characteristics formerly contributing to genetic load will be favoured by new selective forces while other previously established alleles will become detrimental and decrease in frequency. Therefore, a certain amount of genetic load may be considered the necessary cost of evolutionary responsiveness (Crow and Abrahamson, 1965).

Simple models of genetic change, at a single locus with two possible alleles, have been developed by imposing recurrent mutation and selection on the Hardy-Weinburg equilibrium conditions which apply in the absence of these forces (Haldane, 1937; Li, 1961; Cavalli-Sforza and Bodmer, 1971). The precise form of the model depends upon the genetic behaviour of the alleles involved and the nature of the selective forces acting on them. New mutant alleles are usually recessive (detrimentally expressed only in homozygous carriers - genotype aa). However, some are dominant (detrimentally expressed in all carriers - genotypes aa and Aa) or heterotic (detrimentally expressed in aa homozygotes but advantageously expressed in Aa heterozygotes). The effects of hypothetical selective forces on the frequencies of alleles of each type are outlined in Table 1. The effects of selection may be balanced by those of mutation to produce a model of the following general form:

$$\Delta q = q - q' - \mu(1 - 2q) \qquad (1)$$

where Δq = decrease in relative frequency of allele a per generation,

q = relative frequency of allele a before selection,

q' = relative frequency of allele a after selection,

$= (q - sq^2)/(1 - sq^2)$ for recessive alleles,

$= q(1 - s)/(1 - 2sq + sq^2)$ for dominant alleles,

$= (q - sq^2)/(1 - 2sq^2 - s(1 - 2q))$ for heterotic alleles with equally detrimental homozygotes,

s = relative selection against individuals expressing detrimental characteristics ($0 \leq s \leq 1$),

μ = rate of mutation from one allele to the other .

The term representing mutation effects in equation (1) may be simplified to $\mu(1 - q)$ by ignoring reverse mutation rates (from a to A) when q is very small (Li, 1961) or modified to incorporate unequal rates of forward and reverse mutation (Falconer, 1960). Forward and reverse mutation rates are here assumed to be equal and mutation is assumed to act prior to selection.

The genetic response to selection pressure depends upon the initial allele frequency (Figure 1). There is no response at equilibrium ($\Delta q = 0$) when selective forces are exactly balanced by recurrent mutation. The allele frequency will deviate from equilibrium only when there has been a recent change in the equilibrium point due to a change in selection pressure or mutation rate. The frequency will then approach its new equilibrium value at a rate which depends upon the degree of displacement from equilibrium, the selection pressure and the mutation rate.

The mutation rate has a major influence upon the equilibrium value of the allele frequency only when dominant or recessive alleles are involved and equilibrium values in the absence of mutation are zero. It can be shown algebraically (Li, 1961; Cavalli-Sforza and Bodmer, 1971) or empirically (Figure 1) that the equilibrium values are approximately μ/s and $\sqrt{\mu/s}$ for dominant and recessive alleles respectively. The equilibrium frequency of a heterotic allele is 0.5, regardless of mutation rate, when both homozygotes are at the same selective disadvantage. With asymmetric selection pressures of s and t against aa and AA homozygotes respectively, the equilibrium frequency of allele a is ap-

proximately t/(s + t) and the influence of mutation is minor (Li, 1961; Berry, 1972).

Mutation has a significant direct effect upon the rate of genetic response to selection only when mutation rates approach or exceed 10^{-3} mutations/locus. The change in Δq as mutation rates drop to lower levels rapidly becomes negligible (Figure 1). The proportional effect of mutation, relative to that of selection alone, is greatest at low selection pressures and extreme allele frequencies. Mutation enhances the loss of detrimental alleles at very high frequencies, especially when those alleles are dominant. It retards the loss of detrimental alleles at very low frequencies, especially when those alleles are recessive. Since high recurrent mutation rates are required to produce these effects, it is unlikely that radiation-induced mutation at simple genetic loci would directly alter the response to selection at those loci in mammalian populations. However, such effects may occur in insect and seed plant populations when higher mutation rates are induced.

4. INDUCED VARIABILITY AND SELECTION

The direct effects of induced mutation on selection response, discussed above, must be distinguished from the indirect effects which occur as a result of accumulated variability in the population. An increased rate of mutation induces population variability by generating new alleles at each locus and maintaining them at sufficient equilibrium frequencies to prevent their accidental loss from the population. Accumulation of new alleles increases the potential range of character expression and of selective action (Berry, 1972). Although most new alleles are deleterious, the probability that one will become advantageous when environmental conditions change is maximized by a diverse system of multiple alleles. By maintaining substantial frequencies of potentially useful multiple alleles, mutation enables a finite popula-

tion to respond to stronger selective pressures, without extinction, than would otherwise be possible. However, the cost of maintaining this responsiveness is an increased number of mutant loci and an increased genetic load due to a continual loss of individuals carrying alleles that are not currently useful. The genetic load per mutant locus is approximately μ and 2μ for recessive and dominant mutant alleles respectively (Kimura and Ohta, 1971); it is s/x for each system of x mutually heterotic mutant alleles (Kimura and Crow, 1964).

The number of multiple alleles maintained at a locus depends upon the number of changes possible in its genetic structure, the likelihood of each change, and the adaptive significance of each change for the organism. Mutation affects each genetic locus differently. Nevertheless, simplifying assumptions allow some generalizations. Wright (1966) assumed that all new alleles were equally deleterious and demonstrated that very few dominant alleles can be maintained in a small finite population (Table 2). However, the number of possible alleles increased greatly with mutation at rates above 10^{-6} mutations/locus. A larger number of different recessive alleles was maintained, and the proportional increase in that number with mutation was slightly smaller. The smallest proportional effect of mutation occurred at heterotic loci, where very large numbers of alleles were normally maintained. However, the number was still very sensitive to mutation rates above 10^{-6} mutations/locus. Although fewer alleles can be maintained when their selective values differ from one another, the number will be strongly dependent on the mutation rate in most populations.

When induced mutation results in a greater range of character expression, new selective forces can act against a greater range of character values without driving the population to extinction. To the extent that survivors transmit their fitness characteristics to their offspring, this permits rapid evolutionary changes in the character of the population. Thus, changes in population mean may be expressed as a function of selection differential and heritability, as follows (Falconer, 1960; Crow and Kimura, 1970):

$$\Delta M = (M_{PR} - M_P)\, h^2 \qquad (2)$$

where M_P = mean character value of the parental population,

M_{PR} = mean character value of the parents that survive and reproduce,

$M_{PR} - M_P$ = selection differential,

h^2 = heritability ($0 \leq h^2 \leq 1$).

Heritability is usually determined experimentally as a regression coefficient describing the dependence of offspring character values on those of their parents; maternal and paternal parents are assigned average mid-parent character values (Ehrman and Parsons, 1976). The regression coefficient is usually positive, and may be used to define an "additive genetic" component (V_A) of population variance (V_P) such that $h^2 = V_A/V_P$. Heritability and additive genetic variance can also be estimated by analysis of variance between, and within, inbred lines.

Mutation effects upon accumulated population variability, and hence potential selection differential, are important both in artificial selection programs and in natural populations exposed to rapidly changing environmental conditions. These effects may be large, even at natural mutation rates, and are therefore of greater evolutionary significance than the direct effects of mutation on selection response. The latter become significant only at the highest possible rates of radiation-induced mutation.

5. VARIABILITY IN IRRADIATED POPULATIONS

Variability usually increases in irradiated populations as mutant alleles accumulate. In some populations the increase may be observed after a single generation of parental irradiation, while in others

it may take several generations to appear. Increased variability has most often been reported after acute ancestral exposure, but similar effects have been reported at chronic low doses.

Wallace (1958) measured the standard deviation of relative fitness for particular genotypes among the progeny of fruit flies (*Drosophila melanogaster*) whose male parents had been irradiated after emergence with 500 R of X-rays. The variability in fitness increased, by factors as high as 1.07, in flies of certain genotypes whose ancestors had been exposed to X-rays. In flies of other genotypes there was no significant increase in variability, indicating that the induction of population variability is non-random and subject to some degree of genetic control.

Ojomo and Chheda (1972) irradiated cowpeas (*Vicia unguiculata*) as embryos in the seed with 10 to 25 kR of X-rays and measured the yield variability of their progeny. In a "local brown" variety the standard deviation of the yield increased by a factor of 1.24, while in a "westbred" variety no increase was observed. The westbred variety was the product of a previous program of selection for yield and was much more variable than the local brown variety prior to irradiation. This suggests a limit to the amount of variability that can be induced in the population.

Gianola, Chapman and Rutledge (1977a) exposed the spermatogonia of laboratory rats to an X-ray dose of 450 R/generation for nine generations and measured the variability of weight gain in generation 16. The standard deviation of weight gain at 10 weeks of age had increased after ancestral irradiation by a factor of at least 1.6, and induced variability had persisted for seven generations in the absence of irradiation.

Trabalka and Allen (1977) examined variability in laboratory populations of mosquito fish (*Gambusia affinis*) whose parents had been captured from the waters of radionuclide-contaminated White Oak Lake in

Tennessee. The parents had received average dose rates between 0.059 and 0.120 R/day prior to mating, and the previous 60 generations had been exposed to even higher dose rates. The standard deviations of body length in females and critical thermal maximum in males were increased in ancestrally exposed fish by factors of 1.73 and 2.79, respectively. However, brood size variability was not increased significantly.

Krivolutskii, Smurov and Snetkov (1972) reported an increase in the morphological variability of centipedes (*Pachymerium ferrugineum*) collected from radionuclide-contaminated soils. Soil activity due to ^{90}Sr ranged from 0.6 to 3.4 $\mu Ci/m^2$* and variability was increased by a factor of 1.61 in males. Female variability was not increased relative to that in unexposed populations. Presumably, some of the induced variability in males was genetic and heritable, however, no attempt was made to distinguish genetic effects from somatic ones. This can often be done by allowing breeding stock from contaminated areas to reproduce in the laboratory at normal background levels.

Gorman, Kim and Taylor (1977) examined genetic polymorphism in a natural population of whiptail lizards (*Cnemidophorus tigris*) after ten years of exposure to γ-radiation at rates of 1 to 2 rads/day. Population variability was not increased by irradiation although accumulated doses were sufficient to produce sterility in the oldest females. However, significant changes in allele frequency were observed, suggesting that selective forces attributable to radiation may have been eliminating induced variability as the population responded to changes in selection pressure.

The degree to which irradiation increases population variability depends upon the characteristic considered and the underlying genetic mechanism by which it is determined. Changes in variability with irradiation may differ greatly between populations of the same species,

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* 1 Ci = 37 GBq

depending upon each population's evolutionary history and current selection pressures.

5.1 DOSE RELATIONSHIPS

Since mutation frequency is dose dependent, induced variability often depends predictably upon radiation dose. However, other factors which influence variability can complicate dose relationships. Changes in selection pressure with radiation stress may be particularly important at high doses. Dose relationships have usually been investigated at high doses, approaching those which maximize induced variability.

Kumar (1972) examined the dose relationships of induced variability in brown sarson (*Brassica campestris L.*). Variability in seed production was measured in the progeny of plants irradiated as embryos in the seed. The range in seed count/silique increased by a factor of three after a parental γ-radiation dose of 20 kR, the lowest dose applied (Figure 2). Lesser increases were observed at higher doses until variability approached normal control levels at 100 kR. The optimum dose which maximizes induced variability may be well below the dose that precludes population survival.

Seetharam (1976) induced variability in linseed plants by exposing their parents to γ-radiation as embryos in the seed. The range in plant height increased by a factor of 1.23 after a parental dose of 40 kR, the lowest dose applied (Figure 2). Variability returned to normal control levels at a dose of 80 kR. Variability in the number of tillers/plant exhibited a similar dose relationship, but the range in seed oil content increased with each increment in parental exposure and was maximum at 80 kR, the highest dose applied.

Bari (1971) irradiated flax plants (*Linum usitatissimum L.*) as embryos in the seed and measured the variability of seed production and growth in their progeny. The standard deviation of seed yield increased

by a factor of 1.26 after a parental X-ray dose of 25 kR (Figure 2). Lesser increases were observed at lower and higher doses. The highest parental dose of 150 kR substantially reduced the variability of seed yield in the progeny, relative to controls with no radiation history. The standard deviation of plant height was increased by parental radiation at all doses but a definite dose relationship was not apparent.

Kasim, Shamsi and Sofajy (1977) exposed broad bean plants to γ-radiation, as embryos in the seed, and examined progeny variability in the date of flowering. The standard deviation of flowering time in one variety increased by a factor of 3.93 after a parental dose of 500 R (Figure 2). Lesser increases were observed at lower and higher doses from 100 R to 1 kR. In two other varieties, although irradiation increased variability at all doses, dose relationships were poorly defined.

Daly (1973) induced variability in wall-cress plants (*Arabidopsis thaliana*) by γ-radiation of their parents as embryos in the seed. A direct linear relationship was observed between the standard deviation of flowering time and the parental radiation dose (Figure 2). Variability was increased by a factor of 3.07 at the highest applied dose 150 kR (410 R/h). Variability also increased with fast neutron radiation doses between 0 and 16 krad. Over this dose range, neutron radiation was more effective than γ-radiation for inducing population variability.

The diversity of dose relationships for the induction of expressed variability may be attributed to differences in the radiation sensitivity of the genetic systems controlling character expression. Differences in the dose dependence of mutation frequency are probably of primary importance. Additional differences in the degree to which radiation stress alters selection pressures may also be involved. There appears to be an optimum dose for maximizing induced variability in particular genetic systems, and this dose may be well below the maximum tolerated by the population. Finally, radiation effects on the heri-

tability of induced variation must be considered in order to predict effects on the population's responsiveness to new selection pressures.

5.2 HERITABILITY

Heritability represents the proportion of population variability which will be transmitted to and expressed by successive generations. Since some of the expressed variability in any population is non-genetic, heritability is usually much less than one (Falconer, 1960). Dominance relationships between alleles also reduce heritability, in a manner which depends on the relative frequencies of dominant and recessive alleles (Ehrman and Parsons, 1976). In a simple two-allele genetic system without dominance, maximum heritability is expected when allele frequencies are equal, at 0.5. The reduction in heritability with dominance of one allele over the other is greatest when recessive alleles are present at high frequency.

Gianola, Chapman and Rutledge (1977b) found a decrease in the heritability of weight gain in rats whose ancestors had been exposed to X-rays, even though the standard deviation of weight gain had increased. The net result was a decrease in the predicted ability of the ancestrally irradiated population to respond to new selection pressures. The reduction in heritability was not due to an increase in the proportion of population variability attributable to somatic radiation damage, since only distant ancestors had been irradiated. However, genetic instability may accompany the induction of new variability so that genetic variation is not reliably transmitted to the offspring. Alternatively, heritability may be reduced by radiation-induced changes in allele frequency, away from the values that maximize additive genetic variance.

Ojomo and Chheda (1972) examined the heritability and variability of seed yield in cowpea plants (*Vicia unguiculata*) after parental exposure to X-rays. In one variety, progeny variability was increased by ancestral seed doses of 10 to 25 kR while in another variety varia-

bility was decreased by similar doses. Heritability was increased in the progeny with increased variability, and decreased in progeny of the other variety. Daly (1973) reported a similar direct relationship between the heritability and variability of flowering time in *Arabidopsis thaliana* after ancestral irradiation with either neutrons or X-rays. These results are contrary to the findings of Gianola, Chapman and Rutledge (1977b) in rats, and suggest that general relationships between heritability and induced variability should not be expected. Both quantities must be determined experimentally in order to predict the response to selection in irradiated populations.

6. SELECTION IN IRRADIATED POPULATIONS

Natural selective forces acting on a population are usually poorly defined; artificial selective forces in experimental populations can be quantified more accurately. Gianola, Chapman and Rutledge (1979) subjected laboratory rat populations to ten generations of selection for either increased or decreased body weight. Rats in experimental populations were given X-ray doses of 450 R/generation at ages of 10 to 14 weeks, while control populations were exposed only to natural background radiation. The decrease in body weight with negative selection was more rapid in irradiated populations by factors ranging from 1.4 to 2.0 in each generation. These populations were more variable and tolerated larger selection differentials than did the control populations. However, the increase in body weight with positive selection pressure was no greater in irradiated populations than in controls, in spite of greater selection differentials. This was attributed to a reduced heritability of new variation induced at the upper end of the size range. Heritability varies with allele frequencies and may either increase or decrease as selection proceeds.

Kumar (1972) subjected brown sarson (*Brassica campestris L.*) populations to several generations of selection for increased seed yield. Seeds in experimental populations were given γ-radiation doses ranging from 20 to 100 kR/generation, prior to and during selection. In spite of an initial drop in seed yield due to dominant lethal effects on irradiated embryos, the increase in yield with selection was more rapid in irradiated populations than in controls, by an average factor of 1.34. Most of the increase in yield occurred during the first generation of selection. Since variability continued to increase in later generations, a reduction in the heritability of yield variation was implied. Selection for early flowering produced similar results and invited similar conclusions.

Scossiroli and Scossiroli (1959) subjected fruit fly (*Drosophila melanogaster*) populations to 11 generations of selection for an increased number of stenopleural hairs. Experimental populations were exposed to X-ray doses of 3 kR/generation. The increase in stenopleural hair count was greater in inbred and outbred experimental populations than in controls, by factors of 4.94 and 2.18, respectively. Irradiation enhanced the selection response by increasing heritable variability.

Ayala (1969) irradiated male fruit flies (*Drosophila birchii*) with X-ray doses of 2 kR/generation for three generations prior to selection. The experimental populations derived from these flies were then allowed to increase in size for approximately 40 generations, as were control populations. Natural selection pressures, due to intraspecific competition, presumably increased with population density. After 20 generations the ancestrally irradiated experimental populations became more productive than controls and began to increase more rapidly. The rate of increase was proportional to population variability and was 1.86 to 3.36 times greater in experimental populations than in controls. Similar experiments with *Drosophila serrata* (Ayala, 1966) produced similar results. Experimental populations with radiation histories responded more rapidly than controls to the selective pressures of compe-

tition, became more productive, and eventually stabilized at greater densities. Final densities in irradiated populations exceeded those in control populations by factors of 1.64 to 2.07. In *D. serrata*, as in *D. birchii*, several generations passed before the selection response of irradiated populations began to exceed that of controls. Several generations of genetic recombination may be required to incorporate induced variability into an integrated genetic system in a stable, heritable form (Dyer, 1971).

Crenshaw (1965) irradiated male flour beetles (*Tribolium confusum*) in highly inbred populations with 500 R of X-rays and examined productivity in three generations of progeny maintained under new, suboptimal conditions of temperature and relative humidity. Productivity was initially lowered by these conditions, but recovered after two generations. Recovery was greater in ancestrally irradiated populations than in controls by a factor of 1.97. This was attributed to a greater adaptive response made possible by induced variability. Wallace (1956) demonstrated that induced variability may enhance productivity even in the absence of selection by increasing the level of heterozygosity in heterotic systems. However, it is unlikely that such heterosis alone could account for the degree of improvement observed by Crenshaw.

Enhancement of the response to new selective forces, while commonly reported in irradiated populations, is not always observed. Womack and Bogart (1968) subjected populations of laboratory mice to five generations of selection for increased body weight at 28 days of age. Mice in experimental populations were given X-ray doses ranging from 25 to 100 R/generation (37 R/min) immediately prior to mating, while a control population was not irradiated. The increase in body weight was not significantly greater in irradiated populations, although heritable variability surpassed that of the control population. This was due to a decline in population size after irradiation and a consequent reduction in the selection pressure that could be tolerated without extinction. All populations were small, highly inbred and unable to

sustain the increase in genetic load that accompanied irradiation. Under these conditions, enhancement of the selection response cannot be expected.

Bartlett, Bell and Anderson (1966) subjected flour beetle (*Tribolium castaneum*) populations to 11 generations of selection for increased pupal weight. Adults in experimental populations were irradiated with X-ray doses of 100 R and 1 kR/generation. The increase in pupal weight was greater and more rapid in an unirradiated control population than in exposed populations. The smallest increase occurred in the population exposed to the highest dose, although heritable variability increased in proportion to the dose received. Low selection pressure in irradiated populations was responsible for the poor response. Selection pressure was intentionally reduced, when sterile matings occurred after several generations of irradiation, in order to prevent extinction of the small, inbred experimental populations. The results may be relevant to such populations, but are probably not typical of large natural populations exposed to the combined action of radiation and selection.

Abplanalp et al. (1964) exposed chicken semen to X-ray doses of 1 to 1.5 kR/generation for seven generations prior to selection for increased egg production. Production was initially reduced by irradiation as a result of increased genetic load. Normal production was restored after several generations of selection in the absence of irradiation, but further improvement occurred more slowly in the irradiated population than in the control. Variability was increased by irradiation, but this was offset by a loss of heritability at above-normal levels of production.

Increased variability enhances the selection response only when sufficient heritability is maintained. A reduction in the heritability of new variations may delay or prevent incorporation of adaptive characteristics by the population as a whole. Under strong selection pressure, such delay may be crucial in small populations which depend for their survival on rapid improvement.

7. RADIOADAPTATION

All studies of selection in irradiated populations are complicated by the fact that radiation itself acts as a selective force. Radiation-resistant individuals survive longer and reproduce more effectively than sensitive individuals exposed to damaging doses of radiation. To the extent that these individual differences are heritable, the radiation sensitivity of the population may be expected to decrease in response to continued exposure over many generations.

Cooley (1973) examined the productivity of the aquatic snail, *Physa heterostropha*, in a radionuclide-contaminated seep on the Oak Ridge Reservation. The dose rate was 0.65 rads/day but previous generations had received greater exposures. Egg production was greater in the exposed population than in a nearby control population. This was considered a possible adaptation to radiation-induced mortality. Mortality was not measured directly, but exposed snails were more heavily parasitized than controls.

Dishler and Rashal (1973) studied populations of wall-cress *Arabidopsis thaliana*, consisting of two different varieties. In unirradiated control populations the most fertile variety predominated. However, when seeds were exposed to γ-radiation doses of 30 kR prior to planting, the other variety became dominant. This variety was less fertile but its seeds were more resistant to γ-radiation.

Kaplan et al. (1972) counted meiotic chromosome aberrations in the germ cells of barley plants grown for several generations on soil contaminated with ^{90}Sr. The frequency of normal meiosis was initially decreased in proportion to ^{90}Sr concentration in the soil but returned to control levels after several generations. This may have been due to the selective elimination of radiosensitive genetic material from the

chromosomes, or to selective changes in the genetic system controlling meiotic processes.

Wozakowska-Natkaniec (1977) examined radiosensitivity in aquatic populations of duckweed (*Lemna minor*) from naturally radioactive water bodies in Poland. Activity levels ranged from 5 to 200 μCi*/L. Additional γ-radiation doses required to reduce population growth by 50% ranged from 8 to 10 kR in these populations. In other populations, growing in non-radioactive water, doses of 4 to 8 kR produced the same effect. Thus radioresistance was increased in radioactive water by factors as high as 2.5.

Kratz (1975) measured radiosensitivity in fruit fly (*Drosophila nebulosa*) populations from the Iron Hills region of Brazil. Experimental populations, derived from areas of high natural background radiation, had been exposed for many generations to dose rates from 1 to 1.5 mR/h. Adult survival, 72 hours after an additional γ-radiation exposure of 90 kR (230 R/min), was greater in these populations, by factors of 1.25 to 1.56, than in controls from normal background areas. The decrease in production of offspring after irradiation of males with 3 kR was greater in control populations, by a factor of 1.07, than in those from high background areas.

Cordeiro, Marques and Veiga-Neto (1973) measured radiosensitivity in populations of another species of fruit fly (*Drosophila willistoni*) from the Iron Hills region. The decrease in production of offspring after exposure of males to γ-radiation doses of 1 kR was greater in control populations than in those from high background areas, by a factor of 1.18. This was at least partly due to a greater reduction in the hatchability of the eggs produced after irradiation of the control populations.

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* 1 Ci = 37 GBq

Nöthel (1970) irradiated fruit flies (*Drosophila melanogaster*) with X-ray doses of 2.1 kR/generation for 227 generations. Radiosensitivity was determined in females exposed to 2.5 kR after one generation without exposure. Survival of their offspring to maturity was greater in females with radiation histories than in controls without ancestral irradiation. Ancestral exposure increased radioresistance by a factor of 1.9.

Nalborczyk, Zklawska and Kolakowska (1971) studied radiosensitivity in Scots pine populations from highland and lowland areas of Poland. Highland areas were exposed to greater levels of cosmic radiation than lowland areas. The median lethal X-ray dose (10 kR at 1 kR/min) required to prevent germination in 50% of seeds from highland populations was twice that determined in seeds from lowland populations. Doses of 1 kR were reported to stimulate seedling growth in highland plants and to inhibit growth in lowland plants. These differences were attributed to radioadaptation in highland areas. Other environmental factors related to altitude, in addition to cosmic radiation, may account for altitudinal differences in radioresistance, although these were not discussed.

Il'enko, Isaev and Ryabtsev (1974) studied radiosensitivity in voles (*Clethrionomys rutilus*) from radionuclide-contaminated areas in the U.S.S.R. Activity of ^{90}Sr in the soil averaged 1.2 $\mu Ci/m^2$. The median lethal dose of γ-radiation required to kill 50% of the population in 30 days was greater in populations from contaminated areas than in control populations, by a factor of 1.03. However, a similar increase in the radioresistance of wood mice (*Apodemus sylvaticus*) from contaminated areas was not observed. The difference was attributed to a greater consumption of contaminated lichens by voles. Radioadaptation of vole populations was suggested, but radiostimulation of repair mechanisms in individuals may have been involved.

Radioadaptation may take many forms. Birth rates may be increased by improvement in fertility, embryonic survival, litter size or

litter frequency. Alternatively, survival of irradiated adults may be improved. These evolutionary strategies are not mutually exclusive, but one or the other generally predominates.

7.1 TEMPORAL PATTERNS

The rate of radioadaption depends upon the characteristics of the genetic system controlling radioresistance, as well as the selection pressure. Improvement of radioresistance may be observed after a single generation of irradiation, or it may be delayed for many generations, depending upon the amount of variability inherent in the system and the rate at which new heritable variability is induced.

Stahler (1971) irradiated mosquito (*Aedes aegypti*) populations with γ-radiation doses of 2 kR/generation (125-250 R/min) to the eggs. Radiation resistance in each generation was determined from egg hatchability, before and after irradiation. Hatchability after irradiation was initially less than 9% of the control value before irradiation; after 90 generations it was 71% of the control value. Radioresistance was increased in a stepwise fashion by a final factor of 8.07. The stepwise pattern of increase was characterized by an intermediate plateau phase, during which radioresistance did not improve. This was attributed to the exhaustion of previously existing variability in radioresistance. Further improvement became possible only after newly induced variability had been incorporated into the genetic system in a stable, heritable fashion.

Zhestyanikov (1963) followed the change in radioresistance of bacterial (*Escherichia coli*) populations exposed to continuous γ-radiation. The acute lethal dose required to kill 90% of the population in 24 hours was determined at monthly intervals. After 22 months of exposure to daily doses of 1.8 to 2.3 R, the lethal dose was increased by a factor of 2.14. The stepwise pattern of increase in radioresistance was characterized by an intermediate plateau phase, similar to that observed

by Stahler in mosquito populations. This response pattern may be considered typical of variable populations in which additional variation can still be induced. However, if the genetic system has been depleted of mutable alleles, improvement will cease once existing variability has been exhausted. In a strain of *E. coli* previously selected for radioresistance, only a single step of improvement was observed in response to renewed selection pressure.

Zhorno and Surikova (1967) followed the change in radioresistance of laboratory mice exposed to daily γ-radiation doses of 20 mR for six generations. Spermatogenesis was examined in males after an acute X-ray dose of 600 R. The proportion of seminiferous tubules containing spermatogonia 30 days after acute exposure was increased by ancestral irradiation. However, the increase in radioresistance approached a maximal level after one generation of ancestral irradiation. There was little subsequent improvement once existing variability in radioresistance had been exhausted.

Emery (1972) followed the change in radioresistance of previously irradiated peanut (*Arachis hypogaea L.*) populations exposed to additional γ-radiation for several generations. Seeds in alternate generations were exposed to doses of 15 kR (1-6.8 R/min) and survival to maturity was determined for the seeds of the next generation. Survival was increased to maximal levels after two generations of treatment. Improvement apparently ceased to occur once existing variability in radioresistance had been exhausted. A subsequent decrease in survival was attributed to a reduction in population size and an increased genetic load due to inbreeding.

Populations characterized by a large amount of inherent variability in radioresistance will respond to irradiation with a rapid increase in radiation tolerance. In other, relatively invariable populations, the response will depend upon the accumulation of new heritable

variability. This may result in a stepwise pattern of improvement as variability is first exhausted and then renewed.

7.2 DOSE RELATIONSHIPS

Increased radioresistance with acute ancestral radiation exposure is usually found to be dose dependent. Since selection pressure increases with dose, the adaptive response of the exposed population may be expected to exhibit a similar increase over some dose range. Platonova and Sakharov (1962) examined this dose relationship in buckwheat (*Fagopyrum esculentum*) populations exposed to γ-radiation. Seeds were exposed to doses ranging from 10 to 50 kR (449-600 R/min) for four generations and survival to maturity was determined. Plant survival improved in successive generations at all doses above 10 kR. Improvement was proportional to dose, and survival after four generations of exposure at 50 kR exceeded that of the first exposed generation by a factor of 13.3. Tetraploid populations of the same species adapted in the same way, but were initially more resistant to radiation, and improved more slowly (Figure 3).

The increase in radioresistance with chronic ancestral irradiation usually depends on the dose rate. Eriksen and Emborg (1978) examined the radiosensitivity of soil microflora in a strawberry field irradiated with γ-radiation. After 18 months of continuous irradiation, soil samples were collected at various distances from the ^{60}Co source and exposed to an acute dose of 4000 krads. The density of surviving bacteria was reduced by acute exposure in inverse proportion to the preliminary dose rate. Bacterial radioresistance was increased at least 30 times by the highest preliminary dose rate of 180 rads/h. Highly resistant strains isolated from the γ-field suggested that radioadaptation of particular species had occurred, although changes in the species composition of the microflora probably contributed to its overall resistance.

Kiselev et al. (1961) isolated various bacterial species from naturally radioactive mineral waters in the U.S.S.R. and determined their sensitivity to X-rays. The median dose required to kill 50% of the culture increased in all species with the activity level of the water. The greatest increase in radioresistance was observed in *Bacillus adhaerens* which was 11 times more resistant when from waters with background dose rates of 0.12 mR/h than from waters with background dose rates of 0.03 mR/h. Radioadaptation occurred in *Micrococcus sulfureus* exposed to background dose rates as low as 0.03 mR/h (Figure 3).

Cherezhanova, Aleksakhin and Smirnov (1971) examined radiosensitivity in populations of herbaceous plant species grown for 11 years in experimental plots contaminated with ^{90}Sr. Soil activity varied from 1.0 to 3.7 $\mu Ci/m^2$. The frequency of normal cell division in the leaf meristem was determined after subjecting plants to an additional activity of 1.5 Ci for 24 hours. Resistance to disruption of cell division was increased by ancestral exposure in contaminated plots, in proportion to the soil activity. Radioresistance was increased by a factor of 1.25 at the highest activity level in common agrimony (*Agrimonia eupatoria L.*) and by smaller factors in the other species examined (Figure 3). However, radioresistance was not conclusively shown to increase from one irradiated generation to the next, and individual radiostimulation, rather than adaptation, may acccount for much of the observed improvement.

The direct relationship between preliminary dose or dose rate and radioadaptation is commonly, but not consistently, observed. Shevchenko (1970) examined radiosensitivity in algae (*Chlorella* sp.) isolated from radionuclide-contaminated soils in the U.S.S.R. Soil activity, due to ^{90}Sr and ^{90}Y, ranged from 0.5×10^{-3} to 4.5 $\mu Ci/g$. The median lethal dose of γ-radiation was determined in the laboratory for algae from contaminated and control plots. Radioresistance was 1.5 times greater after five years at a soil activity level of 0.05 $\mu Ci/g$ than in control plots. Lesser increases occurred at lower and higher soil activities, suggesting an optimum activity level for radioadaptation (Figure 3). As

radioresistance increased in later years, the optimum activity level appeared to decrease.

Maisin et al. (1960) irradiated ten successively derived yeast (*Saccharomyces cerevisiae*) colonies with preliminary X-ray doses ranging from 5 R to 10 kR and measured survival in the final colony after an additional dose of 200 kR. Survival was increased by preliminary exposure at all doses, but was maximized by small doses of 74 R. Since all preliminary doses were sub-lethal, radioadaptation must be attributed to differential effects of radiation on the productivity of sensitive and resistant strains. Resistance persisted in subsequent, unirradiated generations.

Marques (1973) irradiated fruit-fly (*Drosophila nebulosa*) populations and measured their ability to compete with unirradiated *D. willistoni*. Preliminary γ-radiation for 14 generations was followed by doses of 1 kR/generation (71-100 R/min) during competition. The competitively superior *D. nebulosa* always replaced *D. willistoni* and the replacement time was used as a measure of its radiosensitivity. Replacement time was increased by irradiation during competition but this effect was ameliorated by preliminary irradiation. Preliminary doses of 500 R/generation minimized the effect of later doses and induced a greater radioresistance than higher preliminary doses of 1 kR/generation. While high doses presumably increase selection pressure, the variability on which selection must act may be maximized at lower doses.

7.3 EFFECTS OF GENETIC LOAD

Radioadaptation often enhances the performance, under radiation stress, of a previously exposed population. However, ancestral exposure may increase genetic load to the point that net population performance under additional radiation is not improved relative to previously unexposed controls. Gol'zberg and Vorobtsova (1976) found this to be the case in an ancestrally irradiated fruit fly (*Drosophila melanogaster*)

population (2 kR/generation at 141 R/min) treated with an additional γ-radiation dose of 60 kR (2.6 kR/min). This dose reduced survival to a greater extent in a control population than in one which was previously exposed, but the net survival was highest in the control population. The authors considered their results illustrative of radioadaptation. However, similar results have been interpreted by other authors as evidence against an adaptive response.

Brower, Hossain and Tilton (1973) irradiated experimental populations of the rice weevil (*Sitophilus oryzae*) with γ-radiation doses up to 2 kR for five generations and measured adult life span in the next generation after additional exposures up to 50 kR (2.1 kR/min). Life span was reduced by the additional exposure, in inverse proportion to the ancestral dose. However, since ancestral irradiation alone had reduced the life span, the resulting longevity in experimental populations was slightly less than that in controls exposed for the first time. The authors concluded that radioadaptation had not occurred. This conclusion contradicts that of Gol'zberg and Vorobtsova (1976) although their results were very similar. The differences in interpretation are evidently semantic; the results of both studies indicated that the adaptive response, if it can be referred to as such, was insufficient to compensate for the increased genetic load in ancestrally irradiated populations. An equally limited adaptive response, based on adult survival, was reported by Abdul-Matin (1975) in irradiated populations of *S. oryzae*. Other species, such as the cowpea weevil (*Callosobruchus maculatus*) and the meal moth (*Plodia interpunctella*) showed no apparent adaptive response under similar experimental conditions (Hossain, Brower and Tilton, 1972a, 1972b).

Spalding, Strang and Le Stourgeon (1963) examined radiosensitivity in laboratory mouse populations with different radiation histories. One population received γ-radiation doses of 200 rads for 15 generations; a sub-population was irradiated for 10 generations and allowed to recover. The median lethal dose was determined in generation 17 after a recovery

period of one to six generations. The lethal dose in the first irradiated population was less than that in unirradiated controls while that in the sub-population exceeded the control value. The differences were insignificant, but suggested that improvement may become apparent only after selection is relaxed and genetic load eliminated from the population.

Brower (1974a) irradiated experimental populations of the rice weevil (*Sitophilus oryzae*) with γ-radiation for 25 generations and determined reproductive capacity in the next generation after an additional exposure of 2 kR. This dose reduced reproductive capacity to a greater extent in control populations than in experimental populations ancestrally exposed to 1 and 2 kR. However, since ancestral irradiation alone had increased genetic load and reduced reproductive capacity, the resulting productivity in experimental populations did not exceed that in controls exposed for the first time. Similar studies of productivity in irradiated populations of *Callosobruchus maculatus* and *Tribolium castaneum* (Brower 1974b, 1974c) provided no evidence of adaptive response. Reproductive radiosensitivity in these species is either invariable or under very weak genetic control.

Radiation exposure facilitates radioadaptation by inducing population variability in radioresistance and selecting the most resistant individuals to survive and reproduce. However, ancestral exposure also increases the proportion of survivors carrying genetic defects. Population mean fitness under continued exposure depends upon the relative magnitude of these effects, and may either increase or decrease.

8. SUMMARY

The direct effects of recurrent mutation on the response to a fixed selection pressure are minor although they may become significant in seed plants and insects at the highest reported rates of radiation-

induced mutation. However, long-term mutation effects upon accumulated population variability, and hence potential selection differentials, may be large, even at natural mutation rates. There appears to be an optimum radiation dose for maximizing induced variability, which varies greatly among species, strains and genetic systems, and may fall well below the maximum dose tolerated by the population. Effects of dose rate on population variability have not been investigated although increased variability has been reported at low doses and dose rates. Induced variability tends to enhance a population's response to selection but this effect may be delayed or prevented by an initial reduction in the heritability of induced variation. In irradiated populations, the detrimental effects of increased genetic load may exceed the beneficial effects of selection for adaptive characteristics.

Selection for radioresistance has been demonstrated following acute or chronic exposure over many generations, although some reports of this phenomenon fail to distinguish between the effects of selection on the population and those of individual radiostimulation. An optimum dose for maximizing radioadaptation is often apparent. However, the optimum dose and the degree of adaptation possible depend upon the dynamics and dose relationships of population variability, heritability and accumulated genetic load. These relationships must be understood, with particular reference to the genetic systems controlling radioresistance, in order to predict adaptive responses in irradiated populations.

REFERENCES

Aastveit, K. 1968. Effects of combinations of mutagens on mutation frequency in barley. In Mutations in Plant Breeding II, International Atomic Energy Agency, Vienna. pp. 5-14.

Abdul-Matin, A.S.M. 1975. On the possible development of radioresistance in *Sitophilus oryzae* (L.) following exposures of successive generations to ionizing radiation. International Journal of Applied Radiation and Isotopes 26:625-626.

Abplanalp, H., D.C. Lowry, I.M. Lerner and E.R. Dempster. 1964. Selection for egg number with X-ray induced variation. Genetics 50:1083-1100.

Abrahamson, S., M.A. Bender, A.D. Conger and S. Wolff. 1973. Uniformity of radiation-induced mutation rates among different species. Nature 245:460-462.

Ayala, F.J. 1966. Evolution of fitness. I. Improvement in the productivity and size of irradiated populations of *Drosophila serrata* and *Drosophila birchii*. Genetics 53:833-895.

Ayala, F.J. 1969. Evolution of fitness. V. Rate of evolution of irradiated populations of *Drosophila*. Proceedings of the National Academy of Science (U.S.A.) 63:790-793.

Baldwin, W.F. and W.G. Cross. 1966. Effects of fast neutrons on eye colour mutations in *Dahlbominus*. Nature 210:1396-1397.

Bari, G. 1971. Effects of chronic and acute irradiation on morphological characters and seed yield in flax. Radiation Botany 11:293-302.

Bartlett, A.C., A.E. Bell and V.L. Anderson. 1966. Changes in quantitative traits of *Tribolium* under irradiation and selection. Genetics 54:699-713.

BEIR. 1980. The Effects on Populations of Exposure to Low Levels of Ionizing Radiation. Biological Effects of Ionizing Radiation, Report III. National Academy of Sciences, National Research Council, Washington, D.C.

Berry, R.J. 1972. Genetical effects of radiation on populations. Atomic Energy Review 10:67-100.

Brower, J.H. 1974a. Fitness and radioresistance of the rice weevil, *Sitophilus oryzae* (Coleoptera:Curculionidae), after 25 generations of substerilizing irradiation. Journal of the Kansas Entomological Society 47:437-444.

Brower, J.H. 1974b. Inability of populations of *Callosobruchus maculatus* to develop tolerance to exposures of acute gamma irradiation. Annals of the Entomological Society of America 67:287-291.

Brower, J.H. 1974c. Radioresistance of the red flour beetle, *Tribolium castaneum* (Coleoptera:Tenebrionidae), exposed to sublethal doses of gamma irradiation for 25 generations. Canadian Entomologist 106:241-246.

Brower, J.H., M.M. Hossain and E.W. Tilton. 1973. Radiation sensitivity of successively irradiated generations of *Tribolium castaneum* (Herbst) (Coleoptera, Tenebrionidae) and *Sitophilus oryzae* (L.) (Coleoptera, Curculionidae). Journal of Stored Products Research 9:43-49.

Cavalli-Sforza, L.L. and W.F. Bodmer. 1971. The Genetics of Human Populations. Freeman, San Francisco.

Cherezhanova, L.V., R.M. Aleksakhin and E. G. Smirnov. 1971. Cytogenetic adaptation of plants under a chronic exposure to ionizing radiation. Genetika 7:30-37. (Soviet Genetics 7:437-442).

Cooley, J.L. 1973. Effects of chronic environmental radiation on a natural population of the aquatic snail *Physa heterostropha*. Radiation Research 54:130-140.

Cordeiro, A.R., E.K. Marques and A. Veiga-Neto. 1973. Radioresistance of a natural population of *Drosophila willistoni* living in a radioactive environment. Mutation Research 19:325-329.

Crenshaw, J.W. 1965. Radiation induced increases in fitness in the flour beetle *Tribolium confusum*. Science 149:426-427.

Crow, J.F. 1957. Possible consequences of an increased mutation rate. Eugenics Quarterly 4:67-80.

Crow, J.F. and S. Abrahamson. 1965. Genetic effects of ionizing radiation. Chap. 13 in The Science of Ionizing Radiation, L.E. Etter, ed., Charles C. Thomas Publ., Springfield, Ill.

Crow, J.F. and M. Kimura. 1970. An Introduction to Population Genetics Theory. Harper and Row, Publ., Inc., New York.

Daly, K. 1973. Quantitative variation induced by gamma rays and fast neutrons in *Arabidopsis thaliana*. Radiation Botany 13:149-154.

Demerec, M. and R. Latarjet. 1946. Mutations in bacteria induced by radiations. Cold Spring Harbor Symposium on Quantitative Biology 11:38-50.

Dishler, V.Ya. and I.D. Rashal. 1973. The process of adaptation and irradiated synthetic populations of *Arabidopsis thaliana*. I. Response of M_1 and M_2 plants to irradiation. Latvijas PSR Zinatnu Akademijas Vestis 7:24-28. (Russian with English summary).

Doll, H. and J. Sandfaer. 1969. Mutagenic effect of gamma rays, diethyl sulphate, ethyl methanesulphonate, and various combinations of gamma rays and the chemicals. In Induced Mutations in Plants, International Atomic Energy Agency, Vienna. pp. 195-206.

Dubinin, N.P. 1964. Problems of Radiation Genetics. Oliver and Boyd Ltd., Edinburgh.

Dubinin, N.P., V.A. Shevchenko and M.D. Pomerantseva. 1973. Effect of ionizing radiation on populations (radiation-genetic aspects). Chap. 8 in Radioecology, V.M. Klechkovskii, G.G. Polikarpov and R.M. Aleksakhin, eds., John Wiley and Sons Ltd., Chichester.

Dyer, K.F. 1971. Effects of radiation on populations of *Drosophila melanogaster* with different genetic structures. I. Changes in fitness and productivity. Australian Journal of Biological Sciences 24:565-573.

Ehrman, L. and P.A. Parsons. 1976. The Genetics of Behavior. Sinauer Associates Inc., Sunderland, Mass.

Emery, D.A. 1972. Effect of reirradiation on radioresistance in peanuts (*Arachis hypogaea* L.). Radiation Botany 12:137-150.

Eriksen, W.H. and C. Emborg. 1978. Increase of radiation resistance of a soil microflora exposed to long-term gamma irradiation. Applied Environmental Microbiology 36:618-619.

Falconer, D.S. 1960. Introduction to Quantitative Genetics. Oliver and Boyd, London.

Gianola, D., A.B. Chapman and J.J. Rutledge. 1977a. Effects of ancestral X-irradiation followed by random mating on body weight of rats. Genetics 86:849-860.

Gianola, D., A.B. Chapman and J.J. Rutledge. 1977b. Estimates of genetic parameters of body weight in descendants of X-irradiated rat spermatogonia. Genetics 86:861-864.

Gianola, D., A.B. Chapman and J.J. Rutledge. 1979. Responses to selection for body weight in descendants of X-irradiated rats. Genetics 91:627-645.

Gol'zberg, K.L. and I.E. Vorobtsova. 1976. Effect of single and fractionated irradiation on life span of offspring of irradiated and nonirradiated *Drosophila* males. Radiobiologiya 16:732-735. (Radiobiology 16:108-112).

Gorman, G.C., Y.J. Kim and C.E. Taylor. 1977. Genetic variation in irradiated and control populations of *Cnemidophorus tigris* (Sauria, Teiidae) from Mercury, Nevada with a discussion of genetic variability in lizards. Theoretical and Applied Genetics 49:9-14.

Grahn, D. 1972. Genetic effects of low level irradiation. Bioscience 22:535-540.

Green, E.L. 1968. Genetic effects of radiation on mammalian populations. Annual Review of Genetics 2:87-120.

Gustafsson, A. 1972. The genetic architecture of phenotype patterns in barley. In Induced Mutations and Plant Improvement, International Atomic Energy Agency, Vienna. pp. 7-12.

Haldane, J.B.S. 1937. The effect of variation on fitness. American Naturalist 71:337-349.

Holm, N.W. and R.J. Berry, editors. 1970. Manual on Radiation Dosimetry. Marcel Dekker, Inc., New York. p. 238.

Hossain, M.M., J.H. Brower and E.W. Tilton. 1972a. Sensitivity to an acute gamma radiation exposure of successively irradiated generations of the cowpea weevil. Journal of Economic Entomology 65:1566-1568.

Hossain, M.M., J.H. Brower and E.W. Tilton. 1972b. Radiation sensitivity of successively irradiated generations of the Indian meal moth. Journal of Economic Entomology 65:673-676.

I.A.E.A. 1971. Sterility Principle for Insect Control or Eradication. International Atomic Energy Agency, Vienna.

I.A.E.A. 1972. Induced Mutations and Plant Improvement. International Atomic Energy Agency, Vienna.

I.A.E.A. 1976. Effects of Ionizing Radiation on Aquatic Organisms and Ecosystems. International Atomic Energy Agency, Vienna.

Il'enko, A.I., S.I. Isaev and I.A. Ryabtsev. 1974. Radiosensitivity of some rodent species and possibility of their adaptation to artificial biogeocenosis contamination with strontium-90. Radiobiologiya 14:572-575. (Radiobiology 14:126-130).

Inagaki, E., K. Fujikawa, M. Uchibori, Y. Teranishi and Y. Nakao. 1977. X-ray brood-mutation pattern in a strain of *Drosophila melanogaster*. Journal of Science of Hiroshima University, Series B, Division 1 (Zoology) 27:159-164.

Kaplan, I.S., A.A. Kaplan, R.M. Aleksakhin and V.V. Khvostova. 1972. Effect of chronic irradiation with strontium-90 and external gamma-irradiation on cytogenetic changes in some agricultural crops. Genetika 8:52-59. (Soviet Genetics 8:986-991)

Kasim, M.H., S.R.A. Shamsi and S.A. Sofajy. 1977. Increased genetic variability in the M_2 generation of three gamma-irradiated broad bean (*Vicia faba* L.) varieties. Pakistan Journal of Botany 9:11-16.

Kawai, T. 1969. Relative effectiveness of physical and chemical mutagens. In Induced Mutations in Plants, International Atomic Energy Agency, Vienna. pp. 137-152.

Kayhart, M. 1956. A comparative study of dose-action curves for visible eye-color mutations induced by X-rays, thermal neutrons and fast neutrons in *Mormoniella vitripennis*. Radiation Research 4:65-76.

Kimura, M. and J.F. Crow. 1964. The number of alleles that can be maintained in a finite population. Genetics 49:725-738.

Kimura, M. and T. Ohta. 1971. Theoretical aspects of population genetics. Monographs in Population Biology 4:1-219.

Kiselev, P.N., K.P. Kashkin, Yu.B. Boltaks and G.A. Vitovskaya. 1961. Acquisition of radioresistance by microbe cells inhabiting media with increased levels of natural radiation. Mikrobiologiya 30:207-213. (Microbiology 30:194-198).

Kratz, F.L. 1975. Radioresistance in natural populations of *Drosophila nebulosa* from a Brazilian area of high background radiation. Mutation Research 27:347-335.

Krivolutskii, D.A., A.V. Smurov and M.A. Snetkov. 1972. Effect of soil contamination with strontium-90 on the variability of some organisms. Zhurnal Obshchei Biologii 33:587-591. (Russian with English summary).

Kumar, P.R. 1972. Radiation-induced variability in improvement of brown sarson. Radiation Botany 12:309-313.

Li, C.C. 1961. Mutation, selection and population fitness. In Mutation and Plant Breeding, National Academy of Sciences - National Research Council Publ. 891, Washington, D.C. pp. 30-47.

Lyon, M.F. and T. Morris. 1969. Gene and chromosome mutation after large fractionated or unfractionated radiation doses to mouse spermatogonia. Mutation Research 8:191-198.

Maisin, J., E. Van Duyse, A. Dunjic, J. Van der Merckt, A. Wambersie and D. Werbrouck. 1960. Acquired radio-resistance, radio-selection and radio-adaptation. In Immediate and Low Level Effects of Ionizing Radiation, A.A. Buzzati-Traverso, ed., Taylor and Francis Ltd., London. pp. 183-194.

Marques, E.K. 1973. The development of radioresistance in irradiated *Drosophila nebulosa* populations. Mutation Research 17:59-72.

Müller, H.J. 1927. Artificial transmutation of the gene. Science 66:84-87.

Nalborczyk, E., B. Zelawska and M. Kolakowska. 1971. Effects of X-rays on seed germination and growth of Scots pine (*Pinus sylvestris* L.) seedlings of different provenience. Acta Societatis Botanicorum Poloniae 40:403-412.

Newcombe, H.B. 1962. Genetic effects in populations with special reference to studies in man, including ABCC results. Radiation Research 16:531-545.

Nilan, R.A. and C.F. Konzak. 1961. Increasing the efficiency of mutation induction. In Mutation and Plant Breeding, National Academy of Sciences - National Research Council Publ. 891, Washington, D.C. pp. 437-460.

Nöthel, H. 1970. Investigations on radiosensitive and radioresistant populations of *Drosophila melanogaster*. Mutation Research 10:463-474.

Odum, E.P. 1965. Summary. In Ecological Effects of Nuclear War. Brookhaven National Laboratory Report, BNL-917. pp. 69-72.

Ojomo, O.A. and H.R. Chheda. 1972. The variation for some characters in cowpeas, *Vigna unguiculata* (L.) Walp following ionizing radiation. Radiation Botany 12:373-380.

Oster, I.I. 1963. The mutational spectrum with special reference to the induction of mosaics. In Repair from Genetic Radiation Damage and Differential Radiosensitivity in Germ Cells, F.H. Sobels, ed., Pergamon Press, Oxford. pp. 51-62.

Pawar, S.E., R.G. Thakare and D.C. Joshua. 1978. Relative biological effectiveness and mutagenic efficiency of fast neutrons in *Cajanus cajan*. Indian Journal of Experimental Botany 16:656-659.

Platonova, R.N. and V.V. Sakharov. 1962. Selection for radioresistance of diploid and autotetraploid forms of buckwheat (*Fagopyrum esculentum* Moenh). Radiobiologiya 2:595-600. (Radiobiology 2:123-131).

Rana, R.S. and M.S. Swaminathan. 1967. Relationship between chimeras and mutations induced by ^{60}Co γ-rays and 2 MeV fast neutrons at specific loci in bread wheats. Radiation Botany 7:543-548.

Russell, W.L. 1965. Studies in mammalian radiation genetics. Nucleonics 23:53-56.

Russell, W.L. 1972. The genetic effects of radiation. In Peaceful Uses of Atomic Energy, Vol. 13, International Atomic Energy Agency, Vienna. pp. 487-500.

Scossiroli, R.E. and S.S. Scossiroli. 1959. On the relative role of mutation and recombination in responses to selection for polygenic traits in irradiated populations of *D. melanogaster*. International Journal of Radiation Biology 1:61-69.

Searle, A.G. 1974. Mutation induction in mice. Advances in Radiation Biology 4:131-207.

Seetharam, A. 1976. Induced variability for quantitative characters following gamma ray treatment in linseed. Genetica Iberica 28:259-266.

Selby, P.B. 1977. Genetic effects of radiation. In Energy Sources for the Future, CONF-760744. pp. 389-401.

Shevchenko, V.A. 1970. Genetic adaptation of *Chlorella* populations to chronic effects of ionizing radiation. Genetika 6:64-73. (Soviet Genetics 6:1042-1049).

Spalding, J.F., V.G. Strang and W.L. Le Stourgeon. 1963. The effect of ancestral irradiation exposure on radioresistance in their descendants. Radiation Research 18:479-486.

Stahler, N. 1971. Changes in survival and tolerance levels in a radiation-resistant strain of *Aedes aegypti* (Diptera: Culicidae) during 90 generations. Annals of the Entomological Society of America 64:1247-1249.

Trabalka, J.R. and C.P. Allen. 1977. Aspects of fitness of a mosquitofish *Gambusia affinis* population exposed to chronic low-level environmental radiation. Radiation Research 70:198-211.

UNSCEAR, 1977. Sources and Effects of Ionizing Radiation. United Nations Scientific Committee on the Effects of Atomic Radiation, Report to the General Assembly. United Nations, New York.

Venkateswarlu, S., R.M. Singh, R.B. Singh and B.D. Singh. 1978. Radiosensitivity and frequency of chlorophyll mutations in pigeon pea. Indian Journal of Genetics and Plant Breeding 38:90-94.

Wallace, B. 1956. Studies on irradiated populations of *Drosophila melanogaster*. Journal of Genetics 54:280-293.

Wallace, B. 1958. The average effect of radiation-induced mutations on viability in *Drosophila melanogaster*. Evolution 12:532-556.

Webber, B.B. and F.J. de Serres. 1965. Induction kinetics and genetic analysis of X-ray induced mutations in the ad-3 region of *Neurospora crassa*. Proceedings of the National Academy of Sciences 53:430-437.

Wolff, S. 1967. Radiation genetics. Annual Review of Genetics 1:221-244.

Womack, J. and R. Bogart. 1968. Effects of irradiation on selection response in mice. Journal of Heredity 59:269-274.

Woodwell, G.M. and A.H. Sparrow. 1965. Effects of ionizing radiation on ecological systems. In Ecological Effects of Nuclear War, Brookhaven National Laboratory Report, BNL-917. pp. 20-38.

Wozakowska-Natkaniec, H. 1977. Ecological radiosensitivity of *Lemna minor* L. and *Spirodela polyrrhiza* (L.) Schleiden. Monographiae Botanicae 55:53-106. (Polish with English summary.)

Wright, S. 1966. Polyallelic random drift in relation to evolution. Proceedings of the National Academy of Science 55:1074-1081.

Zhestyanikov, V.D. 1963. Radioresistance of *Escherichia coli* cultivated under continuous gamma-irradiation. Radiobiologiya 3:847-853. (Radiobiology 3:96-107.)

Zhorno, L.Ya. and K.K. Surikova. 1967. Changes in radiosensitivity of spermatogenic epithelium in several generations of mice kept against a high radiation background. Byulletin Eksperimental'noi Biologii i Meditsiny 64:82-84. (Bulletin of Experimental Biology and Medicine 64:1339-1340.)

TABLE 1

EFFECTS OF HYPOTHETICAL SELECTIVE FORCES ON ALLELE FREQUENCIES AT TWO-ALLELE LOCI[1]

	Genotype	Fitness	Relative Frequency	
			Before Selection	After Selection
LOCUS I[2]	AA	1	p^2	p^2
	Aa	1	$2pq$	$2pq$
	aa	$1 - s$	q^2	$q^2(1 - s)$
			1	$1 - sq^2$
LOCUS II[3]	AA	1	p^2	p^2
	Aa	$1 - s$	$2pq$	$2pq(1 - s)$
	aa	$1 - s$	q^2	$q^2(1 - s)$
			1	$1 - 2sq + sq^2$
LOCUS III[4]	AA	$1 - s$	p^2	$p^2(1 - s)$
	Aa	1	$2pq$	$2pq$
	aa	$1 - s$	q^2	$q^2(1 - s)$
			1	$1 - 2sq^2 - s(1 - 2q)$

1 Symbolism defined in text, pp. 6 and 7
2 LOCUS I - Allele a recessive
3 LOCUS II - Allele a dominant
4 LOCUS III - Alleles a and A mutually heterotic

TABLE 2

NUMBER OF DELETERIOUS MULTIPLE ALLELES MAINTAINED AT A SINGLE LOCUS BY DIFFERENT MUTATION RATES (μ) IN POPULATIONS OF SIZE N*

N	μ	Allele Type		
		Recessive ($s = 0.1$)	Semidominant ($s = 0.01$)	Heterotic ($s = 0.1$)
10^5	10^{-6}	2.7	1.3	73
	10^{-5}	23	13	126
10^6	10^{-6}	27	13	289
	10^{-5}	226	133	700

* From Wright (1966)

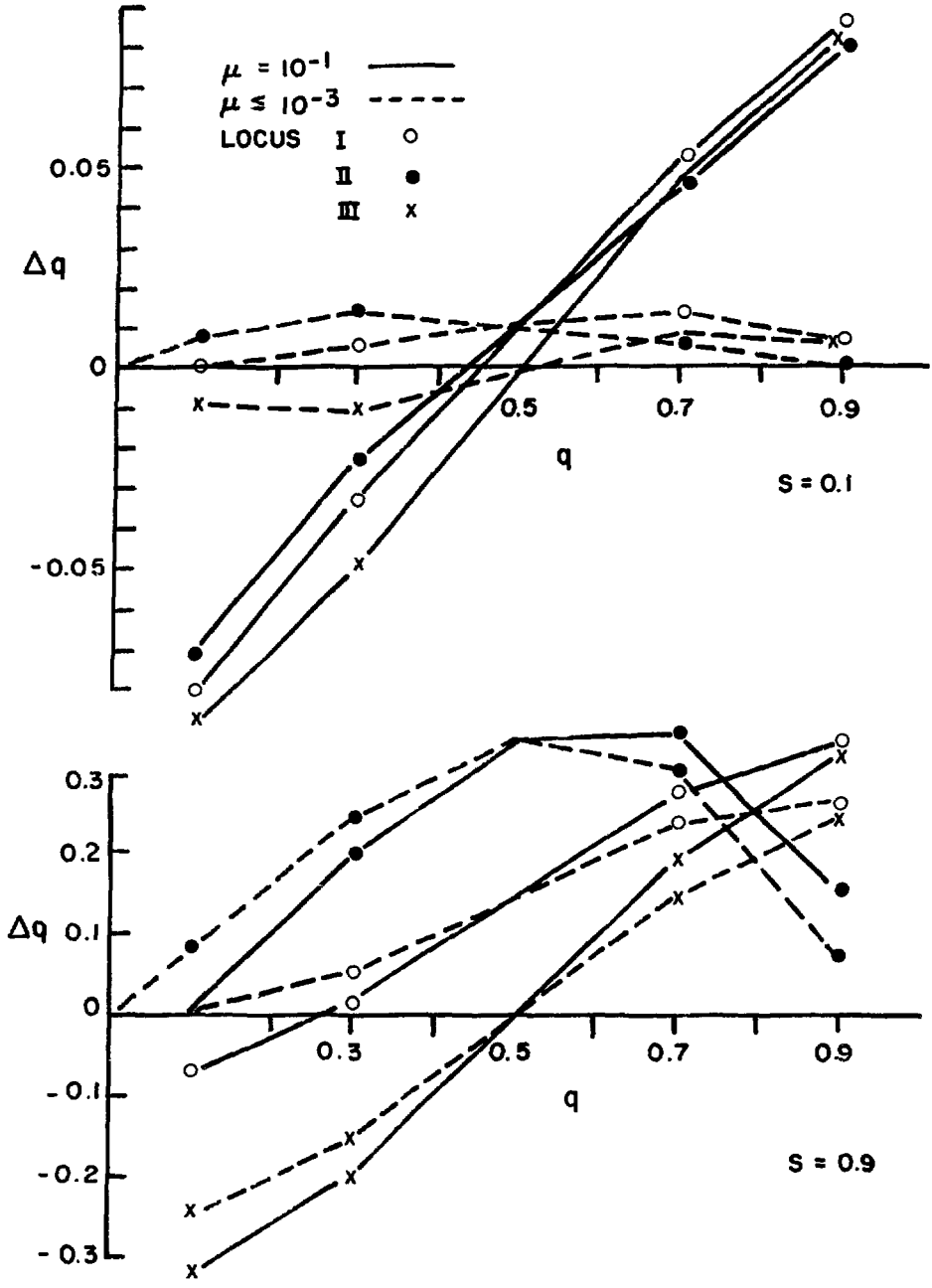

FIGURE 1: Decrease (Δq) in Detrimental Allele Frequency after One Generation of Selection (s) at Different Mutation Rates (μ).

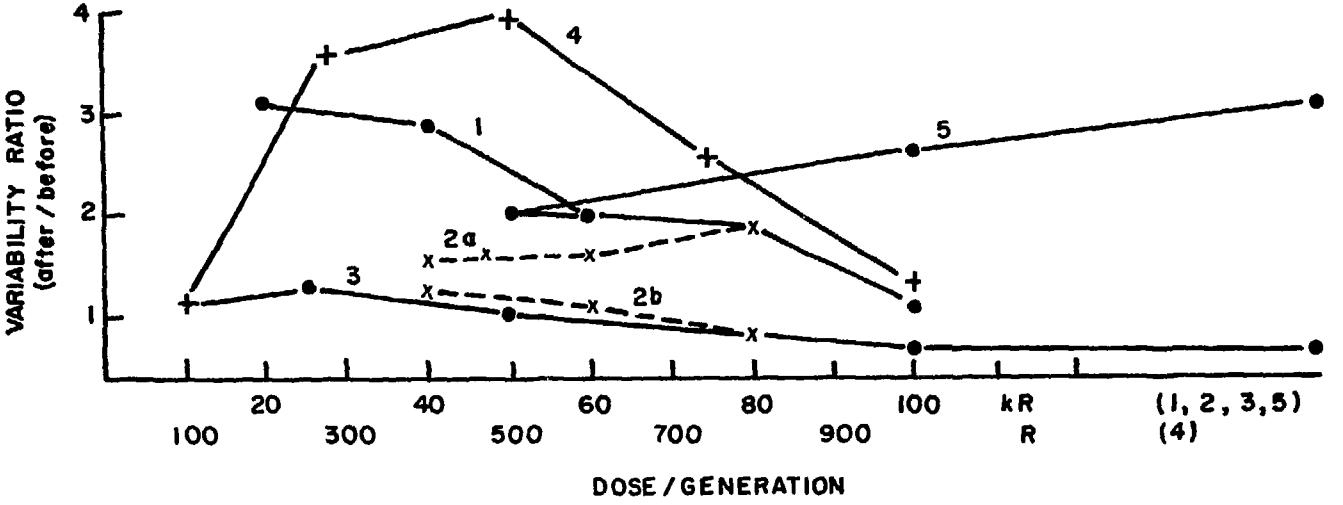

FIGURE 2: Dose Relationships for Increase in Population Variability after Ancestral Irradiation

1. Range of seeds/silique (sarson) (Kumar, 1972).
2. a) Range of seed oil content (linseed) (Seetharam, 1976).
   b) Range of plant height (linseed) (Seetharam, 1976).
3. Standard dev. of seed yield (flax) (Bari, 1971).
4. Standard dev. of flowering time (bean) (Kasim et al., 1977).
5. Standard dev. of flowering time (wall-cress) (Daly, 1973).

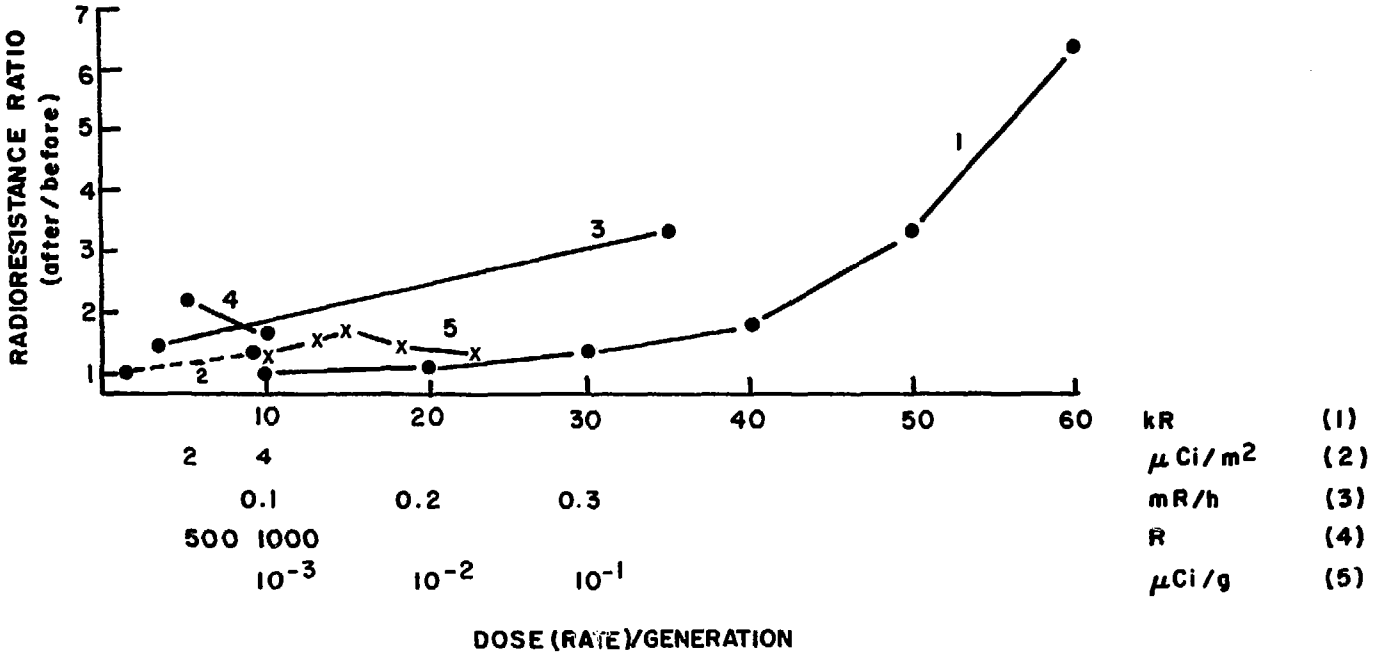

FIGURE 3: Dose Relationships for Increase in Radioresistance after Ancestral Irradiation

1. Survival at 10-60 kR (buckwheat) (Platonova and Sakharov, 1962)
2. Normal mitosis at 1.5 Ci (agrimony) (Cherezhanova et al., 1971)
3. Median Lethal dose (*Micrococcus*) (Kiselev et al., 1961)
4. Competitive ability at 1 kR (fruit fly) (Marques, 1973)
5. Survival at 30 kR (*Chlorella*) (Shevchenko, 1970)

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