GENETIC CONTROL OF RADIATION SENSITIVITY IN *SACCHAROMYCES CEREVISIAE*[1,2]

MICHAEL A. RESNICK[3]

Donner Laboratory, University of California, Berkeley, California 94720

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THE removal of lethal lesions induced by UV, X rays, as well as a variety of mutagens has been shown to be under genetic control in bacteria and has been termed dark repair (HAYNES 1966; HOWARD-FLANDERS and BOYCE 1966; SETLOW 1968). Although the order of the steps involved may differ, two mechanisms of repair proposed by HOWARD-FLANDERS and BOYCE (1966) and by HAYNES (1966) essentially involve recognition of the lesion, removal of the lesion and adjoining DNA in the same strand, and resynthesis using the opposite strand as a template.

A dark repair system for the removal of UV and X-ray damage also appears to exist in the yeast *Saccharomyces cerevisiae*. Survival increases when irradiated cells are held in buffer after irradiation rather than being plated immediately on nutrient medium (PATRICK, HAYNES and URETZ 1964). Furthermore, several radiation-sensitive mutants have been isolated in yeast (NAKAI and MATSUMOTO 1967; SNOW 1967; LASKOWSKI, LOCHMANN, JANNSEN and FINK 1968; COX and PARRY 1968).

In the present study genes previously identified by NAKAI and MATSUMOTO (1967) and by SNOW (1967) have been subjected to further genetic analysis using newly isolated mutants of these genes. Mutants exhibiting sensitivities to radiation different from those previously reported are also described. The isolation of mutants sensitive to only X rays is of particular relevance in characterizing the repair mechanisms in yeast.

MATERIALS AND METHODS

Yeast strains: Saccharomyces cerevisiae heterothallic strains were used. All strains were obtained or derived from those of Dr. ROBERT K. MORTIMER. Genetic markers have been described (RESNICK 1969).

Media: The YEPD, synthetic complete (C), omission (C-X) and sporulation media were previously described (RESNICK 1969). Depending on the genetic characteristic being tested, the following were also added to the synthetic complete medium: 20 mg/l tyrosine, 20 mg/l isoleucine, 150 mg/l valine, 50 mg/l phenylalanine, 60 mg/l canavanine (in C-AR). Strains exhibiting the petite phenotype were unable to grow on a medium containing 3% glycerol, 0.025% dextrose, 1% yeast extract, 1% Bacto-peptone, and 2% agar. The medium used for scoring the ability to ferment galactose (as indicated by a change in color of the agar from blue to yellow) contained 1% yeast extract, 2% Bacto-peptone, 2% agar and 2% galactose (sterilized separately). Follow-

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[3] Present address: National Institute for Medical Research, Mill Hill, London, NW 7, England.

ing sterilization, these ingredients were mixed and the pH adjusted to 8.0 with ½ N NaOH. To this 0.3% (v/v) of a 1% brom-thymol-blue solution in ethanol was added.

Methods: Procedures for mating, sporulation, and tetrad analysis (RESNICK 1969), as well as random spore analysis (GILMORE 1967), have been described. The technique used by HAWTHORNE and MORTIMER (1969) for determining the linkage of pairs of suppressors was used to assess whether the centromere-linked gene S_3 was linked to any of the centromere-linked genes identified in the present study. A strain containing the *uvs* or *xs* gene to be tested was mated with another that carried S_3 and *can*. The *can* allele is suppressible, confers resistance to canavanine, and in the presence of S_3 a *can* strain is canavanine sensitive. When a sonicated suspension of a sporulated diploid whose genotype is $+/uvs\ S_3/+\ can/+$ is plated on C-AR+CAN medium, only *can* spores that do not contain S_3 will grow. If a *uvs* or an *xs* gene is not linked to S_3, then 50% of the spore colonies which arise (all of them "+" regarding S_3) should be UV or X-ray sensitive, respectively.

Mutations leading to radiation-sensitivity were induced by exposing cells (10^8 cells/ml) to 0.1 M $NaNO_2$, pH 4.5, for 60 min (approximately 30% survival). Cells were then plated to YEPD after dilution to yield about 50 colonies per plate. The technique of NAKAI and MATSUMOTO (1967) was employed to detect the sensitive mutants. The colonies that arose after nitrous acid treatment were replica plated to three YEPD plates. One plate was irradiated with 300 ergs/mm^2 UV, another with 75 kR X rays, and the third received no irradiation. The plates were examined after one day (UV) or two days (X rays). Growth of the imprints on the irradiated plates was compared to that on the unirradiated plate. At these exposures, enough cells in the replica imprints had survived to result in a nearly confluent growth. Absence of growth of an imprint on an irradiated plate indicated that cells of the corresponding colony were radiation-sensitive.

To determine if any mutants were allelic, the radiation-sensitivities of the diploids formed by all pairwise crosses of the mutants were examined. Individual zygotes resulting from the mating of pairs of mutants were isolated. Streaks of the diploid colonies arising from these zygotes were replica plated to YEPD and irradiated with UV and X rays in the same manner as that used for the isolation and characterization of the radiation-sensitive mutants. Allelism was indicated by a lack of growth of the irradiated imprint of $mutant_x \times mutant_y$.

The procedure for determining survival curves has been described (RESNICK 1969). Cells were irradiated by UV with or without photoreactivating light (for light sources see RESNICK 1969) or by X rays from a beryllium-window tube (Machlett OEG 60) at a dose rate of 250 r/sec (50 kvp, 25 ma). The dose modifying factor, DMF, is the factor of increase in dose required to obtain equal survival between two strains or two conditions of irradiation.

RESULTS

Mutants sensitive to UV and X rays: Eleven radiation-sensitive mutants were isolated from approximately 3300 colonies that had arisen after treatment of cells of X1687-101B (*a ad 2–1 ly 1–1 le 1–12 ar 4–17 hi 5–2*) with nitrous acid. Three mutants were UV-sensitive (*uvs*), seven were X-ray sensitive (*xs*), and one was sensitive to both types of radiation (*uxs*).

Analyses of asci from crosses of radiation-sensitive mutants with non-sensitive strains are presented in Table 1. Three of the X-ray sensitive mutants, 378, 380, and 382, when crossed to non-sensitive strains, exhibited either poor sporulation or low spore viability and were, therefore, excluded from further study. For the rest of the mutants, 2:2 segregation of the sensitive:non-sensitive phenotype was observed. Thus the radiation-sensitive phenotypes exhibited by the mutants were concluded to be under the control of chromosomal genes. Since the *uxs* phenotype also segregated in a 2:2 fashion (among 44 asci examined; this study and FOGEL,

TABLE 1

Numbers of PD, NPD and T asci for radiation-sensitive* vs. *miscellaneous genes*

Mutant strain	Sensitivity	Numbers of asci																		Segregation	
		ar 4–17†			*hi 5–2*			*ly 1–1*			*le 1–12*†			*thr 1*			*α*			1st Division	2nd Division
		PD	NPD	T	PD	NPD	T	PD	NPD	T	PD	NPD	T	PD	NPD	T	PD	NPD	T		
KC370	*uvs*	3	0	3	2	0	4	1	0	5	1	3	2	1	2	3	2	1	5	4	2
KC371	*uvs*	1	3	4	0	1	8	4	1	4	3	4	2	1	2	6	1	0	8	6	2
KC373	*uvs*	3	2	3	2	0	6	1	1	6	2	2	4	2	0	6	3	1	4	5	3
KC372	*uxs*	3	3	4	3	4	2	1	4	5	4	2	4	3	2	5	0	6	4	6	4
KC376	*xs*	0	1	6	2	0	7	2	1	4	1	1	6	1	1	5	0	2	4	2	5
KC377	*xs*	2	2	8	2	1	9	1	5	6	1	4	7	2	1	9	2	4	5	5	8
KC381	*xs*	3	1	12	3	5	9	6	1	9	1	1	15	2	2	12	0	2	16	2	15
KC383	*xs*	1	1	5	1	1	6	1	2	4	2	1	5	1	0	7	.	.	.	3	5
KC378	*xs*	No sporulation or low spore viability																			
KC380	*xs*	No sporulation or low spore viability																			
KC382	*xs*	No sporulation or low spore viability																			

* PD = parental ditype; NPD = nonparental ditype; T = tetratype.
† *ar 4* and *le 1* exhibit, respectively, 16.8% and 4.9% second division segregation (MORTIMER and HAWTHORNE 1966).

personal communication), it was considered to be due to a single gene mutation rather than mutations in two separate genes, one controlling X-ray sensitivity and the other UV sensitivity.

Allelism of mutants: Replica-imprints of diploids homozygous for single mutations (homo-allelic) exhibited no growth when irradiated with the dose and type of radiation to which the given mutant was sensitive. However, considerable growth was observed on replica-imprints of crosses between mutants and non-sensitive strains; thus, all the mutants were considered to be recessive. Among crosses involving all pairwise combinations of mutant isolates two pairs of mutants were observed to be allelic: 370–371 and 377–381. Mutant 373 was determined to be allelic with UV^{s}_{1}, a UV-sensitive mutant isolated by NAKAI and MATSUMOTO (1967). In allelism tests of 371 and 373 with the centromere-linked *uvs* mutant isolated by SNOW (1967), *uvr-9*, mutants 371 and *uvr-9* were found to be allelic.

Although it was possible to identify some mutants that were allelic by these tests, other instances of allelism could have escaped detection. Such would be the case if two mutants at the same locus were able to complement so that a diploid formed from them was less sensitive to radiation. To resolve whether mutant pairs that yielded non-sensitive responses in the above tests were allelic or not, the spores of asci derived from these crosses were analyzed. The identification of non-sensitive spores in a small sample of asci was considered to be sufficient evidence for a mutant pair not being allelic. No further cases of allelism were observed beyond those noted above. Mutants identified in this study as well as allele designations are presented in Table 2.

Centromere linkage of mutants: None of the mutants were linked to any of the genes tested (Table 1). However, the *uxs* and the *uvs* mutants appeared to be centromere-linked since the second division segregation (SDS) frequencies of these mutants were less than 2/3 (MORTIMER and HAWTHORNE 1966); they were tested further. The *uvs 9* and the *uvs 1* mutants were centromere-linked (as

TABLE 2

Radiation sensitive mutants

Mutant	Sensitive to	2nd division segregation frequency*	Mutant gene designation
uvs (370)	UV	.46	*uvs 9–2*†
uvs (371)	UV	. .	*uvs 9–3*†
uvs (373)	UV	.39	*uvs 1–2*‡
uxs (372)	UV and X ray	.59	*uxs 1*
xs (376)	X ray	.71	*xs 1*
xs (377)	X ray	.62	*xs 2–1*
xs (381)	X ray	.87	*xs 3*
xs (383)	X ray	.63	*xs 2–2*

* Compiled from data presented in Table 1 and text.
† Allelic to *uvr-9* (SNOW 1967).
‡ Allelic to UV^{s}_{1} (NAKAI and MATSUMOTO 1967).

discussed below) while the *uxs* mutant was not, based on a frequency of SDS (26/44) not significantly different from 2/3 (this study and FOGEL, personal communication).

To identify the chromosomes on which *uvs9* (strain 371) and *uvs 1* (strain 373) were located, strains marked by these genes were crossed to strains that had centromere-linked genes on chromosomes I through XVI (MORTIMER and HAWTHORNE 1966; HAWTHORNE and MORTIMER 1969). All crosses, except the one involving super-suppressor S_3 (see MATERIALS AND METHODS) were analyzed by tetrad analysis. Neither *uvs 9* nor *uvs 1* was found to be linked to S_3. The frequency of colonies on C-AR+CAN that were *uvs* was not significantly different from .50 for crosses involving *uvs 9–3* or *uvs 1–2*: .509 (251/493) and .529 (111/210), respectively.

The results of tetrad analyses of crosses involving either *uvs 9* or *uvs 1* and the rest of the centromere-linked genes are presented in Table 3. Since the ratio of PD:NPD asci is significantly greater than 1 (16:3) for the gene pair *uvs 1–ty 7*, the *uvs 1* gene is linked to the centromere of chromosome XVI. Based on the evidence which follows, *uvs 1* is 19.5 centimorgans from the centromere of chromosome XVI and on the arm opposite to that marked by *ty 7*. In a cross containing *uvs 1* and *ty 7* the second division segregation frequencies of *uvs 1* and *ty 7* were .32 (12/37) and .54 (22/41), respectively. When results for all experiments in the present study were included, the SDS for *uvs 1* was .39 (37/94; compiled from various crosses involving *uvs 1* and the centromere-linked genes in Table 3). If *uvs 1* and *ty 7* are on the same arm of chromosome XVI, no NPD asci would be

TABLE 3

Numbers of PD, NPD, and T asci for* uvs 9–3 *and* uvs 1 vs. *centromere-linked genes*

Chromosome number	Centromere-linked gene	*uvs 9–3* PD	NPD	T	*uvs 1–2* PD	NPD	T
1	*ad 1*	1	2	4	3	4	7
2	*ga 1*	1	2	6	3	3	6
3	*hi 4*	2	1	19	3	5	12
4	*tr 1*	3	1	9	4	3	5
5	*ur 3*	3	3	8	3	5	4
6	*hi 2*	3	1	7	1	2	9
7	*le 1*	3	4	2	3	1	4
8	*ar 4*	1	3	4	3	2	3
9	*ly 1*	7	4	17	1	1	6
10	*is 3*	10	3	11	5	3	3
11	*met 14*	4	2	4	9	4	10
12	*thr 5*	4	2	4	3	1	9
13	*ly 7*	3	3	8	1	1	10
14	*p 8*	4	2	5	3	4	4
15	S_3†	.	.	.	.	.	.
16	*ty 7*	3	2	10	16	3	19

* PD = parental ditype; NPD = nonparental ditype; T = tetratype. Summary of results from various crosses.

† See text.

expected. Among the 19 tetratype asci, 5 were first division for *uvs 1*, 13 were first division for *ty 7*, and 1 was 2nd division for both markers. These results would not be expected if *ty 7* were linked to *uvs 1* on the same arm of chromosome XVI.

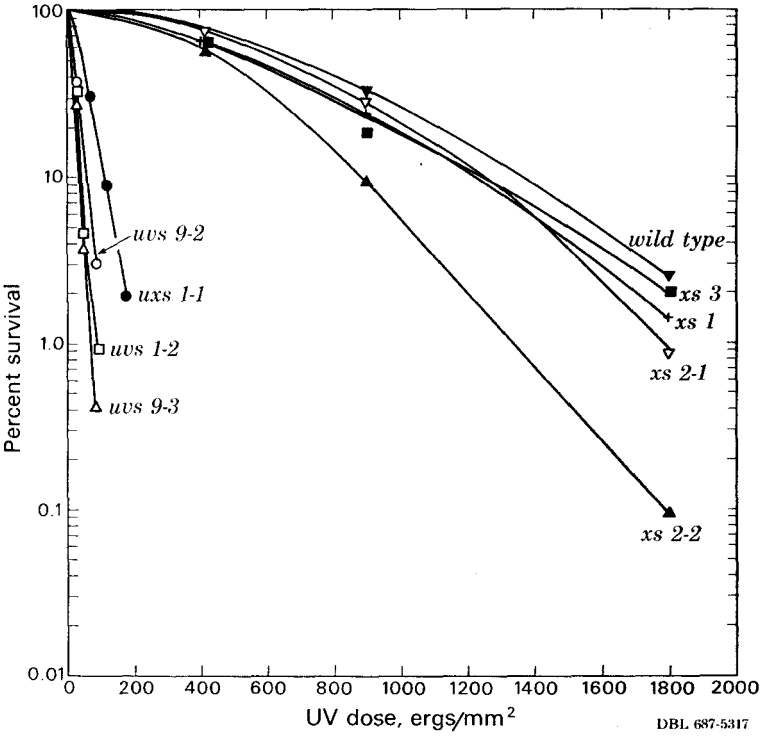
The *uvs 9* gene appears to be unlinked to any of the centromere-linked genes identifying chromosomes I through XVI (Table 3). It is, therefore, considered to mark the centromere of a newly identified chromosome XVII at a distance of 23 centimorgans [SDS is .46 (22/48)]. Eighteen chromosomes have been observed cytologically in *S. cerevisiae* (TAMAKI 1965).

Suppressibility of mutants: MARKOVITZ and BAKER (1967) have reported the suppressibility of radiation sensitivity in bacteria by an ochre suppressor and concluded that the product of the corresponding gene is a polypeptide. Similarly the suppressibility of the radiation-sensitive mutants in the present study was tested. Revertants arising on replica-imprints of the mutants on C-LY-HI were isolated. Since thesse revertants were presumably due to the presence of super-suppressors (HAWTHORNE and MORTIMER 1969), the suppressibility of the radiation-sensitive mutants could be assessed. None of the mutants was resistant when a super-suppressor for *ly 1–1* and *hi 5–2* was present. Some of the mutants, however, may still be suppressible since all possible super-suppressors are not selected by this system (GILMORE 1967; HAWTHORNE and MORTIMER 1969).

Sporulation of radiation sensitive mutants: All pairwise crosses of the *uvs* and *uxs* mutants exhibited good sporulation and spore viability. However, either no sporulation or no viable spores were observed in the crosses *xs 1/xs 1*, *xs 3/xs 3*, or *xs 1/+ +/xs 3*. The *xs 1* and *xs 3* genes, therefore, seem to affect the sporulation processes. Lack of sporulation associated with *xs 1* and *xs 3* is a recessive trait since sporulation was observed in crosses involving these genes with other strains. The inability of the cross heterozygous for both *xs 1* and *xs 3* to sporulate may be due to these genes being allelic. On the other hand, factors not associated with these mutants may prevent sporulation. No sporulation was observed in a cross involving *xs 1* and *xs 2–1*, while in the cross *xs 1* × *xs 2–2* good sporulation and spore viability occurred.

Another X-ray sensitive mutation in yeast, reported by PUGLISI (1967), also prevents sporulation. This mutant differs from *xs 1* and *xs 3* in that it is dominant and has not been isolated in a haploid. It is possible that mutants of these genes interfere with recombination during meiosis in a manner analogous to that reported for recombinationless mutants of *E. coli* (CLARK and MARGULIES 1965; HOWARD-FLANDERS 1966). Similarly, UV-sensitive mutants of yeast (COX and PARRY 1968), *Neurospora crassa* (LANIER 1968), *Aspergillus nidulans* (CHANG 1968) and *Ustilago maydis* (HOLLIDAY 1967) that affect meiosis have been reported.

Sensitivity of mutants to X rays and UV: Haploid and homozygous diploid strains marked by any one of the mutations *uvs 9–2*, *9–3*, or *uvs 1–2* are very sensitive to UV radiation (Figures 1 and 2). The DMF of these strains compared to the wild-type parent-strain (X1678–101B) and a wild-type diploid was between 20 and 30 in the dose range examined. Although the mutants were sensitive to UV, no increased sensitivity to X rays was observed (Figure 3). The responses



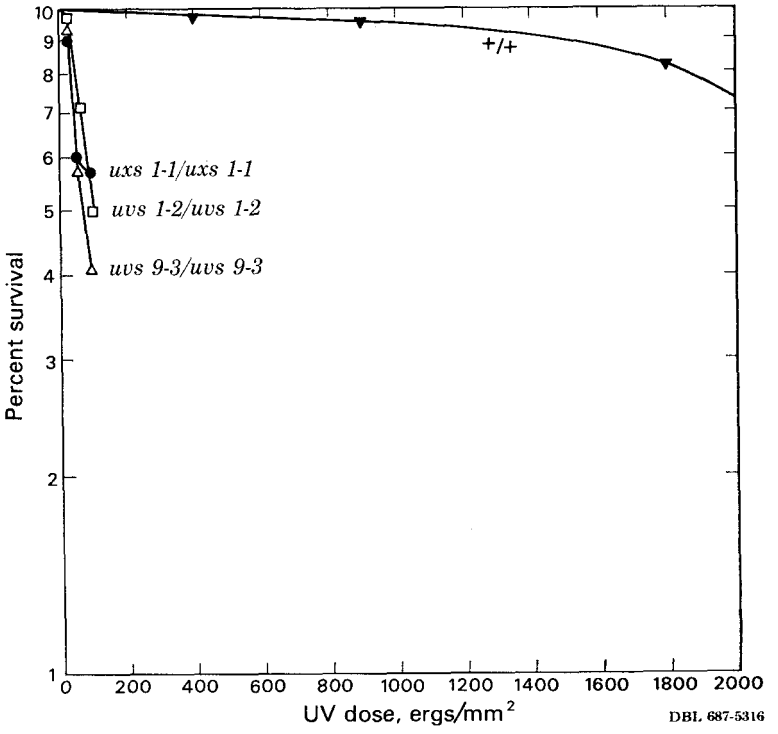


FIGURE 1.—Survival after UV-irradiation of haploid wild type and radiation-sensitive strains.

FIGURE 2.—Survival after UV-irradiation of a diploid wild-type strain and strains sensitive to UV.

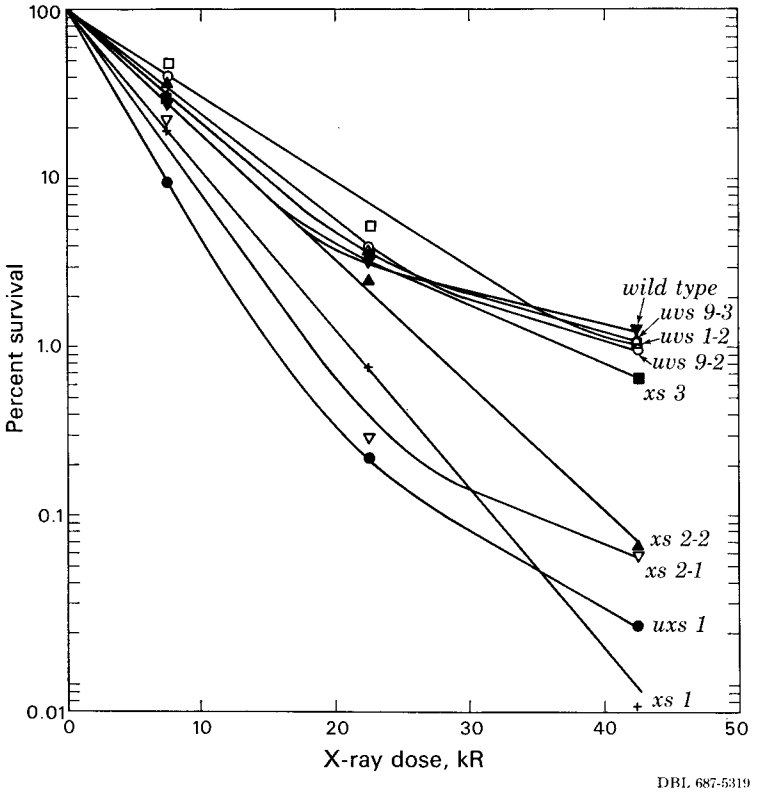


FIGURE 3.—Survival after X-ray irradiation of haploid wild-type and radiation-sensitive strains.

of these mutants to UV and ionizing radiation are, therefore, in agreement with those reported for *uvr-9* (SNOW 1967) and UV^{s}_{1} (NAKAI 1967) to which *uvs 9–2 9–3* and *uvs 1–2*, respectively, are allelic.

Responses of the *uvs* mutants to UV are also presented in Figure 4. All curves have a shoulder at low doses, indicating that a low level of repair may occur in the *uvs* mutants or that a threshold accumulation of UV damage is required for the killing of a cell. Another type of repair, photoreactivation (PR), resulted in the same level of survival for the three *uvs* strains (Figure 4). Since photoreactivation removes UV-induced pyrimidine dimers (SETLOW 1966) and at the doses applied to the *uvs* strains the survival of the wild-type haploid was nearly 100% (without PR), the dark repair system in yeast is very efficient in removing the damage that remains following PR in the *uvs* strains; this sector of damage may be due to dimers remaining after PR or other lesions.

At low doses, the X-ray responses of the *xs* mutants were similar to that of the wild-type strain (Figure 3). The DMF's of these mutants were between 1 and 1.5. However, at high doses the tailing of the X-ray survival curve observed for the wild-type strain was either absent or much lower for the *xs* mutants (for *xs 3* this becomes apparent at the high doses used for isolation of mutants). This

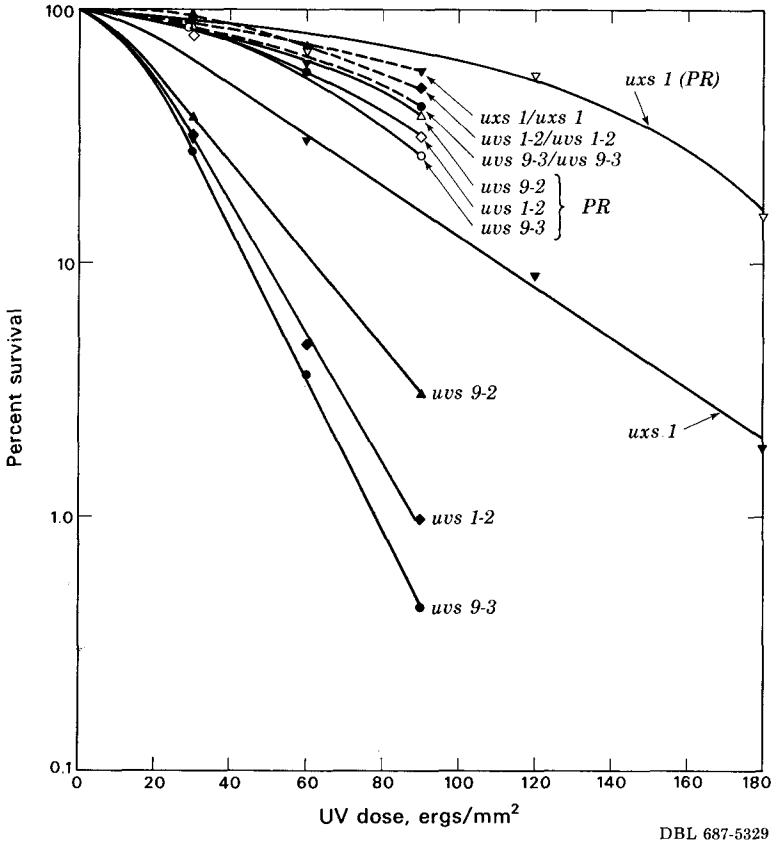


FIGURE 4.—Survival after UV-irradiation of wild-type and UV-sensitive haploid and diploid strains. Closed and open symbols: No PR and PR, respectively. Solid and dashed lines represent haploid and diploid responses, respectively.

tail indicates a resistant fraction in the population identified as budded cells (BEAM *et al.* 1954). These budding cells are much more resistant to X rays than interdivisional cells. Since the frequency of budded cells was about 10–20% for the irradiated *xs* and *XS* cells, the *xs* phenotypes probably result from alterations of genes that normally confer resistance to X rays during budding.

The decrease of the shoulders in the X-ray survival curves of the *xs/xs* diploid strains when compared to the wild type (Figure 5) indicates that the genes controlling X-ray resistance during the haploid budding stage also affect the resistance of diploid cells. The wild-type diploid was at least 2 times more resistant to X rays compared to any of the diploids homozygous for *xs* mutations (Figure 5). Furthermore, the relative reduction of the shoulder in the survival curve of *xs/xs* strains appeared to be related to the decrease in tailing of the haploid survival curve. The correlation between the shoulder and the tailing in the survival curves of *xs/xs* diploids and *xs* haploid strains, respectively, indicates that the factor that confers resistance to X rays in budding haploid cells also confers

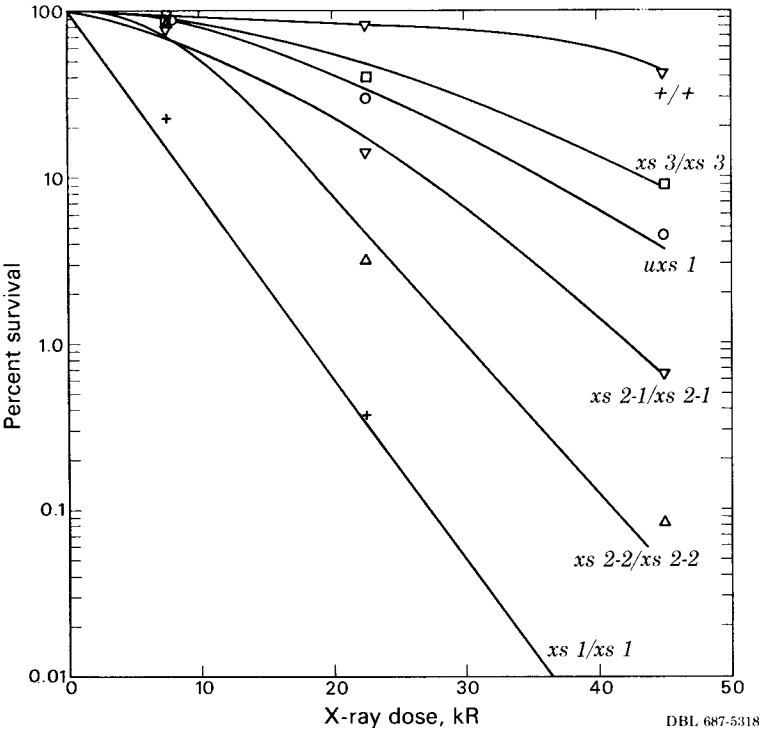


FIGURE 5.—Survival after X-ray irradiation of diploid wild-type and radiation-sensitive mutants.

resistance to diploid cells. The mechanism of repair that may be involved, however, requires further investigation.

All of the *xs* mutants, except *xs 2–2*, have the same sensitivity to UV as the wild type (Figure 1). Since the strain carrying the *xs 2–2* mutation was about 1.5 times as sensitive to UV as a strain with *xs 2–1*, which was not sensitive to UV, *xs 2–1* is possibly a leaky mutation of the *xs 2* gene. This is corroborated by the increased X-ray resistance of the *xs 2–1/xs 2–1* diploid over the *xs 2–2/xs 2–2* diploid (Figure 5). Therefore, the *xs 2* locus can be considered to affect X-ray sensitivity and to a very small extent UV sensitivity, whereas the *xs 1* and *xs 3* loci affect only sensitivity to ionizing radiation.

DISCUSSION

Based on genetic analyses and radiation responses of the mutants isolated in this and other studies (NAKAI and MATSUMOTO 1967; SNOW 1967; LASKOWSKI *et al.* 1968; COX and PARRY 1968), the genetic control of radiation sensitivity in yeast appears to be complex. COX and PARRY (1968) have identified 22 loci involved with sensitivity to either UV or to both UV and X rays. As shown in the present study there are also genes affecting sensitivity to X rays but not to UV.

(A gene that controls photoreactivation of UV-induced damage in yeast has also been identified (RESNICK 1969).)

Similarities are apparent for the genetic control of radiation sensitivity in bacteria and yeast. Mutants have been isolated in each organism that are sensitive to UV or both UV and X-rays (HOWARD-FLANDERS and THERIOT 1966). However, in yeast the *uxs* mutants do not display the same degree of sensitivity to UV as the most sensitive *uvs* mutants (*uvs 9* and *uvs* 1). Furthermore, in the present study mutants of yeast that are sensitive only to X-rays have been identified while no such mutants have been reported for bacteria. KATO and KONDO (1967) have isolated a mutant of bacteria that does not exhibit repair of X-irradiated phage DNA although UV damage is repaired. However, this mutant is able to repair X-ray damage induced in its own genome. It is likely, therefore, that in bacteria there may be only one pathway for the repair of radiation-induced damage, while in yeast there are at least two pathways (COX and PARRY 1968; RESNICK 1968) one of which may be similar to the repair system in bacteria. The identification of *uxs* mutants, indicates common steps in these pathways and multilocus control of sensitivity implies more than one step in each of the pathways.

Based on evidence obtained with mutants of bacteria that are UV and X-ray sensitive and also recombination-deficient, HOWARD-FLANDERS, RUPP, and WILKINS (1968) have proposed that one of the mechanisms for increasing survival may involve a type of recombination-repair of lethal damage. A similar situation may exist for *Saccharomyces cerevisiae* as it appears to for *Ustilago maydis* (HOLLIDAY 1967). Two mutants *xs 2* and *xs 3* in the present study have been shown (RODARTE, personal communication) to be allelic with recombination-deficient mutants identified by RODARTE, FOGEL, and MORTIMER (1968). Other mutants (4/10) identified by these authors were also X-ray sensitive, while none of the UV-sensitive mutants identified by SNOW (1968) were recombination-deficient. Thus since some steps involved in the pathway leading to the alteration of X-ray induced lethal damage are also involved in recombination, it is possible that in this pathway damage is removed by a mechanism of recombination repair as has been suggested for bacteria. Although the nature of repair pathways for UV-induced damage is not known, it appears that at least one of them is involved with the excision of UV-induced pyrimidine dimers (RESNICK 1968).

In addition to some mutants affecting recombination, others have been shown to have altered spontaneous and induced mutation frequencies. A mutant of the *uvs 9* gene exhibits much higher UV-induced mutation frequencies than the wild type for all loci studied (RESNICK 1968), while another mutant *xs 1* has a much higher spontaneous mutation frequency (VON BORSTEL, GRAHAM, BROT, and RESNICK 1968). Thus it appears that in yeast there is an intimate relationship between various genetic processes and the mechanisms for the repair of radiation-induced lethal damage.

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SUMMARY

Eight mutants of genes that control radiation sensitivity in *Saccharomyces cerevisiae* have been isolated after nitrous acid treatment. Three are sensitive to UV (*uvs 9–2*, *9–3*, and *uvs 1–2*), one is sensitive to UV and X rays (*uxs 1*), and four are X ray sensitive (*xs 1*, *xs 2–1*, *2–2*, *xs 3*). The *uvs 1* gene is 19.5 centimorgans from the centromere of chromosome XVI and *uvs 9* is 23 centimorgans from the centromere of a newly identified chromosome XVII.

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