GENES AFFECTING SENSITIVITY TO ULTRAVIOLET LIGHT IN THE *malB* REGION OF THE CHROMOSOME OF *ESCHERICHIA COLI*¹

YOUNG S. CHUNG AND JOSEPH GREENBERG²

Palo Alto Medical Research Foundation, Palo Alto, California 94301

Received October 24, 1967

THIS report is another in a series describing loci of genes affecting ultraviolet (UV) sensitivity (resistance) in *Escherichia coli* strain B. We have already shown that strain B itself is sensitive to UV light compared to most strains of *E. coli*, because of a mutation in a gene in the *lac tsx** region (GREENBERG 1964a). Genes responsible for increased UV sensitivity of mutants of strain B have been located: B_{III} (RÖRSCH, EDELMAN and COHEN 1963) linked to *his* (GREENBERG 1965); *uvr2* (in strain Bs2) (HILL and SIMSON 1961) linked to *malB* (GREENBERG 1964b). Bs1 is a double mutant, one gene *uvr1A* linked to *gal* making the strain unable to reactivate irradiated phage (HCR), the other *uvr1B* linked to *malB* (MATTERN, ZWENK and RÖRSCH 1966; GREENBERG 1967). In parallel studies of UV sensitive mutants of K-12, HOWARD-FLANDERS, BOYCE and THERIOT (1966) have shown that the gene, *uvrA*, one of three which make the host HCR, is linked to *metA*. SCHWARTZ (1966) found the order of markers in this region of the chromosome to be *metA malB uvrA*.

We will show by transductional analysis with phage P1 that the gene, *uvr2*, for radiation sensitivity in strain Bs2 is also linked to *metA* and *malB* and that the order of markers in the region is *metA malB uvr2 uvrA*.

MATERIALS AND METHODS

Bacterial strains: The strains of bacteria used and their properties are shown in Table 1. Strain B and all its derivatives, unless otherwise noted, are naturally Mal and λ^r . Strain B251 is Mal⁺ λ^s as a result of the transduction of the *mal*⁺ gene into it (ARBER and LATASTE-DOROLLE 1961). PAM 36, a His derivative of W1895, HfrC was treated with 1-methyl-3-nitro-1-nitrosoguanidine (MANDELL and GREENBERG 1960) to induce 12 Mal mutants. Six of these were λ^s and were able to recombine with AB325 (*malA* λ^r) to give *mal*⁺ λ^s recombinants at a high frequency. The other Mal mutants were λ^r , were unable to recombine with AB325 to give Mal⁺ recombinants, and were considered to be *malA*. Recombination experiments performed with the six *mal* λ^r mutants placed their *mal* marker in the region of *malA*. In this small series of Mal mutants there were no *malA* λ^s nor any *malB* λ^r . Recombination experiments performed with one of the *mal* λ^s mutants (PAM 201) as recipient, placed the *mal* marker between *ara*⁺ and *arg*⁺. The *mal* λ^s marker of PAM 201 was, therefore, probably *malB*.

Media and culture methods: The media and culture methods unless otherwise described were as described by GREENBERG (1964a). All incubations were done at 37°C.

* Abbreviations unless otherwise stated are those recommended in DEMEREC, ADELBURG, CLARK and HARTMAN (1966).

¹ This work was carried out under Public Health Service Grant CA 05687-06

² Recipient of a Public Health Service Career Development Award

TABLE 1
Characteristics of strains used

Strain	Sex	Characters	Origin
<i>Derivative of strain B</i>			
B	F ⁻	<i>mal</i> λ^r	R. HILL
Bs2	F ⁻	<i>mal</i> λ^r <i>uvr2</i>	R. HILL
PAM 206		<i>mal</i> ⁺ λ^s <i>uvr2</i>	Transduction of Bs2
B251	F ⁻	<i>mal</i> ⁺ λ^s	W. ARBER
<i>Derivative of strain K-12</i>			
AB325	F ⁻	<i>lac gal his serA xyl mtl argF thi</i> <i>malA</i> λ^r <i>ton tsx str</i>	A. L. TAYLOR
AB1886	F ⁻	<i>arg pro leu thr his thi met</i> ⁺ <i>lac ara xyl tsx str uvrA</i>	P. HOWARD-FLANDERS
PAM 207		<i>arg</i> ⁺ <i>pro leu thr his thi metA lac</i> <i>ara xyl ton tsx str uvrA</i>	Transduction of AB1886
AB1911	F ⁻	<i>arg metA gal lac xyl tsx</i>	E. ADELBERG
PAM 202		<i>arg</i> ⁺ <i>metA gal lac xyl tsx</i>	Transduction of AB1911
MPE 1	Hfr	<i>his malB str</i>	M. SCHWARTZ
P10	Hfr	<i>thr leu str malB</i> λ^r	F. JACOB
PAM 201	Hfr	<i>his met</i> λ^s <i>malB</i>	Mutant (NG) of HfrC W1895 of J. LEDERBERG

Bacteriophages: All phage were grown on various hosts by the plate method (ADAMS 1959). P1*k*c was grown on strain B through three or more passages in order to increase the titer. Adsorption was generally high $\approx 90\%$. The phages used were: P1*k*c obtained from Dr. C. YANOFSKY, and λ *vir*, a virulent strain of phage λ obtained from Dr. D. KAISER.

Ultraviolet sensitivity: Two methods for assay of radiation sensitivity were employed, a long, quantitative method and a rapid, semiquantitative method. In the rapid method, strains to be assayed were incubated overnight in tryptone broth containing 1% tryptone, 0.5% NaCl and 0.5% yeast extract. Short streaks of culture were applied by capillary tube to tryptone agar containing tryptone 1%, NaCl 0.5% and BBL (Baltimore Biological Laboratories, Baltimore, Md.) agar 1.5%. Plates were irradiated with ultraviolet light from a 10 W General Electric germicidal lamp with a maximum output at 2537 Å, at a distance of 51.5 cm from the source. The lamp delivered 15.4 ergs/mm²/sec. Plates irradiated for 10 and 30 sec were usually sufficient to

FIGURE 1.—The appearance of streaks of cultures of seven strains of *E. coli* following irradiation for various intervals of time and incubation for 18 hours at 37°C.

distinguish resistant and sensitive clones. Plates were incubated approximately 18 hours and examined for the appearance of the streaks (Figure 1).

Definitive UV survival curves were obtained as follows: cultures of strains to be examined were grown overnight in tryptone broth. Appropriate dilutions in phosphate buffer (0.2 M) containing 0.8% NaCl were spread on tryptone agar plates and irradiated with UV as above. The colonies were counted after incubation for 24 hours. During and subsequent to irradiation plates were kept in subdued light. The survival curves of several pertinent strains treated in this way are shown in Figure 2.

Transduction: Transduction was performed as described by LENNOX (1955). Recipient bacteria were grown overnight in tryptone broth and diluted 1:25 in 5 ml of fresh tryptone broth, incubated and shaken at 130 strokes/minute in a water bath until log phase with a count of approximately 5×10^8 viable cells/ml was reached. Cells were centrifuged, washed and resuspended in sterile H_2O containing 2.5×10^{-3} M $CaCl_2$. Phage were added to the cell suspension at a multiplicity of 0.5 to 1.0. The mixture of cells and phage were incubated with gentle shaking for 20 minutes to allow phage adsorption. The mixture was centrifuged, the cells concentrated 10-fold, spread on suitable agar for selection and incubated for 3-5 days. The supernatant after centrifugation was assayed for adsorption. Three controls were always used: (1) Transducing lysates were spotted onto a plate of complete medium to test for sterility. (2) Cells without phage were spread onto selective medium to assay for reversions. (3) Viable count of bacteria after adsorption of phage was determined.

Selection of prototrophic transductants was done by plating on appropriately supplemented Davis minimal (DM) medium (Difco). Mal^+ transductants were selected on DM medium with

FIGURE 2.—Survival following exposure to UV of parental strains used in this paper. Symbols: ○ AB1886; ▼ Bs2; ● B; ▲ PAM 201; ○ AB1911; △ P10; □ MPE.

maltose as carbon source. As an unselected marker *mal* was assayed on BTB (brom thymol blue, Difco) to which was added agar at 1.5% and the test sugar at 1%. Sensitivity to λ phage was done by streaking overnight cultures across a streak of λ *vir* grown on the appropriate strain to eliminate problems of restriction and modification, i.e., B recipients were cross-streaked against λ *vir* grown on strain B251; K-12's against λ *vir* grown on K-12.

Host cell reactivation: This method was as described by GREENBERG (1967).

RESULTS AND DISCUSSION

Cotransduction of malB and uvr2: Data from recombination experiments (GREENBERG 1964b) suggested that the gene responsible for UV sensitivity in strain Bs2 is in the *malB* region. Phage P1 grown in strain B251 (P1·B251) was used to transduce *mal*⁺ to strain Bs2. It should be recalled from Figure 1 that B251 is resistant to UV relative to Bs2. Eighty-eight per cent of the 137 transductants were resistant to UV, i.e., irradiated streaks of overnight cultures and their survival curves following UV radiation resembled those of strain B251 (Figure 3, compare Figure 2). All of the Mal⁺ transductants were sensitive to λ *vir*:B251, demonstrating that resistance to λ was a pleiotropic effect of the *mal*

FIGURE 3.—Survival following exposure to UV of transductants between P-B251 and Bs2 selected for Mal⁺. Each closed circle represents reading of a single transductant and open circle represents B251 parental type.

marker. Similar experiments with strain K-12 donors W1895 and AB1911 confirmed these data; all the Mal transductants were λ sensitive and 82% and 90%, respectively, were as UV resistant as strain B251.

There were now available Mal⁺ derivatives of strain Bs2 which were still UV sensitive (e.g. PAM 206). When P1·PAM 206 was used to transduce mal⁺ to strain B, 78% of the transductants were as sensitive to UV as strain Bs2. Therefore, whether strain Bs2 was donor or recipient, the cotransductional frequency of mal and uvr2 was high.

The malB region of *E. coli* is complex in that there are known to be alleles which are malB λ^s and malB λ^r (SCHWARTZ 1966). Strain MPE is a strain with a malB λ^s allele and P10 is a strain with a malB λ^r allele. A similar dualism of mal λ^r and λ^s alleles exists at the malA locus (LEDERBERG 1955). *E. coli* B is unable to ferment maltose and is λ^r . ARBER and LATASTE-DOROLLE (1961) were able to cotransduce λ sensitivity with mal⁺ from strain K-12 to B, indicating that these properties were pleiotropic. The mal marker of strain B was analogous, at least, to that of strain P10. We, therefore, wanted to know if the uvr2 gene was cotransducible with malB⁻ λ^s . For this we had available PAM 206, which is a Mal⁺ transductant of Bs2 in which uvr2 was conserved. We were now looking for the cotransduction to Mal⁺ and UV sensitivity of the malB λ^s strain, MPE. However, it was not possible to transduce Mal⁺ from PAM 206 to strain MPE though transduction of outside markers such as His⁺, Thr⁺ and Leu⁺ was possible. malB⁺ could not be transduced to P10 (malB λ^r) either. However, PAM 201 (malB λ^s) could be transduced to Mal⁺ by P1·PAM 206, and 14% of the 230 Mal⁺ transductants were as sensitive to UV as strain Bs2. This suggested that uvr2 was more closely linked to malB λ^r (of strain B) than to malB λ^s (of strain PAM 201). However, other explanations for the differences in linkages of uvr2 to malB λ^s and malB λ^r can be entertained. The point remains, however, that uvr2 is linked to malB λ^s and to the mal λ^r of strain B.

Cotransduction of malB and uvrA: P1·AB1886 (uvrA) was used to transduce mal to strain B. Fifty per cent of the transductants were as sensitive to UV radiation as the donor (Figures 4 and 5) and all the UV sensitives were HCR. The P1 mediated cotransductional linkage of uvrA and the malB λ^s locus of MPE reported by SCHWARTZ (1966) was 70%.

P1·AB1886 was also used to transduce mal⁺ to PAM 201. Six per cent of the 200 mal⁺ transductants were UV sensitive (Figure 4).

SCHWARTZ (1966) had shown that it was possible to identify λ^r and λ^s mal mutants in both the malA and malB regions. He showed, furthermore, that uvrA was linked to malB λ^s (strain MPE) and not to malA. We have shown that uvrA is linked to the mal marker of strain B, which is, therefore, malB. However, since it is λ^r it is more like the malB marker of strain P10. Furthermore, uvr2 and uvrA were both linked to malB λ^s of PAM 201 and, based on cotransductional frequencies, uvr2 was closer to malB λ^s and malB λ^r than was uvrA, but both uvrA and uvr2 appeared to be more closely linked to malB λ^r than to malB λ^s of PAM 201. These data did not allow precise ordering of markers because the orders uvr2 malB uvrA and malB uvr2 uvrA were both possible.

FIGURE 4.—UV sensitivity of transductants between P1-AB1886 and PAM 201 and B selected for *Mal*⁺. Open circle represents UV sensitive transductants between P1-AB1886 and B. Open triangle represents UV sensitive transductants between P1-AB1886 and PAM 201. Symbols: ■ AB1886; ● B; ▲ PAM 201.

Cotransduction of metA uvr2 and malB: A three-point test would provide more reliable evidence of the order of markers. *uvrA* was known to be linked not only to *malB* but also to *metA* (HOWARD-FLANDERS, BOYCE and THERIOT 1966; SCHWARTZ 1966). The linkage of *uvr2* to *metA* was examined by using P1-Bs2 to transduce *metA* to AB1911. As seen in Table 2, UV sensitivity as well as *arg*⁺

TABLE 2
Linkage of arg, metA, malB and uvr2

Selected markers	Number of transductants scored	<i>met</i> ⁺	Frequency in percent of unselected donor markers			
			<i>arg</i> ⁺	<i>mal</i>	<i>uvr2</i>	<i>mal uvr2</i>
<i>arg</i> ⁺	100	18	..	0	0	0
<i>met</i> ⁺	150	..	30	2.8	2.8	0

The donor was P1-Bs2 (*mal*⁺ *uvr2*) and the recipient was AB1911 (*metA arg*).

FIGURE 5.—The appearance following UV radiation of streaks of *Mal*⁺ transductants between P1-AB1886 and B.

and *mal* were transduced with *metA*⁺, but none of the *Arg*⁺ transductants were either *Mal* or UV sensitive. In transductions in which *Arg*⁺ was selected, 18% were *Met*⁺ and none were *Mal* or UV sensitive. These results were confirmed in experiments using P1-Bs2 to transduce *metA*⁺ to PAM 202 (Table 3). Of the *Met*⁺ transductants 6.7% were *Mal* and 3.9% were UV sensitive. All the *Mal* transductants were λ^r . All the UV sensitive transductants were *Mal* but not all *Mal* transductants were UV sensitive. These transductions established the linkage of *metA* and the *malB* locus of strain B, confirmed the close linkage of *malB* and *uvr2*, and established the probable order of genes as *arg metA malB uvr2*.

Cotransduction of *uvrA* *metA* and *malB*: P1-AB1886 was used to transduce *metA*⁺ to AB1911. UV sensitivity was found in 7.2% of the 150 *Met*⁺ transductants.

In order to perform a three-point test it was necessary to derive a strain in which *metA* or *malB* were present with *uvrA*. *metA* was put into AB1886 with P1-PAM 202 (the *arg*⁺ transductant of AB1911, *metA* conserved) by transduc-

TABLE 3
*Ordering of metA, malB and uvr2 by three factor crosses**

Number of <i>met</i> ⁺ transductants scored	Frequency in percent of donor markers					
	<i>mal</i>	<i>uvr2</i>	<i>uvr2</i> ⁺	<i>mal</i> ⁺	<i>uvr2</i>	<i>uvr2</i> ⁺ <i>mal</i> <i>uvr2</i>
963	6.7	4.0	93.3	0	2.7	4.0

* The donor was P1-Bs2 (*mal* λ^r *uvr2*) and the recipient was PAM 202 (*metA* *arg*⁺).

TABLE 4

*Ordering of metA, malB, and uvrA by three factor crosses**

Donor strains	Number of <i>met</i> ⁺ transductants scored	Frequency in percent of donor markers					
		<i>mal</i>	<i>uvrA</i> ⁺	<i>uvrA</i> ⁺	Mal ⁺ <i>uvrA</i>	<i>uvrA</i> ⁺	Mal <i>uvrA</i>
B <i>malB</i> λ ^r	150	6.0	4.0	0	93.3	4	2.0
P10 <i>malB</i> λ ^r	475	13.7	9.5	3.8	82.5	5.7	8.0
PAM 201 <i>malB</i> λ ^s	110	6.3	4.5	2.7	91	1.8	4.5
MPE <i>malB</i> λ ^s	258	11.2	5.4	0.4	88.4	5.0	6.2

* The phage lysates were prepared from B, P10, PAM 201, MPE and the recipient was PAM 207 (*uvrA metA*).

ing *arg*⁺. None of the *Arg*⁺ transductants were UV resistant, but 18% were *metA*.

One of these *metA* (*uvrA* conserved) transductants of AB1886 (PAM 207) was used as a recipient for *metA*⁺ using each of four *malB* strains; B, MPE, P10 and PAM 201. Results of these transductions are shown in Table 4. In each instance the *malB* marker was transferred more frequently than *uvrA*⁺. It was also clear that *uvrA* and all the *malB* genes were closely linked. The linkage of *metA* to *malB*, λ^s or λ^r, was similar, or, at least, there was no clear difference in linkage to either *malB* λ^r or *malB* λ^s. The data, therefore, did not permit the definite establishment of the order of the *malB* λ^r or λ^s markers relative to *metA*, nor was there any unequivocal evidence that there were two *malB* cistrons.

There were now available four different *malB uvrA* strains each with a different source of the *malB* gene: PAM 203 from strain B, PAM 204 from MPE, PAM 205 from 201, and PAM 211 from P10. However, none of these were transducible for *mal*⁺ with any donor, though absorption of P1 was normal and transduction of the outside markers in these derivatives was possible. The *mal* marker and *uvrA* could, however, be transduced from them to AB1911 when *Met*⁺ transductants were selected (Table 5). The frequency of the unselected marker, *malB*, in these recipients was higher than in our previous experience. However, as previously observed, *malB* was more frequently transduced with *metA* than was

TABLE 5

*Ordering of metA, malB and uvrA by three factor crosses**

Donor strains†	Number of <i>met</i> ⁺ transductants scored	Frequency in percent of donor markers					
		<i>mal</i>	<i>uvrA</i>	<i>uvrA</i> ⁺	Mal ⁺ <i>uvrA</i>	<i>uvrA</i> ⁺	Mal <i>uvrA</i>
PAM 203 λ ^r	1060	11.8	8.3	88.2	0	3.5	8.3
PAM 204 λ ^s	100	16.0	6.0	83.0	1.0	11.0	5.0
PAM 205 λ ^s	156	12.8	6.4	87.2	0	6.4	6.4
PAM 212 λ ^r	401	12.7	3.4	87.3	0	9.3	3.4

* The recipient was AB1911 (*metA arg*).

† All *uvrA malB* derivatives of AB1886.

uvrA, but it was still not possible to decide an order of *malB* markers, λ^r and λ^s , relative to *metA*.

From these transductions there were now available derivatives of AB1911 which carried *uvrA* and also one or another *malB* marker. PAM 208 (*uvrA malB* λ^r), PAM 209 (*uvrA malB* λ^s), PAM 210 (*uvrA malB* λ^s) and PAM 212 (*uvrA malB* λ^r) were obtained, respectively, from PAM 203, PAM 204, PAM 205 and PAM 211. These were transducible for *Mal*⁺, using P1·B251, with results shown in Table 6. Linkage of *uvrA* to *malB* λ^r was 28 to 40%, to *malB* λ^s 9 to 12%. These results are the reciprocal of those obtained earlier in which *uvrA* was transduced from AB1886 at a frequency of 50% with *malB* λ^r and 6% with *malB* λ^s . It is evident that *uvrA* lies to the right of both *malB* λ^s and *malB* λ^r , relative to *metA*, and appears to be more closely linked to *malB* λ^s than *malB* λ^r . This was also true, as shown earlier, of *uvr2*. These data would seem to imply the order of

markers is *metA malB* $\lambda^s malB$ λ^r [*uvrA*]. However, this could not be confirmed by analysis of frequency of cotransductants of *metA* with *malB* λ^r and *malB* λ^s , both of which appeared equally linked to *metA*.

Transduction of UV resistance from AB1886 to Bs2: When P1·AB1886 was used to transduce *mal*⁺ to Bs2, 26% of the 214 transductants were UV resistant; i.e., their UV survival curves resembled that of strain B (Figure 6). The reciprocal transduction could not be done because, as mentioned above, *mal*⁺ could not be transduced into *mal* derivatives of AB1886 whether λ^s or λ^r . The *mal*⁺ marker was transducible into PAM 208 (*malB* $\lambda^r uvrA) by P1·PAM 206 and none of 300 transductants was UV resistant. These results are compatible with the order suggested in the previous section, namely *malB uvr2 uvrA* because with this order of markers and Bs2 as donor, an extra crossover would be required for UV resistance.$

Taken together the data in this report reconcile genetic lines of evidence from several sources. They suggest that the order of markers in the region is *arg metA* (*malB* $\lambda^s malB$ λ^r) *uvr2 uvrA* (Figure 7). On the genetic evidence and phenotypic differences *uvr2* and *uvrA* are mutations in two different but closely linked cistrons. This suggests the possibility that together these two cistrons form an operon concerned with repair after UV damage, with the *uvrA* cistron effecting

TABLE 6
Ordering of malB λ^r and *malB* λ^s *

Recipient strains†	Number of <i>Mal</i> ⁺ transductants scored	Frequency of <i>uvrA</i> ⁺ in percent
PAM 208	150	40
PAM 209	150	9
PAM 210	200	12
PAM 212	180	28

* The donor was P1·B251 (*mal*⁺ λ^s).

† All *uvr malB* derivatives P1·B251 of AB1911.

FIGURE 6.—UV survival of transductants between P1-AB1886 and Bs2. Symbols: ■ AB1886; ○ B, ● transductants.

deletion of thymine dimers in UV damaged regions (HOWARD-FLANDERS, BOYCE and THERIOT 1966) and a cistron concerned with repair replication, as suggested by HOWARD-FLANDERS and BOYCE (1966). The relationship of such an operon with other UV genes such as *uvrB* and *uvrC*, both of which are concerned with

FIGURE 7.—Genetic map of the *malB* region of the *E. coli* chromosome. The distances are given in terms of cotransduction frequencies, using phage P1.

deletion of UV-induced thymine dimers, is not clear. However, it is noteworthy that at each linkage group, *uvrA* and *malB*, *uvrB* and *his*, *uvrC* and *gal*, there is reported to be one or more genes for UV sensitivity which produce an HCR⁺ phenotype (VAN DE PUTTE, VAN SLUIS, VAN DILLEWIJIN and RÖRSCH 1965). The *uvr2* gene is epistatic to the *lon* (or *fil*) gene of strain B; i.e., if the *uvr2* gene is inoperative (sensitive) (GREENBERG 1964b; DONCH and GREENBERG, unpublished), the condition of the *lon* gene has no discernible effect on UV sensitivity. Furthermore, if the *uvr2* gene is inoperative (UV sensitive), no filaments are induced by UV regardless of the state of the *lon* gene. This suggests that *uvr2* and *lon* represent genes in a common sequential pathway, the *uvr2* function occurring earlier.

SUMMARY

The gene (*uvr2*) responsible for radiation sensitivity of strain Bs2 is linked to both *malB* and *metA*, as shown by transduction with P1. The order of markers is *metA malB uvr2*. The gene *uvrA* was found in the same region of the chromosome, confirming earlier work of SCHWARTZ. *uvr2* appears to be closer to *malB* than is *uvrA* and pseudo-reciprocal crosses indicate that the order of markers is *metA malB uvr2 uvrA*. Some, but not all, Mal mutants in the *malB* region are pleiotropic with λ resistance, again confirming earlier data. The results suggest, but do not prove, that there may be two different *malB* cistrons, the *malB* λ' cistron being more closely linked to the UV resistance genes in the region.

LITERATURE CITED

- ADAMS, M. H., 1959 *Bacteriophages*. Interscience Publishers, Inc., New York.
- ARBER, W. and C. LATAS-TE-DOROLLE, 1961 Erweiterung des Wirtsbereiches des Bakteriophagen λ auf *Escherichia coli* B. Pathol. Microbiol. **24**: 1012-1018.
- DEMEREK, M., E. A. ADELBURG, A. J. CLARK, and P. E. HARTMAN, 1966 A proposal for a uniform nomenclature in bacterial genetics. Genetics **54**: 61-76.
- GREENBERG, J., 1964a A locus for radiation resistance in *Escherichia coli*. Genetics **49**: 771-778.
- 1964b A locus for radiation sensitivity in *Escherichia coli* B_{s-2}. Genetics **50**: 639-648.
- 1965 Locus for radiation sensitivity in *Escherichia coli* B_{III}. Mutation Res. **2**: 297-303.
- 1967 Loci for radiation sensitivity in *Escherichia coli* strain B_{s-1}. Genetics **55**: 193-201.
- HILL, R. F., and E. SIMSON, 1961 A study of radiosensitive and radioresistant mutants of *Escherichia coli* strain B. J. Gen. Microbiol. **24**: 1-14.
- HOWARD-FLANDERS, P., R. P. BOYCE, and L. THERIOT, 1966 Three loci in *Escherichia coli* that control the excision of pyrimidine dimers and certain other mutagen products from DNA. Genetics **53**: 1119-1136.
- HOWARD-FLANDERS, P., and R. P. BOYCE, 1966 DNA repair and genetic recombination: studies on mutants of *Escherichia coli* defective in these processes. Radiation Res. Suppl. **6**: 156-184.
- LEDERBERG, E. M., 1955 Pleiotropy for maltose fermentation and phage resistance in *Escherichia coli* K-12. Genetics **40**: 580-581.
- LENNOX, E. S., 1955 Transduction of linked genetic characters of the host by bacteriophage P1. Virology **1**: 190-206.

- MANDELL, J. D., and J. GREENBERG, 1960 A new chemical mutagen for bacteria, 1-methyl-3-nitro-1-nitrosoguanidine. *Biochem. Biophys. Res. Commun.* **3**: 575-577.
- MATTERN, I. E., H. ZWENK, and A. RÖRSCH, 1966 The genetic constitution of the radiation-sensitive mutant *Escherichia coli* B₈₋₁. *Mutation Res.* **3**: 374-380.
- RÖRSCH, A., A. EDELMAN, and J. A. COHEN, 1963 The gene-controlled radiation sensitivity in *Escherichia coli*. *Biochim. Biophys. Acta* **68**: 263-270.
- SCHWARTZ, M., 1966 Location of the maltose A and B loci on the genetic map of *Escherichia coli*. *J. Bacteriol.* **92**: 1083-1089.
- VAN DE PUTTE, P., C. A. VAN SLUIS, J. VAN DILLEWIJN, and A. RÖRSCH, 1965 The location of genes controlling radiation sensitivity in *Escherichia coli*. *Mutation Res.* **2**: 97-110.