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Isolation and genetic characterization of a thymineless death-resistant mutant of *Escherichia coli* K12: Identification of a new mutation (*recQ1*) that blocks the RecF recombination pathway

Hiroaki Nakayama[1], Koji Nakayama[1], Ritsuko Nakayama[1], Nobuto Irino[1], Yasuko Nakayama[1]*, and Philip C. Hanawalt[2]

[1] Department of Microbiology, School of Dentistry, Kyushu University, Fukuoka 812, Japan
[2] Department of Biological Sciences, Stanford University, Stanford, California 94305, USA

Summary. An *Escherichia coli* K12 mutant resistant to thymineless death (TLD) was isolated, and its genetic analysis led us to identify a new mutation (*recQ1*) located between *corA* and *metE* on the standard linkage map. The mutation was found to result in increased sensitivity to ultraviolet light and deficiency in conjugational recombination when placed in the *recBC sbcB* background, indicating that it blocked the RecF pathway of recombination. It seemed likely that this mutation is also capable of causing partial resistance to TLD, but we reserve the possibility of a separate mutation closely linked to *recQ1* giving rise to this phenotype. The original mutant was shown to carry an additional mutation probably in the vicinity of the *uhp* locus, which was also required for the full TLD resistance of the mutant to be expressed.

Introduction

Genetic data indicate that there are two distinct pathways of recombination operating in the bacterium *Escherichia coli*. One, called the RecBC pathway, is the major pathway in wild-type cells as assayed by a conjugational cross, and requires the functional $recA^+$ as well as $recBC^+$ genes. The residual recombination activity exhibited by *recB* or *recC* mutants in such a cross, around 1% of the wild-type level, has been attributed to the other pathway, the RecF pathway, in which the $recF^+$ and $recJ^+$ genes (Horii and Clark 1973; Lovett and Clark 1983) and the wild-type allele for *rec-259* (Lloyd et al. 1983) are involved in addition to $recA^+$. This latter pathway is thought to be activated by a mutation in the *sbcB* locus specifying exonuclease I, thus accounting for the nearly normal recombination proficiency seen in *recBC sbcB* mutants (Barbour et al. 1970). Recent work has also shown that the RecF pathway is DNA damage-inducible (Lovett and Clark 1983).

The genes of the RecF pathway also seem to be involved in other processes that may or may not be related to recombination. For example, *recF* and *recJ* mutations reduce or abolish repair of single-strand gaps in postreplication repair and ultraviolet(UV)-induced reactivation of UV-irradiated phages (see Lovett and Clark 1983). A *recF* mutation also affects UV-induction of λ prophage (Armengod and Blanco 1978) and makes cells more resistant than wild type to thymineless death (TLD) (Nakayama et al. 1982). This last finding is of interest because the mechanism for the lethal process (Cohen and Barner 1954) is still only poorly understood.

As mentioned earlier, three loci have so far been identified as being involved in the RecF pathway: *recF*, *recJ*, and *rec-259* (Lovett and Clark 1983; Lloyd et al. 1983). In the present paper, we describe the identification of a new mutation, *recQ1*, that blocks this pathway. The mutation was initially found in an *E. coli* mutant HN15, isolated in this study as a strain unusually resistant to TLD. Although the original mutant was normal with respect to UV sensitivity and recombination proficiency, the *recQ1* mutation made cells recombination-deficient and UV-sensitive when transferred into the *recBC sbcB* background. We also consider that this mutation is very likely to be responsible for the TLD resistance as well, which seems to corroborate a significant role played by some RecF-related function in TLD.

Materials and methods

Bacterial strains and plasmids. These are listed in Table 1.

Culture media. The minimal medium used in all experiments involving thymine starvation or nutritional selection in genetic crosses was Tris-minimal medium (see Nakayama and Couch 1973). Supplements were added as needed to this medium at 1 μg/ml (thiamine), 2 μg/ml (thymine in liquid medium), 4 μg/ml (thymine in plates) or 20 μg/ml (amino acids). For plates agar (Wako Chemical Co., Osaka, Japan) was added to 1.5%. The buffered salts medium base was Tris-minimal medium less glucose and disodium hydrogen phosphate. Nutrient agar, nutrient soft agar (Nakayama et al. 1982), L-broth, and L-agar (Nakayama et al. 1983) were as described except that yeast extract (Difco, Detroit, USA) was replaced by that of BBL (Cockeysville, USA). P1 broth was also described (Oeda et al. 1981). Streptomycin and kanamycin were used at 200 and 30 μg/ml, respectively.

Thymine starvation and determination of TLD sensitivity. This was carried out as described previously (Nakayama

Offprint requests to: H. Nakayama

* On leave of absence from Medical College of Miyazaki

Abbreviations: UV, ultraviolet light; NG, N-methyl-N′-nitro-N-nitrosoguanidine; Km, kanamycin; Sm, streptomycin; TLD, thymineless death; ^{r}, resistant; ^{s}, sensitive

Table 1. Bacterial strains and plasmids used

Designation	Genetic markers	Derivation/source/reference
Bacterial strain		
AB2497	F$^-$ *thr-1 leu-6 argE3 his-4 proA2 thi-1 thyA12 thyR14 lacY1 galK2 ara-14 xyl-5 mtl-1 rpsL31 tsx-53 sup-37*	Bachmann (1972)
AT2471	HfrH *tyrA thi relA spoT*	Obtained from B. Bachmann
CGSC4318	Hfr(AB313) *thi-1 leu-6 gal-6 lacY1* or *lacZ4 sup-54*	Obtained from B. Bachmann
HN15	As AB2497 but *tld-1* (*recQ1*) *tld-2*	See text
HN39	As HN15 but *thyA*$^+$	JC5029 (Bachmann 1972) × HN15, Thy$^+$ selection
HN71	HfrC *metB1 rel-1 thyA thyR*	This laboratory
HN219	As HN15 but *ilv*	UV mutagenesis → penicillin screening
JG77	Hfr(R1) *rha rpoB*	Obtained from A. Ganesan
KD2046	As AB2497 but *uvrD252* (*recL152*)	P1 [JC8471 (Horii and Clark 1973)] × KD2110, Ilv$^+$ selection
KD2110	As AB2497 but *ilv*	This laboratory
KD2157	F$^-$ *ilv-145 metE46 his-4 trpC3 pro thi mtl-1 malA1 ara-9 galK2 lac-114 rpsL ton thyA*::Tn*5 thyR*	This laboratory; a derivative of AB2277
KD2168	F$^-$ *rha thyR lac rpsL polA12 ilv-2168*::Tn*5*	This laboratory
KD2169	As AB2497[a] but *ilv-2168*::Tn*5 recB22 recC21 sbcB15*	This laboratory; a derivative of JC7623 (Kushner et al. 1972)
KD2170	As KD2157 but *metE*$^+$ *tld-1* (*recQ1*)	P1(HN15) × KD2157, Met$^+$ selection
KD2171	As KD2157 but *metE*$^+$	P1(HN15) × KD2157, Met$^+$ selection
KD2172	As KD2157 but *metE*$^+$ *ilv*$^+$ *tld-1* (*recQ1*)	P1(HN15) × KD2157, Met$^+$ selection
KD2173	As KD2157 but *metE*$^+$ *ilv*$^+$	P1(HN15) × KD2157, Met$^+$ selection
KD2179	As KD2169 but *ilv*$^+$ *tld-1* (*recQ1*)	P1(HN15) × KD2169, Ilv$^+$ selection
KD2180	As KD2169 but *ilv*$^+$	P1(HN15) × KD2169, Ilv$^+$ selection
KD2184	As HN15 but *ilv-2168*::Tn*5*	P1(KD2168) × HN15, Km^r selection
KD2185	As KD2169 but *tld-1* (*recQ1*)	P1(KD2184) × KD2179, Km^r selection
KD2189	As AB2497 but *ilv-2168*::Tn*5*	P1(KD2168) × AB2497, Km^r selection
KD2191	As KD2169 but *metE70*	P1[χ478(Berg and Curtiss 1967)] × KD2169 → *ilv*$^+$ *metE* transductant (KD2190) → P1(KD2168) × KD2190, Km^r selection
KD2196	As AB2497 but *tld-1* (*recQ1*)	P1(HN15) × KD2189, Ilv$^+$ selection
KD2198	As KD2169 but *uvrD252* (*recL152*)	P1(KD2046) × KD2191, Met$^+$ selection
KD2199	As KD2169 but *corA5738*	P1[KO21(Oeda et al. 1981)] × KD2191, Met$^+$ selection
KP77	*metB trp his lac gal tsx*	Obtained from T. Miki
N14-4	F$^-$ *trp gal rpsL uvrD3*	Ogawa et al. (1968)
Plasmid		
F13-1	F′ *lac*$^+$	see Maki et al. (1983)
pKD1	pKP1103 (MiniF[b]) *uvrD*$^+$ *corA*$^+$	Nakayama et al. (1983)
shF111	F′ *ilv*$^+$ *metE*$^+$	Hiraga (1976)
shF8-5	F′ *ilv*$^+$ *uhp*$^+$ *Δ(rbs asn bglB tna dnaA)*	Hiraga (personal communication)

Strains carrying shF111 or shF8-5 were constructed by mating with SH250/shF111 or SH250/shF8-5 (Hiraga 1976), whereas those harboring pKD1 were made by transformation with pKD1 DNA by the method of Norgard et al. (1978)

[a] The *thyA* allele of this strain is different from *thyA12*

[b] Containing the 44.35–49.40 kb region of the F genome and lacking all *tra* genes (T. Miki, personal communication)

et al. 1982). TLD sensitivity of a strain was routinely determined by the survival level after 4 h of thymine starvation at 37° C. In this article, the normal, wild-type sensitivity to TLD is referred to as TLD-sensitive.

Mutagenesis and enrichment procedures for the isolation of the TLD-resistant mutant. Late exponential phase cells of AB2497 harvested from 5 ml culture in supplemented Tris-minimal medium were treated with 100 μg N-methyl-N′-nitro-N-nitrosoguanidine (NG) per ml for 30 min at 37° C according to the method of Adelberg et al. (1965). The treated cells were washed once with buffered salts medium base, resuspended in 100 ml supplemented Tris-minimal medium, and allowed to grow at 37° C overnight with shaking. A 2 ml portion of this overnight culture was diluted into 20 ml fresh supplemented Tris-minimal medium and the culture was shaken at 37° C until a cell density of approximately 5×10^7/ml was reached. The medium was changed to 20 ml thymine-free medium by filtration (Nakayama et al. 1982) and starvation was imposed for 4 h

at 37° C with shaking. The cells were then collected by centrifugation, resuspended in 40 ml supplemented Tris-minimal medium containing thymine, and allowed to grow overnight again by shaking at 37° C. The cycle of outgrowth/thymine starvation/overnight culture was repeated nine more times in essentially the same manner.

Determination of UV sensitivity. Exponential phase cultures in L-broth were diluted appropriately in buffered salts medium base and spread on L-agar plates. The plates were irradiated with various doses of UV and colonies were scored after incubation at 37° C for 24 h. In the present article, the UV sensitivity of a wild-type strain (including a *recBC sbcB* mutant) is referred to as UV-resistant.

Other methods. Bacterial conjugation and transduction with P1 *vir* phage were performed by the standard procedures (Miller 1972) unless specifically noted. The plate mating technique employed for rough estimation of recombination proficiency was as follows. An overnight culture of the donor strain in L-broth was harvested and resuspended in the original volume of buffered salts medium base. A 5 ml portion of this suspension was poured onto a selective plate and liquid was removed by pipetting. After drying, 5 µl each was spotted of overnight L-broth cultures of the recipient strains to be tested. Those giving rise to only a few or no colonies after incubation at 37° C for 2 days were regarded as Rec^-. The presence or absence of the *corA* mutation was determined as described by Oeda et al. (1981).

Results

Isolation and preliminary characterization of TLD-resistant mutant HN15

In an approach to understanding the mechanism for TLD, we attempted to isolate *E. coli* mutants resistant to the lethal phenomenon. NG-treated cells of strain AB2497 were subjected to cycles of thymine starvation, each followed by overnight growth of survivors, as described in Materials and methods. After the tenth cycle, the population was found to consist mostly, if not entirely, of TLD-resistant cells: five randomly selected clones all turned out to be similarly resistant as represented by one of them, HN15 (Fig. 1). This strain was exclusively used in subsequent studies.

One trivial possibility was that a newly introduced mutation had a weak capacity somehow to suppress the *thyA12* mutation, thus causing leakiness in the thymine deficiency. This was ruled out by examining derivatives of HN15 in which the original *thyA12* allele was replaced by new ones: 17 independent *thyA* derivatives including one Tn*5* insertion mutant were isolated from HN39, a $thyA^+$ derivative of HN15, and shown to be as resistant to TLD as HN15 (data not shown). It was also possible that the size of the thymine nucleotide pool was expanded in HN15 in some unknown way, but no direct attempt was made to test this hypothesis. The sensitivity of HN15 to UV was indistinguishable from that of the parent AB2497 (data not shown). The strain was Rec^+ as judged by the efficiency of recombinant formation in Hfr mating and P1 transduction (data not shown).

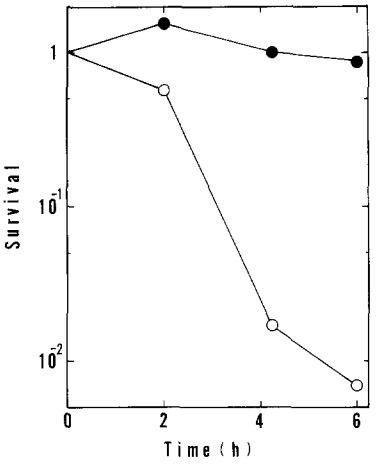

Fig. 1. Thymineless death in TLD-resistant mutant HN15 and its parent AB2497. Symbols: ○, AB2497; ●, HN15

Preliminary mapping of the mutations responsible for the TLD resistance in HN15

Data from Hfr mating experiments suggested that the mutation(s) responsible for the TLD resistance was located in the *xyl-ilv-argE* region of the *E. coli* chromosome. Thus, when HN15 was mated with Hfr(AB313) or Hfr(R1) and $Xyl^+[Sm^r]$ or $Arg^+[Sm^r]$ recombinants were selected, 40% or 88% of the recombinants, respectively, showed higher sensitivities to TLD than HN15. If there is an overlap between the proximal regions of these two Hfr's as documented in the literature (see Low 1972), the simplest possibility was that a single mutation in the overlapping segment, which should be located counterclockwise to *ilv*, could account for the TLD resistance phenotype. However, the results of P1 transduction shown in Table 2 indicated otherwise: At least one mutant locus between *ilv* and *metE* and close to *uvrD* appeared to be required for the TLD resistance of HN15 to be fully manifested, and the presence of this mutation alone seemed to confer partial resistance on the bacterium. This also implies that HN15 has another mutant locus in the region corresponding to the proximal segment of AB313 Hfr and that the full TLD resistance of HN15 requires the presence of both mutations. The former mutation was provisionally designated *tld-1* and subjected to more detailed study. Figure 2 shows that KD2172, a transductant obtained in cross II of Table 2 and presumed to carry *tld-1* but not the other mutation, was partially resistant to TLD as compared to KD2173, a TLD-sensitive counterpart, and the parent KD2157. KD2172 was UV-resistant and recombination-proficient (data not shown). The latter mutation, tentatively named *tld-2*, has not yet been characterized in detail except that it should be located in the vicinity of the *uhp* gene as judged from positive complementation by the F-prime factor shF8-5 (data not shown).

The tld-1 mutation may affect DNA repair and the RecF pathway of recombination

In the light of our previous finding that *recF* mutants are TLD-resistant (Nakayama et al. 1982), we attempted to introduce the *tld-1* mutation into the *recBC sbcB* background,

Table 2. Transductional linkage of the *tld-1* mutation

Cross	Donor	Recipient	Selected marker[a]		Unselected marker[a]		No. of transductants	
							TLD[s]	TLD[r]
I	N14-4[b]	HN219[c]	*ilv*$^+$	(10)	*uvrD*	(5)	5[d]	0
					uvrD$^+$	(5)	0	5
II	HN15[e]	KD2157[f]	*metE*$^+$	(120)	*ilv*$^+$	(74)	9	65[g]
					ilv	(46)	14	32[g]
III	HN15[e]	KD2157[f]	*ilv*$^+$	(120)	*metE*$^+$	(4)	0	4[g]
					metE	(116)	90	26[g]
IV	HN15[e]	KD2189[h]	*ilv*$^+$	(70)	–		59	11[g]

[a] Figures in the parentheses are the numbers of transductants
[b] TLD[s] *uvrD3*; a *uvrD3* strain is UV-sensitive, but not TLD-hypersensitive unlike the *mutU4* (Siegel 1973) and *recL152* (Nakayama et al., unpublished data) strains
[c] TLD[r] *ilv*
[d] Not fully sensitive (partially resistant) because of the presence of the *tld-2* mutation (see text)
[e] TLD[r]
[f] TLD[s] *ilv metE*
[g] Not as resistant as HN15 because of the absence of the *tld-2* mutation (see text and Fig. 2)
[h] TLD[s] *ilv*

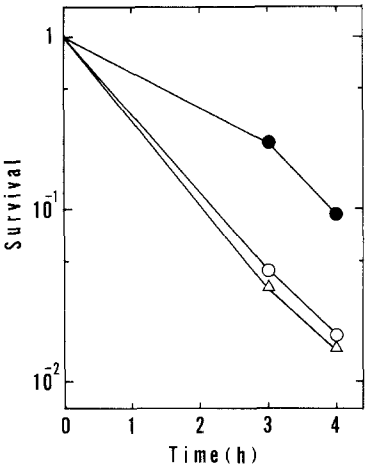

Fig. 2. Thymineless death in the transductants derived from the cross HN15 × KD2157. Symbols: ○, KD2157; ●, KD2172; △, KD2173

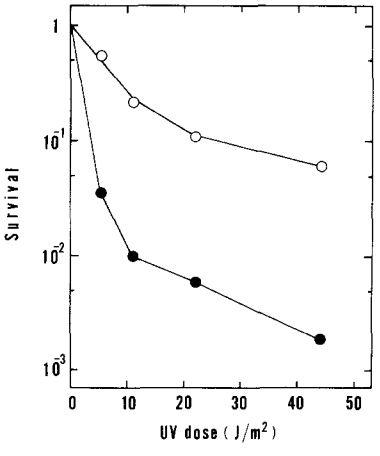

Fig. 3. Effect of the *tld-1* mutation on UV sensitivity in the *recBC sbcB* background. Symbols: ○, KD2180 (*recBC sbcB*); ●, KD2179 (*recBC sbcB tld-1*)

Table 3. Conjugational recombination proficiency of KD2179 harboring the *tld-1* mutation in the *recBC sbcB* background

Recipient	Relative recovery of recombinants or F-ductants per viable recipient		
	Donor		
	KP77/F13-1	HN71 (HfrC)	AT2471 (HfrH)
KD2180 (*recBC sbcB*)	1	1	1
KD2180/pKD1	–	0.15	–
KD2179 (*recBC sbcB tld-1*)	1.13	0.013	0.025
KD2179/pKD1	–	0.013	–

L-broth cultures (about 2×10^8 cells/ml) of the male and female strains were mixed in a ratio of 1:10 (for Hfr matings) or 1:1 (for F′ mating). Following incubation at 37° C for 60 min with gentle shaking, each mixture was plated out for Lac$^+$[Sm^r] F-ductants or Pro$^+$[Sm^r] recombinants after appropriate dilution

where the RecF pathway is thought to be the sole one at work for conjugational recombination (Horii and Clark 1973). When KD2169, a *recBC sbcB* strain, was transduced to Ilv$^+$ with HN15 as a donor, 5 out of 30 such transductants were found to be UV-sensitive and recombination-deficient. Figure 3 and Table 3 show the data for UV survival and recombinational ability, respectively, of KD2179 and KD2180 thus obtained. (Table 3 also shows that F-duction occurred at a normal rate in the Rec$^-$ transductant KD2179.)

The most crucial point was whether or not the mutation causing UV sensitivity and recombination deficiency was identical with *tld-1*, which was responsible for TLD resistance. Direct examination of those transductants for TLD sensitivity did not provide a reliable solution because of the hypersensitivity of *recBC*(*sbcB*) strains to TLD (Nakayama et al. 1982). Therefore, we carried out another se-

Table 4. Coincidence between TLD^r phenotype and UV^s Rec$^-$ phenotype (in the *recBC sbcB* background) in P1 transduction

Donor (*ilv*$^+$ *metE*$^+$)			No. of Ilv$^+$ Met$^+$ transductants			
No.	Derivation[a]	TLD	UV^{r}Rec$^+$	UV^{r}Rec$^-$	UV^{s}Rec$^+$	UV^{s}Rec$^-$
1	II	s	20	0	0	0
2	II	s	20	0	0	0
3	II	r	1	0	0	19
4	II	r	0	0	0	20
5	II	r	2	0	0	18
6	IV	s	20	0	0	0
7	IV	s	20	0	0	0
8	IV	r	1	0	0	19
9	IV	r	1	0	0	19

The recipient was KD2191 (*recBC sbcB ilv metE*). Twenty Ilv$^+$ Met$^+$ transductants from each cross were examined for UV sensitivity and recombination proficiency. The latter test was carried out by the plate mating technique

[a] The transductional crosses in Table 2

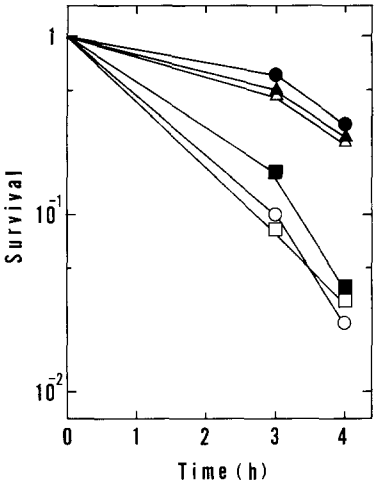

Fig. 4. Effects of the plasmids on TLD resistance conferred by the *tld-1* mutation. Symbols: ●, KD2170 (*tld-1*); ○, KD2170/shF111; ■, KD2171 (*tld*$^+$); □, KD2171/shF111; ▲, KD2172 (*tld-1*); △, KD2172/pKD1

ries of P1 transductions to answer this question. We randomly chose several TLD-sensitive and TLD-resistant strains among transductants from crosses II and IV of Table 2, and used them as donors to transduce KD2191 (*ilv metE recBC sbcB*) to Ilv$^+$ Met$^+$. Examination of resulting transductants revealed that TLD-resistant donors (*tld-1*) gave UV^s Rec$^-$ bacteria at high frequencies while TLD-sensitive donors (*tld*$^+$) yielded only UV^r Rec$^+$ ones (Table 4). This perfect coincidence does not prove but strongly suggests that the *tld-1* mutation responsible for the TLD resistance phenotype in fact caused the UV^s Rec$^-$ phenotype in the *recBC sbcB* background. With the reservation that TLD resistance and the UV^s Rec$^-$ phenotype might be due to closely linked but separate mutations, we tentatively assume that *tld-1* is involved in both phenotypes in the descriptions that follow.

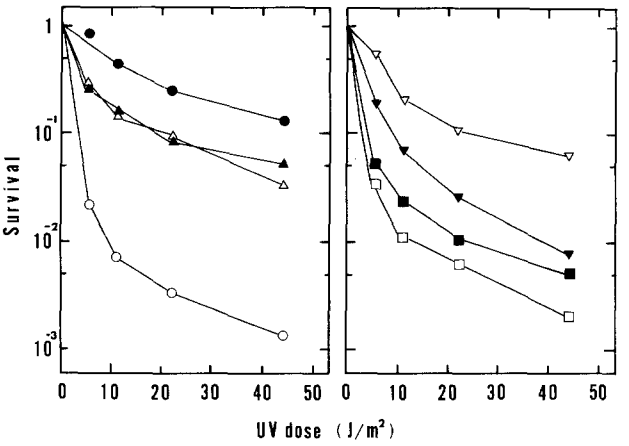

Fig. 5. Effects of the plasmids on the increased UV sensitivity conferred by the *tld-1* mutation in the *recBC sbcB* background. Symbols: ○, KD2185 (*tld-1*); ●, KD2185/shF111; △, KD2169 (*tld*$^+$); ▲, KD2169/shF111; □, KD2179 (*tld-1*); ■, KD2179/pKD1; ▽, KD2180 (*tld*$^+$); ▼, KD2180/pKD1

The tld-1 mutation is recessive

shF111, an F-prime factor carrying the *ilv-metE* segment of the *E. coli* chromosome (Hiraga 1976), was shown to be capable of complementing *tld-1*. First, KD2170 (*tld-1*) became TLD-sensitive in the presence of this plasmid, while KD2171 (*tld*$^+$) was little affected by it (Fig. 4). Second, KD2185 (*recBC sbcB tld-1*) was rendered UV-resistant by the plasmid, whereas it caused no significant changes in UV sensitivity in the *tld*$^+$ counterpart KD2169 (Fig. 5). (KD2185/shF111 was slightly more UV-resistant than KD2169, but we have presently no explanation for this.) These results, also corroborative of the transductionally determined location of *tld-1*, indicate that the mutant is recessive to wild type.

The tld-1 mutation is not an allele of the uvrD gene

The location of *tld-1* raised the possibility that the mutation might be an allele of the *uvrD* gene. However, several lines of evidence indicated that this is not the case. First, when KD2198, an *ilv recBC sbcB uvrD252* strain, was transduced to Ilv$^+$ with KD2196 carrying *tld-1* as donor, 27% of the transductants were found to be UV^r and Rec$^+$ (presumed genotype, *recBC sbcB uvrD*$^+$ *tld*$^+$). This strongly suggests that the *tld-1* mutation is located outside the *uvrD* locus; in similar crosses between different *uvrD* alleles, Kushner et al. (1978) found unusually low yields ($<1\%$) of wild-type recombinants. Furthermore, pKD1, a low copy number miniF plasmid harboring the *uvrD*$^+$ gene (Nakayama et al. 1983), failed to complement *tld-1* with respect to TLD sensitivity (Fig. 4).

On the other hand, results with *recBC sbcB* strains carrying pKD1 were rather equivocal. Apparently, pKD1 enhanced neither recombinant formation nor UV survival substantially in the *recBC sbcB tld-1* strain KD2179 (Table 3 and Fig. 5). In control experiments with the *recBC sbcB tld*$^+$ strain KD2180, however, both recombinant formation and UV survival were repressed by the plasmid (Table 3 and Fig. 5). The reason for this effect of pKD1 is at present unclear.

Table 5. Location of the *tld-1* mutation with respect to *corA* as determined by P1 transduction

Unselected marker		Distribution of Ilv^+ transductants
tld	*corA*	
−	+	18
−	−	0
+	+	14
+	−	68

Donor, KD2196 (*ilv*$^+$ *corA*$^+$ *tld-1*); recipient, KD2199 (*ilv corA tld*$^+$ *recBC sbcB*). Presence or absence of *tld-1* was judged by UV sensitivity

The tld-1 mutation is between corA and metE

Knowing that *tld-1* is distinct from *uvrD*, we went on to locate it with respect to a nearby marker *corA*. KD2199 (*recBC sbcB ilv corA*) was transduced to Ilv^+ with KD2196 carrying *tld-1* as a donor. The data presented in Table 5 clearly indicated that *tld-1* was located clockwise to *corA*. This location is consistent with the conclusion that *tld-1* is not an allele of the *uvrD* gene, and furthermore indicates that it is a hitherto unrecognized mutation, since no loci related to repair or recombination have been described in this segment of the chromosome.

Discussion

Genetic characterization of the TLD-resistant mutant HN15 isolated in this study revealed the presence of a mutation(s) which caused increased UV sensitivity and conjugational recombination deficiency in the *recBC sbcB* background as well as partial resistance to TLD. Based on our transductional data, we consider it highly likely that the single mutation heretofore referred to as *tld-1* is responsible for all of these phenotypes, but the possibility of very closely linked, separate mutations remains. Experiments including gene cloning now underway in the authors' laboratory may offer a definite answer to this problem. Regardless of this uncertainty, however, it can be stated safely that in HN15 there exists a mutation, between *corA* and *metE*, which blocks the RecF pathway of conjugational recombination, since hypersensitivity to UV and recombination deficiency manifested in the *recBC sbcB* background constitute the operational definition of a defective RecF pathway (Horii and Clark 1973). We should like to propose that this mutation be called *recQ1*.

Previous study has indicated that the *recF* mutants are significantly more resistant to TLD than the *recF*$^+$ bacterium (Nakayama et al. 1982). If the *recQ1* mutation is actually responsible for the resistance to TLD, it should corroborate the notion that the RecF recombination pathway per se or some functions related to it are involved somehow in the lethal process (Nakayama et al. 1982). In this connection, it would be interesting to look at whether the *recJ* and *rec-259* mutations also cause TLD resistance. It is hoped that further study of RecF-related functions would finally lead to the understanding of the mechanism for TLD.

recQ1 differs from the *recF* mutation in that the former does not confer increased UV sensitivity in a wild-type background while the latter does (Horii and Clark 1973). The *rec-259* mutation appears to be similar to *recQ1* in this respect (Lloyd et al. 1983). Apparently, the repair pathway for UV damage that is blocked by *recF* mutations in *recBC*$^+$ *sbcB*$^+$ cells does not involve the *recQ*$^+$ gene and the wild-type allele for *rec-259*. In other words, these results might be interpreted to mean that there should be two distinct mechanisms for RecF-related repair of UV-induced damage: one is operating in *rec*$^+$ cells without participation of the *recQ*$^+$ gene and the wild-type allele for *rec-259*, whereas the other is functioning only in *recBC sbcB* cells and does require these two genes.

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