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UGA Suppressor That Maps Within a Cluster of Ribosomal Protein Genes

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A suppressor of UGA mutations (*supU*) maps near or within a cluster of ribosomal protein genes at 72 min on the *Salmonella typhimurium* genetic map. The suppressor is relatively inefficient, and its activity is abolished by *rpsL* (formerly *strA*) mutations. The suppressor is dominant to a wild-type *supU* allele. The map position of this suppressor suggests that it may owe its activity to an alteration of ribosome structure.

Nonsense suppressors in bacteria cause the insertion of an amino acid in response to one of the three nonsense codons (UAG, UAA, or UGA). Nearly all nonsense suppressors are due to the production of an altered tRNA molecule that can recognize one of the nonsense codons, (for reviews see Smith [17] and Steege and Soll [18]). This paper describes a suppressor of UGA mutations in *Salmonella typhimurium* that maps in or near a cluster of genes encoding ribosomal proteins. The map position of this suppressor suggests that it may owe its activity to an alteration in ribosome structure.

MATERIALS AND METHODS

Bacterial strains. Multiply marked strains are listed in Table 1. All are derived from *S. typhimurium* LT2.

Media. The composition of all media has been described previously (6). Kanamycin sulfate was added to a final concentration of 50 μg/ml (rich media) or 125 μg/ml (minimal media). The concentrations of streptomycin sulfate and spectinomycin were 2 mg/ml and 400 μg/ml, in rich media.

Transductional crosses. All transductional crosses, mediated by phage P22, were done as previously described (6).

Construction of a strain with Tn*10* near *supU*. A strain carrying a Tn*10* element transductionally linked to *supU* was constructed as described by Kleckner, Roth, and Botstein (8) and Chumley et al. (3). Tn*10* was allowed to insert randomly into the chromosome of a strain (TR4768) carrying *supU1283* by the method of Kleckner et al. (7). About 1,000 to 5,000 independent insertion mutants were pooled; a phage P22 transducing lysate prepared on this mixed culture was then used to transduce a strain containing a *hisB* UGA mutation (TA389), selecting for inheritance of *supU* (His$^+$). Among the 10^3 to 10^4 His$^+$ transductants, two had simultaneously inherited a Tn*10* insertion (became tetracycline resistant). One of these strains, TT4029, proved to have a Tn*10* insertion 5 to 10% linked to *supU*. The nomenclature used for Tn*10* insertion mutations is described by Chumley et al. (3).

Hfr construction and mapping. The location of the Tn*10* insertion linked to *supU* was roughly mapped by Hfr crosses as described by Chumley et al. (3).

Segregation of duplications. Haploid segregants of strains carrying duplications of the 62- to 83-min region of the *Salmonella* chromosome were isolated as described by Anderson et al. (1). These duplication strains carry a *cysG*::Tn*5* insertion in one copy of the duplication; haploid segregants that have lost the *cysG*$^+$ gene form tiny colonies on minimal plates containing 0.08% (wt/vol) nutrient broth. These tiny colonies were picked and verified to be Cys$^-$ Kan^r, and therefore haploid, or homogenates.

Enzyme assays. The *hisB*-encoded enzyme, histidinol phosphate phosphatase, was assayed by the method of Martin et al. (10).

RESULTS

Isolation and characterization of the UGA suppressor mutant. The *supU1283* suppressor was isolated as a His$^+$ revertant of a strain (TR4765) containing the *hisB2135* (UGA) mutation. This strain also carries the *hisO1242* mutation. In an otherwise wild-type background, *hisO1242* causes constitutive operon expression and results in a rough colony morphology due to overproduction of the histidine biosynthetic enzymes (15). However, strain TR4765 (*hisO1242 hisB2135*) exhibits a smooth colony morphology because of the polar effect of the *hisB2135* mutation on operon expression (4). True revertants of the *hisB2135* mutation in TR4765 have a rough colony morphology; revertants containing external suppressors of *hisB2135* exhibit a smooth colony morphology because suppressors do not fully relieve the polarity of the *hisB* mutation. The suppressor mutation *supU1283* was found in a His$^+$ revertant of TR4765 that forms smooth colonies. We have designated the suppressor locus *supU*.

The suppressor in this revertant strain was shown to be specific for UGA mutations. A strain (TT609) carrying *supU1283* and a deletion of almost the entire *his* operon was used as recipient in a transductional cross with donor phage

TABLE 1. *Multiply marked bacterial strains*[a]

Strain	Genotype
TR692	*hisO1242 hisB2442 hisT1529 aroD5* (Roth, 1970)
TR2246	HfrB2 *recA1 rpsL1 metA22*
TR4765	*hisO1242 hisD6592*(Ts) *hisB2135*
TR4768	*hisO1242 hisD6592*(Ts) *hisB2135 supU1283*
TR5610	*hisO9675 hisG1102 hisT1504*
TR5879	*hisO1242 hisB2135 spc-101*
TR5880	*hisO1242 hisB2135 spc-102*
TR5917	*hisO1242 hisD6592*(Ts) *hisB2135 supU1283 rpsL1*
TR5918	*hisO1242 hisD6592*(Ts) *hisB2135 supU*(Wt) *rpsL1*
TR5921	*hisO1242 hisB2135 rpsL1*
TT172	*cysG1510*::Tn*10*
TT609	Δ*his-644 zee-1*::Tn*10 supU1283*
TT627	*rpsL1 pyrC7*/F'ts-114 *lac*$^+$ *zzf-20*::Tn*10* (Chumley et al., 1979)
TT1218	*argD1883*::Tn*10*
TT2398	Dp118[*lys*(*serA cysG1542*::Tn*5*)(*serA cysG*$^+$)*ilv*]
TT2402	DP122[*lys*(*serA cysG1542*::Tn*5*)(*serA cysG*$^+$)*ilv*]
TT3538	Δ*his-644 zee-1*::Tn*10 supU*(Wt)
TT4029	*hisO1242 hisB2135 zhb-736*::Tn*10 supU1283*
TT4030	*hisO1242 hisB2135 zhb-736*::Tn*10 supU*(Wt)
TT4033	*hisO1242 hisB2135 zee-1*::Tn*10 cysG439*
TT5474	*hisG205 supU1283*
TT5475	*hisG205 supU*(Wt) *zee-1*::Tn*10*
TT5476	*hisO1242 hisC3721 supU1283*
TT5477	*hisO1242 hisC3721 supU*(Wt)
TT5478	*hisO1242 supU1283*
TT5479	*hisO1242 supU*(Wt)
TT5480	*hisO*$^+$ *supU1283*
TT5481	*hisO*$^+$ *supU*(Wt)
TT5482	*hisO1242 hisC3716 supU1283*
TT5483	*hisO1242 hisG3716 supU*(Wt)
TT5484	*hisO1242 hisG3719 supU1283*
TT5485	*hisO1242 hisG3719 supU*(Wt)
TT5486	*hisO1242 hisG3720 supU1283*
TT5487	*hisO1242 hisG3720 supU*(Wt)
TT5488	*hisO1242 hisG3723 supU1283*
TT5489	*hisO1242 hisG3723 supU*(Wt)
TT5490	*hisO1242 hisG3724 supU1283*
TT5491	*hisO1242 hisC3724 supU*(Wt)
TT5494	Dp118[*lys*(*serA cysG1542*::Tn*5*) (*serA cysG*$^+$)*ilv*] *hisB2135*
TT5495	*hisB2135 cysG1542*::Tn*5* (haploid segregant of TT5494)
TT5496	Dp122[*lys*(*serA cysG1542*::Tn*5*) (*serA cysG*$^+$)*ilv*] *hisB2135*
TT5497	*hisB2135 cysG1542*::Tn*5* (haploid segregant of TT5496)
TT5498–TT5500	Dp118[*lys*(*serA supU1283 zhb-736*::Tn*10 cysG1542*::Tn*5*) (*serA supU*(Wt) *cysG*$^+$)*ilv*] *hisB2135*
TT5501–TT5504	Dp122[*lys*(*serA supU1283 zhb-736*::Tn*10 cysG1542*::Tn*5*) (*serA supU*(Wt) *cysG*$^+$)*ilv*] *hisB2135*
TA389	*hisO1242 hisB2135*

[a] Unless otherwise noted, all strains were constructed for this study. For strains containing duplications (e.g., TT2398), the duplications are denoted Dp, followed by an isolation number. The most distal genes known to be included in the duplication are enclosed in parentheses; the genes closest to the endpoints of the duplication that are known not to be duplicated are outside the parentheses. Markers near and in the duplicated region of the chromosome are enclosed in brackets. *rpsL* was formerly *strA*.

grown on various *his* nonsense mutants. Prototrophic (His$^+$) transductants arising from this cross indicated that the donor *his* mutation is suppressed by *supU1283*. If no His$^+$ transductants arise, it is concluded that the donor mutation is not suppressed by *supU*. In this way, we found that *supU1283* suppresses 10 of 12 *his* UGA mutations tested, but does not suppress any of 18 amber or 2 ochre mutations tested. Among the UGA mutations suppressed by *supU1283* is *hisB2442*, which was generated by conversion of an ochre (UAA) mutation to UGA (14).

Suppressor efficiency. The efficiency of suppression was determined by measuring the amount of polarity relief caused by the suppres-

sor in *his* UGA mutants. As shown in Table 2, *supU1283* is quite inefficient.

Mapping the suppressor locus. To map the *supU* locus, we took advantage of some recently developed genetic techniques using transposons (8). A Tn*10* (tetracycline resistance) element was placed near the suppressor locus by standard methods (see above). This Tn*10* element was then used as a region of homology to direct the formation of an Hfr by the insertion into the chromosome of an F′ plasmid that also carries a Tn*10* element (3). The approximate location of the origin of chromosome transfer of the resultant Hfr, and therefore the location of the Tn*10* insertion, was mapped. Results of this preliminary mapping (data not shown) indicated that the suppressor locus lies between 63 (*serA*) and 72 (*cysG*) min on the *S. typhimurium* map.

A more precise location of *supU*, and of the Tn*10* insertion linked to it, was then determined by phage P22 transductional crosses. These results, summarized in Fig. 1, show that *supU* maps near *rpsL* (formerly *strA*) at 72 min. The cotransduction frequencies with nearby markers (Table 3) allow the determination of gene order shown in Fig. 1. (The relative order of *supU* and *spc* is unknown.) The important point to note is that *supU* clearly lies to the left of *rpsL*, tightly linked (99%) to a spectinomycin resistance mutation (*spc*), which places it in or near a large cluster of ribosomal protein genes (11, 13; see below).

***rpsL* mutations abolish *supU* activity.** In the process of mapping *supU*, we discovered that *rpsL* (Str^r) mutations abolish the suppressor activity of *supU1283*. When strain TR4768 (*hisB*[UGA] *supU1283*) is transduced to streptomycin resistance with phage P22 grown on the *rpsL1* mutant, all of the recombinants are His^-, indicating that they have all lost suppressor activity. However, many of these transductants can be shown to possess the *supU1283* suppressor when the *rpsL1* mutation is removed by a second transduction. Of the His^- streptomycin-resistant transductants of TR4768, 24 were picked and transduced to tetracycline resistance with phage grown on a strain (TT4030) containing a Tn*10* insertion (*zhb-736*::Tn*10*) linked to *rpsL* and *supU*. This donor strain carries a wild-type copy of *supU*. Of these strains, 19 yielded His^+ recombinants among the tetracycline-resistant transductants arising in the second cross. This indicates that the *supU1283* mutation is still present, but inactive in the presence of *rpsL1*. The recombinants that are unable to yield His^+ transductants in the second cross are those that had lost *pU1283* in the initial transduction, and their number (5 of 24) represents the linkage of *supU* to *rpsL* reported in Table 3. A weakening effect of *rpsL* mutations on non-

TABLE 2. *Suppressor efficiency*[a]

Strain	Genotype	*hisB* sp act	Suppressor efficiency
TT5480	*hisO*⁺ *supU1283*	1.10	
TT5481	*hisO*⁺ *supU*(Wt)	2.27	
TT5474	*hisO*⁺ *hisG205 supU1283*	0.39	
TT5475	*hisO*⁺ *hisG205 supU*(Wt)	0.34	6.6
TT5478	*hisO1242 supU1283*	19.90	
TT5479	*hisO1242 supU*(Wt)	23.50	
TT5482	*hisO1242 hisC3716 supU1283*	1.31	1.0
TT5483	*hisO1242 hisC3716 supU*(Wt)	1.11	
TT5484	*hisO1242 hisG3719 supU1283*	3.43	<0.1
TT5485	*hisO1242 hisG3719 supU*(Wt)	3.42	
TT5486	*hisO1242 hisB3720 supU1283*	2.97	3.3
TT5487	*hisO1242 hisG3720 supU*(Wt)	2.39	
TT5476	*hisO1242 hisC3721 supU1283*	2.39	1.1
TT5477	*hisO1242 hisC3721 supU*(Wt)	2.19	
TT5488	*hisO1242 hisG3723 supU1283*	2.62	3.7
TT5489	*hisO1242 hisG3723 supU*(Wt)	1.95	
TT5490	*hisO1242 hisC3724 supU1283*	1.86	3.2
TT5491	*hisO1242 hisC3724 supU*(Wt)	1.27	

[a] The ability of *supU1283* to relieve the polarity caused by *hisG* and *hisC* UGA mutations was measured. Histidinol phosphate phosphatase levels (encoded by the *hisB* gene, which is downstream of *hisG* and *hisC*) were assayed by the method of Martin et al. (10). Cells were grown to late log phase (optical density at 650 nm [OD_{650}] of 0.8 to 1.2) in minimal medium plus 0.1 mM L-histidine. Specific activity is $OD_{820/ml/20\ min}/OD_{650}$ of cells. Suppressor efficiency was calculated from effects of the suppressor on polarity by the following formula: efficiency (%) = $[(mt^s - mt^u)/(wt^s - mt^u)] \times 100$. In this formula, mt^s is the *hisB* activity formed in the mutant strain with a suppressor, mt^u is the *hisB* activity formed in the unsuppressed mutant, and wt^s is the enzyme level formed in a strain carrying the suppressor but lacking a polar *his* mutation. All strains in the table which carry the *hisU* mutation are phenotypically His^+.

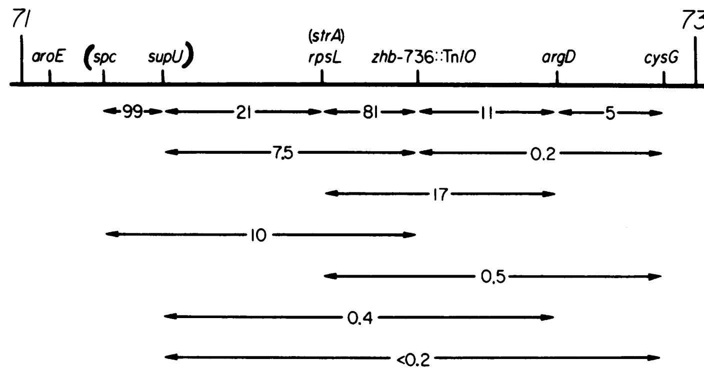

FIG. 1. *Genetic map of the 71- to 73-min region of the Salmonella chromosome. The numbers below the line are P22 cotransduction frequencies (in %), reported in Table 3. Gene designations are those of Sanderson and Hartman (16). Mutations in the spc gene are resistant to spectinomycin (21). The relative order of supU and spc is unknown.*

TABLE 3. *Cotransduction frequencies of genes in supU region*[a]

Gene	Cotransduction frequency				
	supU1283	*rpsL1*	*zhb-736*::Tn*10*	*argD10*	*cysG439*
supU1283		21 (24)	7.5 (855)	0.4 (237)	<0.2 (449)
rpsL1 (*strA1*)	21 (24)		81 (200)	17 (100)	0.5 (200)
argD10	0.4 (237)	17 (100)	11 (400)		5 (200)
cysG439	<0.2 (449)	0.5 (400)	0.2 (400)	5 (200)	
spc-102	99 (298)		10 (621)		

[a] Percent cotransduction frequencies are reported. The numbers in parentheses are the number of transductants tested. In each case, both markers to be tested were selected for in separate transductions, and the other marker scored. The results of these transductions were then pooled and averaged. For example, to test linkage of *zhb-736*::Tn*10* to *supU1283*, Tet^r was selected, and *supU* scored in one cross; *supU* was selected and Tet^r scored in a reciprocal cross. The results of these two experiments were then averaged to give the number reported in the table. The cotransduction values reported for *argD10* and *cysG439* also included experiments using the alleles *argD1883*::Tn*10* (strain TT1218) and *cysG1510*::Tn*10* (strain TT172).

sense suppression has been observed for a number of different tRNA suppressors (19, 21).

Suppressor dominance tests. To determine if *supU1283* is dominant or recessive to the wild-type allele of *supU*, we constructed strains diploid for the *supU* locus by using mutants carrying duplications of this region of the chromosome (2; R. P. Anderson and J. R. Roth, unpublished data). The two strains used (TT2398 and TT2402) carry duplications of independent origin that include the chromosomal region between 62 and 73 min; the duplications include the *serA* and *cysG* genes, but do not include *lys* or *ilv* (Fig. 2). Such duplications are unstable, being frequently lost by recombination between the two copies (1). However, since these strains contain a Tn*5* (kanamycin resistance) insertion element in one of the duplicated *cysG* genes, the duplication can be maintained by simultaneously selecting kanamycin resistance and Cys$^+$; only those bacteria with two copies of *cysG* can exhibit both phenotypes (2).

To determine the dominance, mutation *supU1283* was transduced into one of the copies of the duplication, and then tested for the ability to suppress the *hisB* UGA mutation present in this strain. Phage P22 grown on a mutant (TT4029) containing a Tn*10* insertion near *supU1283* was used to transduce strains (carrying a *his* UGA mutation) that are either diploid (TT5494, TT5496) or haploid (TT5495, TT5497, TR5601) for *supU*$^+$, selecting for inheritance of the Tn*10* insertion (tetracycline resistance). Haploid transductants that inherit Tn*10* and the donor *supU1283* mutation will become His$^+$; diploid transductants that inherit *supU1283* will be His$^+$ if the suppressor is dominant, and His$^-$ if it is recessive. Table 4 shows that *supU1283* is inherited with about the same frequency (4 to 8%) in the diploid as in the haploid recipients, indicating that the suppressor is active in diploids, and is therefore dominant to *supU*(Wt).

To verify that the diploid His$^+$ transductants of strains TT5494 and TT5496 (lines 2 and 4 of

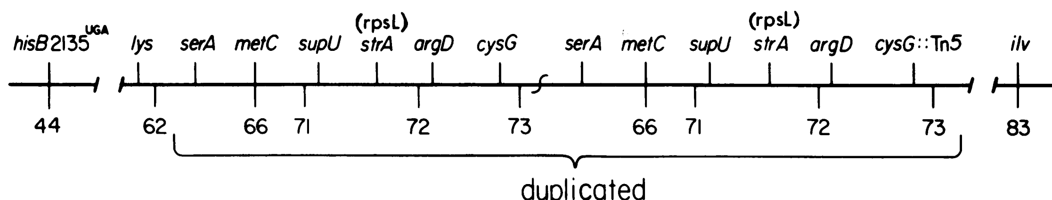

FIG. 2. *Genetic map of the chromosomal duplications used for dominance tests. The extent of the duplicated chromosomal material in strains TT5494 and TT5496 is indicated beneath the map. The join point of the duplicated copies is represented by a wavy vertical line. These two strains were verified to be duplicated for metC and argD. Phage P22 grown on metC::Tn10 or argD::Tn10 insertion mutants was used to transduce TT5494 and TT5496 to tetracycline resistance. All of the transductants that inherited metC::Tn10 or argD::Tn10 (and that remained Cys$^+$ and Kan^r) were Met$^+$ or Arg$^+$, demonstrating that metC and argD are duplicated. Using Cys$^-$ Kan^r haploid segregants of these strains (TT5495 and TT5497) as recipients for the transductions, all of the Tet^r recombinants obtained with phage grown on argD::Tn10 or cysG::Tn10 are Arg$^-$ or Met$^-$.*

TABLE 4. *Cotransduction of zhb-736::Tn10 and supU1283 in strains haploid or diploid for the supU locus*[a]

Donor	Recipient	Recipient genotype	State of *supU*	% His$^+$ transductants
TT4029	TR5601	*hisO9675*(UGA) *supU*(Wt)	Haploid	7
TT4029	TT5494	*hisB2135 supU*(Wt)/supU(Wt)	Diploid	4
TT4029	TT5495	*hisB2135*(UGA) *supU*(Wt)	Haploid	8
TT4029	TT5496	*hisB2135 supU*(Wt)/*supU*(Wt)	Diploid	5
TT4029	TT5497	*hisB2135 supU*(Wt)	Haploid	4

[a] The recipient strains were transduced to tetracycline resistance; the transductants that also inherit and express the linked *supU1283* allele are His$^+$. For each cross, 100 Tet^r transductants were scored. Strains TT5494 and TT5496 are independently isolated mutants that carry duplications of the *supU* region. Strain TT5495 is a haploid segregant (Cys$^-$) of TT5494; TT5497 is a haploid segregant of TT5496. The donor strain (TT4029) carries *zhb-736*::Tn*10* and *supU1283*.

Table 4) carry two copies of *supU*, seven His$^+$ transductants were picked, grown nonselectively for several generations, and then plated to select the Cys$^-$ segregants that have lost the duplication (see above). Some of the haploid segregants should have lost the *supU1283* allele and become His$^-$; some should lose the wild-type *supU* gene and remain His$^+$. Table 5 shows that all of the His$^+$ strains believed to be diploid for *supU* [*supU1283*/*supU*(Wt)] give rise to both His$^+$ (*supU1283*) and His$^-$ [*supU*(Wt)] segregants. This demonstrates that both forms of the *supU* gene are present in the diploids. We conclude that *supU1283* is dominant to wild-type *supU*.

DISCUSSION

Most nonsense suppressors in bacteria are due to alterations in tRNA genes, usually alterations of the tRNA anticodon. The mapping of *supU1283* in or near a cluster of ribosomal protein genes suggests that it may be due to an altered ribosomal protein. Work of Kurland et al. (9) and Olsson and Isaksson (12) has suggested that UGA-specific suppressors might be found among ribosomal mutants.

TABLE 5. *Segregation of supU in duplication-containing strains*[a]

Strain	No. of Cys$^-$ segregants tested	No. of haploid segregants with following genotype:		
		zhb-736::Tn*10* *supU1283*	*zhb-736*::Tn*10* *supU*(Wt)	*supU*(Wt)
TT5498	46	4	3	39
TT5499	46	2	44	0
TT5500	46	2	0	44
TT5501	46	1	0	45
TT5502	46	3	4	39
TT5503	46	1	0	45
TT5504	46	0	0	46

[a] Cys$^-$ segregants were selected (as described in the text) and their genotype, inferred from the phenotype (His and Tet), was determined.

The region of the *Escherichia coli* chromosome between *rpsL* and *aroE* has been precisely mapped by physical and biochemical techniques (11). This region consists of four ribosomal protein operons: the *str* operon, the S10 operon, the *spc* operon, and the alpha operon. The *str* op-

eron is separated from the other three operons by a 13-kilobase piece of DNA containing no known genes. The distance of *supU* from *rpsL* is 17 kilobases, roughly estimated from the cotransduction frequency (20). Even allowing for a large error in this estimate, it clearly places *supU* in or very near the *spc* or S10 operons. Also, the linkage (99% = 0.14 kilobase) of *supU1283* to a spectinomycin resistance mutation, which in *E. coli* affects ribosomal protein gene *rpsE*, suggests that *supU* lies in or very near the *spc* operon. No known tRNA genes lie in this region (5, 13, 17). For these reasons, we favor the hypothesis that *supU* encodes a ribosomal protein. Mutations in ribosomal protein genes are known to affect translational fidelity, although the effects are not codon specific (19, 22, 23). The *supU1283* mutation is distinct in that it seems to only affect reading of UGA codons. It remains possible that *supU* encodes a tRNA, since the results of the dominance tests do not rule out this possibility. Determination of the nature of the *supU* gene product awaits further experimentation.

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